

Genij Ortopedii. 2022. Vol. 28, no. 4. P. 608-618.

Review article

<https://doi.org/10.18019/1028-4427-2022-28-4-608-618>

**Perioperative prognosis of infectious complications after total hip and knee arthroplasties.
Part II (literature review)**

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Abstract

Introduction Risk factors in the perioperative period are important for reduction of the infection rate following total hip and knee arthroplasty. **The objective** of the review was to systematize information on potentially modifiable risk factors for infectious complications following total hip and knee arthroplasty and the possibilities to control them. **Material and methods** For a comprehensive search, PubMed, eLIBRARY, Scopus, Dimensions were used. The search depth was 30 years. **Results** The review reports potentially modifiable risk factors and the possibility to control them in the perioperative period. Patients undergoing total joint replacements often suffer comorbid conditions that must be addressed preoperatively and postoperatively. Comorbidities can be associated with such joint pathologies as oligo-, polyosteoarthrosis, arthroplasty of other joints, septic arthritis or with a history of periprosthetic joint infection. Somatic disorders can be associated with abnormal laboratory findings. All these risk factors cannot be eliminated completely and are detrimental for hip and knee arthroplasty. **Discussion** The current level of information on the risks of infectious complications following total hip and knee arthroplasty may be insufficient to reduce the spread of an infectious agent. There is controversy regarding some predictors of surgical site infection and periprosthetic joint infection. There may be equivocal cause-effect relationships between the patient's potentially unfavorable features and the adverse outcome, which requires further in-depth study of this problem.

Keywords: arthroplasty, surgical site infection, periprosthetic joint infection, prognosis, risk factors

For citation: Bragina S.V., Moskalev V.P., Petrushin A.L., Berezin P.A. Perioperative prognosis of infectious complications after total hip and knee arthroplasties. Part II (literature review). *Genij Ortopedii*, 2022, vol. 28, no 4, pp. 608-618. DOI: 10.18019/1028-4427-2022-28-4-608-618.

INTRODUCTION

Surgical site infection (SSI) after joint replacement (JA) is reported to develop in 2–8 % of patients. Deep SSI is observed in 0.2–5 % of cases [1, 2]. Periprosthetic joint infection (PJI) is a devastating complication that can accompany ES and lead to serious health consequences for the patient [3]. Comparing the mortality rates of patients, the authors report a worse prognosis for PJI patients than for those diagnosed with prostate cancer, breast cancer, Hodgkin's lymphoma, and a number of other common malignancies [4, 5]. Untimely detected or ineffectively debrided chronic infection, the presence of a foreign body, instability of implant, ischemia of tissues at the implant site are adverse factors the appearance of SSIs with resultant decrease in the bactericidal properties of paraprosthesis tissue fluid [6]. The median time to diagnosis of

infection after ES is reported to be 12.2 months, the median time from ES to removal of the implant due to infection to be 18.5 months and for all other complications, 50.5 months. Revision intervention for septic complications after primary arthroplasty is performed after an average of 25.7 months [7]. While the adverse event is not common, it compromises the generally well-established surgical procedure. For more than half a century, the SSI has haunted the orthopaedic community and stimulated researchers to look for ways to predict and minimize the risks of developing this formidable complication of JR [5, 8]. The objective This review is a continuation [9] of systematization of information on potentially modifiable risk factors for infectious complications following total hip and knee arthroplasty and the possibilities to control them.

MATERIAL AND METHODS

The original literature search was conducted on key resources including PubMed, eLIBRARY, Scopus, Dimensions. Search depth was 30 years. The search was

carried out using the keywords in Russian and English: hip arthroplasty, knee arthroplasty, periprosthetic joint infection, surgical site infection, forecasting, risk factors.

RESULTS

The review focuses on potentially modifiable SSI/PJI risk factors.

Somatic pathology

Patient's concomitant diseases are a risk factor for total joint arthroplasty. Diabetes mellitus (DM) is a known independent risk factor for the development of PJI [10–12]. Patients with diabetes and patients with hyperglycemia are at risk of poor wound healing due to pathophysiological reactions associated with lower vascular wall permeability, impaired oxygen delivery and deteriorated redox reactions, neutrophil adhesion, chemotaxis and phagocytosis of immune cells, changes in antibody responses and complement proteins and intracellular bactericidal activity and low protection against bacteria [13]. About 1.2–12 % of DM patients undergoing JR develop complications associated with a postoperative wound [14] with elevated glucose levels increasing formation of biofilms [15]. DM is one of the most common diseases worldwide, and the number of DM patients is predicted to increase to 592 million people by 2035 [16]. The number of DM patients undergoing JR procedures is likely to increase annually, and the pathology significantly increases the risk of severe osteoarthritis [11, 17]. The incidence of DM among patients undergoing ES is reported as high as 12.2 % [11]. PJI develops in 2.19 % of diabetic patients after TKR and in 1.59 % after THR, compared with infection rates of 0.48 % and 0.66 % in non-diabetic patients, respectively [18]. The authors compared uncontrolled diabetes, controlled diabetes, and non-diabetic patients and reported a significantly increased risk of developing PJI with diabetes being not adequately controlled [adjusted OR = 2.28; 95 % CI 1.36–3.81; $p = 0.002$] [19]. The systemic rheumatic pathology affects the musculoskeletal system including joints, with the development of secondary osteoarthritis, there are always patients in this cohort having indications for arthroplasty. Rheumatoid arthritis (RA) is characteristic of the most severe and most common condition of destructive joint damage and impaired function. The pathology is seen in about 5 % of patients with indications for JR [20]. RA was found to be an independent risk factor for the development of PJI, with the incidence of infection being higher in RA patients than in no-RA patients [18, 21, 22].

Both the disease and comorbid conditions are predisposing factors including immunosuppressive therapy [23], including glucocorticosteroid and disease-modifying basic anti-inflammatory drugs (DMARDs),

such as azathioprine, methotrexate, cyclophosphamide, and others that inhibit the production of tumor necrosis factor alpha (TNF- α) or interleukin 6 (IL-6) [24, 25]. A targeted suppression of immunity occurs with use of the therapy with the drugs with resultant violation of the regenerative abilities of tissues and increased rate of SSI/PJI [26]. There is an ongoing discussion in the literature about the algorithms for managing the RA patients in the perioperative period of JR.

The use of such a well-known drug as methotrexate is still discussed in terms of two positions: continuation of methotrexate therapy in the perioperative period with increased incidence of PJI vs continuation of methotrexate therapy in the perioperative period not affecting the incidence of PJI [27]. Carpenter et al. reported that discontinuation of methotrexate a week before and after surgery significantly reduced the risk of PJI in RA patients undergoing elective arthroplasty [28]. A. Grennan et al. reported that patients who continued treatment with methotrexate showed a significantly lower incidence of PJI compared with patients who discontinued methotrexate two weeks before and after surgery, suggesting that methotrexate treatment should not be discontinued in patients with RA prior to elective arthroplasty [29]. Although there is no consensus in the literature on termination of the antirheumatic therapy before JR, experts from the American College of Rheumatology and the British Society of Rheumatology recommended to refrain from taking TNF- α inhibitors during arthroplasty [4, 30, 31]. The International Consensus Group on Joint PJI recommended discontinuation of DMARDs before elective arthroplasty and developed a schedule for discontinuation of various immunosuppressants [4, 32].

Acute hemorrhagic anemia that is common in the postoperative period due to blood loss, is diagnosed with the hemoglobin level measuring less than 12 g/dl in women and less than 13 g/dl in men and occurs in 15–33 % of cases of major joint arthroplasty [32]. Patients with preoperative anemia have a higher risk of developing PJI [10, 12, 21]. Multivariate analysis conducted by M. Greenky et al. showed that the risk of PJI is 2.2 times higher in patients with anemia than in patients without anemia ($p < 0.01$) with preoperative anemia identified as an independent risk factor for PJI [33]. There is a paucity of literature reporting the pathophysiological relationship between preoperative anemia and an increased risk of PJI. There is evidence that patients with preoperative anemia are more likely

to receive perioperative allogeneic blood transfusion, which may be associated with increased PJI [34, 35]. The cause of the anemic syndrome is to be established preoperatively with the status to be corrected until the threshold values are reached [36–38]. Preoperative administration of recombinant human erythropoietin can be considered as an option for preoperative modification of anemia despite the financial costs to reduce the need for blood transfusion in the perioperative period and the risk of developing PJI [38, 39].

Serious liver disease can be associated with a higher rate of complications after JR. Liver cirrhosis and hepatitis B virus (HBV) are independent predictors of PJI [40–42]. There is an increased risk of infection due to chronic anemia, intraoperative and postoperative bleeding. Mechanisms for the development of infectious complications include migration of bacteria from the intestine, dysfunction of polymorphonuclear leukocytes, complement deficiency and dysfunction of the reticuloendothelial system [43]. Researchers have shown the results of excessive activation of pro-inflammatory cytokines – IL-6 and TNF- α in patients with cirrhosis after ES [44, 45]. T. Deleuran et al. reported a greater risk of PJI during the year after arthroplasty in patients with liver pathology as compared to the control group (3.1 % (CI: 1.6–5.2) vs. 1.4 % (CI: 1.3–1.4)) [46]. L.A. Poultides et al. considered comorbid liver disease in patients undergoing THR/TKR as the strongest predictor of PJI, increasing the likelihood of this complication by 2.5 times [47], while P.H. Hsieh et al. emphasized that PJI is difficult to address after THR in patients with cirrhosis [48]. There is opinion regarding the lack of relationship between cirrhosis and PJI [49]. S.J. Kuo et al. reported a significantly higher risk of developing PJI at 0.5-1 year after TKR in males with HBV compared to males without HBV by 18.7 times and by 4.80 times at more than 1 year. The authors recommended discussing the risk of PJI with male HBV carriers prior to TKR [41].

Patients with osteoarthritis associated with serious cardiovascular disease tend to be in the older age group, they may have problems with postoperative wound healing and they are at higher risk of infection [50]. Several teams of authors have demonstrated patients with cardiovascular pathology and postoperative PJI [10, 12, 50, 51]. Pathological conditions that can cause postoperative purulent complications in the cohort of patients include congestive heart failure (HR = 1.28), peripheral valve disease (HR = 1.13), valvular disease (HR = 1.15) and pulmonary circulation disorders

(HR = 1.42) [10]. The researchers also reported that atrial fibrillation and myocardial infarction are also independent concomitant factors associated with a higher risk of PJI due impaired hemostasis treated with aggressive anticoagulants, associated bleeding and infection [50]. For example, the American College of Thoracic Medicine recommends that patients with atrial fibrillation take high doses of aspirin or warfarin. The authors indicated a higher prevalence of PJI – 9 times higher in patients on anticoagulant therapy (5.6 % vs. 0.62 %; $p = 0.02$). Patients, together with cardiologists are to optimize anticoagulant therapy based on the level of international normalized ratio for adequate preoperative preparation and minimizing the risk of PJI [52].

The so-called cardiovascular risks (anemia, chronic inflammation, oxidative stress, activation of the renin-angiotensin-aldosterone system, stress, hyperuricemia, natriuretic factors, etc.) are also associated with progressive kidney dysfunction. Signs of chronic kidney disease are observed in more than 1/3 of patients with chronic heart failure; impaired kidney function seen in 36 % of people aged 60 years and over, in 16 % of people of working age and in 26 % in the presence of cardiovascular diseases [53]. Chronic renal failure (CRF) is a growing problem worldwide. Patients suffering from CRF receive drugs at the initial stage, and many of them require hemodialysis or kidney transplantation at a long term [54]. Patients receiving long-term hemodialysis are often prone to the development of arthropathy of the joints due to intra-articular and para-articular deposition of amyloid β 2-microglobulin [54]. The patients receive lifelong corticosteroids and immunosuppressive therapy after kidney transplantation, and have a higher risk of developing avascular necrosis of the femoral head [55]. Against the background of, a significant number of patients with chronic renal failure require JR 5-6 times more due to erosive osteoarthritis, osteonecrosis as compared to general population [55, 56].

Patients with CRF are also at greater risk of developing PJI (HR = 1.38 after TKR) [10, 54, 55]. However, the risk of infection may be approximately 2–3 times higher in patients on hemodialysis than in immunocompromised patients after kidney transplantation [55, 57]. Both of these cohorts showed an increased incidence of PJI compared with no-CRF patients [55, 57]. The cause of this increased risk is likely multifactorial. Multiple underlying diseases in the patients can multifactorially in combination contribute to an increase in the incidence of PJI [38, 55]. The immunosuppressive nature of therapy associated with

kidney transplantation and hemodialysis may increase the body's susceptibility to infection [55, 58]. However, other researchers suggest otherwise, reporting no increased risk of PJI in kidney transplant patients after JR [59, 60]. Given the potential danger of antibiotic therapy for patients with chronic renal failure, special control is required for the prevention and treatment of infectious complications of arthroplasty [54].

The influence of urinary tract infection (UTI) as a source for the development of PJI is controversial. C-H. Park et al. reported no relationship between these two localizations of infections [61]. Other authors suggest UTI as a risk factor for PJI [62, 63]. Based on a systematic review and meta-analysis C. Wanga et al. reported a significantly increased the risk of PJI in patients with UTI compared with controls without UTI (RR = 3.17; 95 % CI, 2.19–4.59) with UTI and PJI organisms being similar in the same patient including *Enterococcus faecalis* and *Pseudomonas*, which may indicate a hematogenous route of infection of the joint from the genitourinary tract [64]. It would be more correct to separate symptomatic UTI from asymptomatic bacteriuria, since their causal influence on PJI is quite different [65]. Timely and appropriate treatment of symptomatic UTIs in the group of patients may be important to prevent PJI [63]. We will continue on the asymptomatic form of infection further in terms of assessing laboratory parameters.

A patient has a 2.3 times higher risk of developing purulent complications in the presence of human immunodeficiency virus (HIV) compared with HIV-negative patients (RR = 2.28, 95 % CI 2.14–2.43) [66] due to the higher incidence of comorbidities. C.A. Lin et al. reported that HIV was not an independent risk factor for complications in THR/TKR [67]. Compensated HIV patients receiving active antiretroviral therapy with optimization of overall status, including malnutrition, kidney and liver disease, CD4 cluster lymphocyte count and viral load have a lower risk of PJI that is comparable to HIV-negative patients [68–70].

Highly active antiretroviral therapy (HAART) has now changed the course of the disease and patients can live for many decades. HIV and HAART are also risk factors for the development of negative bone metabolism: osteonecrosis, osteopenia and osteoporosis. The demand for total JR in HIV-infected patients will grow. The impact of modern HIV treatment methods on the level of PJI is currently explored. M.A. Enayatollahi et al. reported a systemic review of 25 studies reporting PJI after primary total arthroplasty in HIV-infected patients

with and without hemophilia and data on HAART during arthroplasty. The frequency of PJI was 2.28 % in HIV patients and 10.98 % ($p < 0.0001$) in combined HIV and hemophilia that was 4.8 times more [69].

Although the pathophysiology of the relationship between depression and PJI is unknown, Bozic et al. identified depression as an independent risk factor for PJI (HR = 1.28) [10]. Depression is known to have a direct effect on the immune system making patients more susceptible to infection [4]. In addition to that, depression and psychosis may be associated with malnutrition and the need for postoperative allogeneic blood transfusion, which are separate risk factors for PJI [10, 71]. Given the evidence of a higher risk of developing PJI in patients with depression, it is reasonable to integrate the assessment of depression into the medical examination protocol before elective JR surgery and postpone surgery until a state of well-managed depression with a predicted adverse outcome [4, 72]. Patients with osteoarthritis have a proportionally increased risk of infection in the presence of two or more concomitant diseases including respiratory and other infections, diabetes, anemia, mental and musculoskeletal comorbidities and at least one combination is enough in RA to develop the complication ($p < 0.001$) [73]. The experts conclude that it is necessary to further study the underlying pathology to be associated with the risk of PJI including affective disorders, depression and anxiety, specific genetic factors, and vitamin D deficiency [74].

Joint pathology

A history of joint arthritis is considered among the risk factors for PJI but the specific approaches and attention of researchers to the population of such patients remain surprisingly limited. Patients with autoimmune inflammatory joint diseases such as rheumatoid arthritis (RA), juvenile inflammatory arthritis, ankylosing spondylitis, and psoriatic arthritis have been found to be at a 1.6–8 times higher risk of developing PJI after JR compared to patients with osteoarthritis [75–77].

There have been controversial reports of a relationship between a history of septic arthritis and the development of PJI after total JR of the same joint, with results ranging from significantly higher PJI to very low rates, and even the absence of PJI was reported. A.A. Sultan et al. identified PJI in 8 % of people in a retrospective multicenter study of patients with a history of septic arthritis at an average follow-up period of 4.4 years (range, 3 months to 17 years) with PJI registered only in patients who underwent TKR. The average time to develop PJI was 10 months (min = 2, max = 20), and

the minimum interval after purulent arthritis, which must be maintained for a relatively safe arthroplasty, was 2 years [78]. Adequate preoperative administration of disease-modifying drug therapy, treatment of preoperative anemia, promotion of smoking cessation and improvement of weight control can be practical for the cases of combined pathologies [79].

The experts are convinced (strong consensus 97 % in favour, medium level of evidence) that total JR in patients with post-traumatic hip/knee arthrosis, with history of operations, including open operations with or without metal constructs applied to the joint planned for arthroplasty (strong consensus, 90 % in favour, limited level of evidence) has a higher risk of developing SSI/PJI [74].

With successful treatment of PJI, patients may develop a degenerative disease in another joint and the need for arthroplasty. Literature data on the risk of developing PJI after primary total hip/knee arthroplasty in patients who have previously undergone PJI of the contralateral joints are extremely scarce. However, investigators have confirmed that a history of PJI predisposes patients to PJI in primary total THR/TKR of other joints [80–82]. The main group of patients in these studies was represented by women in more than half of the cases ranging from 53 [80] to 90 % [81], with an average age of 69 years (min = 45, max = 88), an average BMI of 36 kg/m² (min = 22, max = 59) [80]. The control group consisted of patients without a history of PJI before primary arthroplasty of the contralateral joint. B.P. Chalmers et al. reported that the incidence of PJI in the study cohort was 2.4 times higher than in the control group of patients after TKR without a history of PJI (OR 3.3; 95 % CI 1.18–8.97; $p = 0.02$). About 1/3 of patients (27 %) received prolonged antibiotic therapy. Chronic suppression was observed in 85.7 % of PJI patients who had a higher level of PJI (OR 15; $p = 0.002$). The new virulent microorganism was the same as the previous one in 28.6 % of patients, and the risk of PJI was 15 times higher in patients with prolonged antibiotic therapy [80].

H.Bedair et al. aimed at identifying other potential risk factors for a history of PJI that could predict a similar complication at the site of a second arthroplasty. Patients were matched with controls who had no history of PJI after the first arthroplasty by age, gender, diabetic status, BMI, American Society of Anesthesiologists (ASA) scale, institution, and year of surgery (± 2 years). Patients of the main group were shown to have a higher risk of PJI during subsequent hip/knee arthroplasty with

1.1 % versus 0 % in the control group (OR 21.00; 95 % CI 1.25–353.08; $p = 0.035$). With the exception of PJI, the authors identified no other risk factors associated with a second attack of infection. A history of staphylococcal infection manifested itself in the subsequent PJI in 70 %, and in 35 % of controls (OR 4.26; 95 % CI 0.89–27.50; $p = 0.04$) [81].

Laboratory parameters

In a search of SSI/PJI predictors, researchers explore preoperative data and the dynamics of postoperative laboratory blood parameters in patients after THR/TKR. N.A. Korzh et al. reported a low preoperative content of neutrophils in the peripheral blood with the increase at 7 days without a subsequent sufficient (less than 15.6 %) decrease at 14 days as the criteria for predicting the infectious complications. A decreased level of a factor that inhibits the migration of leukocytes, in the presence of the own serum, synovial membrane, cartilage, bone, and specific pathogens, in particular, is an unfavorable factor in the postoperative period. An increased amount of autoimmune granulocytotoxic antibodies (both in serum and in synovial fluid) is considered as an additional criterion indicating the likelihood of complications. Monitoring of a simple and accessible parameter in everyday clinical practice that would reflect an imbalance between various parts of immunity – the ratio of autoimmune lymphocytotoxic and granulocytotoxic antibodies – allows for the control of the early postoperative period and timely administration of a drug therapy to normalize the neutrophil-lymphocyte link [82]. There is an association found between elevated HbA1c [83, 84] and fasting blood glucose [85] and an increased risk of SSI/PJI [14] but the association is not strong. Perioperative hyperglycemia above 200 mg/dL in arthroplastic patients doubles the risk of PJI. Even without a diagnosis of DM, and the likelihood of PJI is 3 times higher if the blood glucose level on the first postoperative day is > 140 mg/dL [51].

Considering the adverse effect of DM and dysglycemia on the development of postoperative complications, researchers are interested in studying glycated hemoglobin – HbA1c – and performed preoperative screening of this parameter. Some 48 % of patients planning THR/TKR did not suffer from diabetes; 19 % had a history of dysglycemia, 33.6 % had no history of dysglycemia; the condition was assessed as pre-diabetic in 31 % of patients and as diabetic in 2.6 % [86].

With a high prevalence of prediabetic patients and high correlation of dysglycemia with perioperative

complications, the authors recommended routine preoperative HbA1c test in all patients followed by endocrinologist consultation for patients with previously undiagnosed dysglycemia. A significantly increased preoperative level of HbA1c would result in delayed elective surgery with glycemic control achieved [86]. The currently established HbA1c level to be a prognostic sign of SSI/PJI would range between 7.5 and 8.0 % [74, 87, 88].

There is discussion in the literature about the HbA1c threshold for preoperative correction of hyperglycemia. N.J. Giori et al. reported that 41 % of diabetic patients whose arthroplasty was delayed for a reduction in HbA1c to ≤ 7 % failed to achieve this level [89]. Researchers consider 8 % to be a more realistic threshold value requiring preoperative optimization of DM patients [90]. Recent evidence suggests that preoperative fructosamine and early postoperative glucose variability may provide a better prognosis of SSI/PJI, and that routine preoperative screening for DM and glycemic control may potentially reduce the occurrence of infection after total joint replacements [74, 91]. However, there are studies that do not support the role of HbA1c in predicting the development of PJI [92]. So, R. Iorio et al. and Kremers et al. showed no correlation between the HbA1c index and the development of purulent complications. The authors reported that HbA1c values cannot be effective predictors of PJI [93, 94].

Chrastil et al. performed glycemic control by HbA1c levels and did not confirm the ability to predict the risk of PJI (HR = 0.86, $p = 0.23$) [85]. The expediency of preoperative examination of the joint fluid obtained by

joint puncture is discussed in publications. This method is an important diagnostic step that must be performed in all cases predicting the risks of surgical intervention in terms of PJI. Analysis of the bacteriological inoculation of aspirate with the isolation of a pure culture and the determination of the sensitivity of the identified pathogen to antibiotics, microscopy and biochemical examination is practical for the perioperative management. There can be difficulties in interpreting the results: firstly, due to the presence of low-virulence microflora, previous antibacterial treatments, and a positive culture result may not always be obtained [95]. Secondly, the majority of experts recommended further studies to determine the clinical significance of microorganisms or microbial dysbiosis detected, including the native joints, without obvious symptoms of infection, with a direct clinical correlation, long-term follow-up and multicenter validation [74, 96]. Many arthroplasty practitioners are concerned about asymptomatic bacteriuria (AB) in patients as a possible risk factor for PJI. However, the available evidence establishing a direct link between AB and PJI is limited [97]. Asymptomatic urinary tract infections are common in obese older women with diabetes. H. Singh et al. reported that AB in these patients did not affect the incidence of early complications after TKR [98]. Other researchers reported AB in the perioperative period of joint arthroplasty as a risk factor for PJI [99, 100], while the use of antibiotic therapy in such patients does not affect the incidence of PJI [101–103], and regular urine screening before elective total joint arthroplasty and treatment of patients with BD is not recommended [104].

DISCUSSION

Potential impact of certain factors in the perioperative period on reducing the risk of SSI/PJI is reported. While significant advances in identifying risk factors have been made in the past decades, controversy remains about some of the risk factors that predispose to SSI/PJI. An unambiguous causal relationship between a potentially adverse event and a negative surgical outcome may be difficult to identify in a patients based on the results of the studies presented. A greater risk of PJI after THR is reported by the National Arthroplasty Registries [105] even though the known risk factors are adjusted for in a large international study of arthroplasty registries [106]. This has led to growing concern about the potential causes of the increased complication rate, because none of the risk factors previously investigated have been able to interpret this increase [106]. Several

explanations for the increased incidence of PJI have been proposed, including an increased number of patients with comorbidities, a history of surgery, and an increase in bacterial resistance in the presence of foreign bodies [107].

Based on microbiological data of intraoperative cultures from patients with SSI, researchers reported an increase in the resistance of pathogenic flora over the past two decades to beta-lactam antimicrobials, among staphylococci, in particular [108]. However, none of the National Endoprosthesis Registries included the results of a PJI incidence study with the content of the microbiology database. The authors believe that review of PJI trends using the National Arthroplasty Registries can provide controversial results with studies reporting underestimated incidence of PJI by

40 % [109, 110]. There is a paucity of publications on preventive measures for the development of SSI/PJI with high risks of surgical intervention, and the impact of extended oral antibiotic prophylaxis is likely to have promising results in terms of a positive effect on a formidable complication. High-risk patients receiving antibiotics only in the perioperative period are more likely to have PJI than the same patients receiving extended antibiotic prophylaxis. Despite the possible benefits of the latter in JR, methodological limitations, and insufficient discussion of potential adverse events with increased development of antimicrobial resistance, the widespread use of such protocols is limited. More research is needed for narrow definition of risk factors for infection and demonstration of the safety and efficacy of extended antibiotic prophylaxis protocols [111]. Another promising area is the use of preoperative antibiotic allergy testing to expand the use of cefazolin. PJI rates are significantly higher when

non-cefazolin antibiotics are used for perioperative prophylaxis of THR/TKR, therefore, perioperative testing and clearance is recommended for all penicillin and cephalosporin allergic patients given the low rate of true positive penicillin allergy and an easily modifiable risk factor that provides choice of antibiotics [112]. Further genetic studies are also required based on the family clustering of PJI joints.

Understanding familial risks may provide additional direction in developing individual-centred pathways to prevent infections in at-risk patients [113]. Important measures for the prevention of purulent complications in the perioperative period of large joint arthroplasty are carried out to optimize the patient's status at the preoperative stage, using perioperative antibiotic therapy, improving the operating environment, managing blood parameters, using high technologies of surgical intervention, high-quality implants, adequate wound treatment and thorough postoperative care.

CONCLUSION

In an attempt to reduce the incidence of infectious complications, researchers undertake to explore many risk factors associated with the development of SSI/PJI after total joint replacement. Not all of them have a clear correlation with purulent postoperative complications. There is a need for further high-quality studies to find and confirm evidence of this relationship for accurate preoperative examination of the patient for elective arthroplasty, perioperative management to reduce the development of adverse effects and complications of the intervention. No unique biological system of the human body, as an individual, is free from the risk of surgical complications, but knowledge and

understanding of these processes helps orthopaedic and trauma surgeons minimize adverse events and reduce the incidence of infection. The information obtained in the search for research results on the possibility of perioperative prediction of the risks of developing purulent complications after THR/TKR demonstrates the interest of the authors in identifying and analyzing risk factors for SSI/PJI, understanding the need for the further study, systematization and data processing using mathematical modeling and software for obtaining a personalized program of perioperative management of the patient at the stages of outpatient and inpatient care and improving the results of surgical intervention.

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The article was submitted 28.02.2022; approved after reviewing 10.03.2022; accepted for publication 23.05.2022.

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