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Review Article

Persistent Wound Leakage After Total Knee And Hip Arthroplasty

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Abstract

In this mini-review the pathogenesis, pathophysiology, diagnosis, treatment and course of prolonged wound leakage after total hip and knee arthroplasty are discussed. It appears there is a disconcerting lack of research and knowledge concerning this topic. Wide variations in definition, classification, diagnosis and treatment hamper patient management, early mobilisation and rehabilitation, as well as the function of the operated joint, severely.

Introduction

The diagnosis and treatment of persistent wound leakage is an important and poorly understood topic in the field of joint arthroplasty. Persistent wound leakage after total knee and hip arthroplasty is associated with a higher risk of developing periprosthetic joint infection (PJI) [1-6]. PJI is a seious complication with great impact on a patient's physical functioning and quality of life. Moreover, PJI is a high financial burden for society. Additional medical costs of PJI are approximately € 30.000 per patient with even higher societal costs because of productivity loss,home care and informal care provided [7,8]. Unfortunately, there are no evidence-based guidelines for the diagnosis and treatment of persistent wound leakage after joint arthroplasty [6].

Numerous issues hamper the development of sound guidelines. First of all, research on wound leakage is hard, as PJI is used as the major endpoint of wound leakage treatment, which has a low incidence (1,5%)- [9]. Secondly, there is no uniformly accepted definition of wound leakage and when to call it persistent. Clinical practices in orthopedic hospitals vary widely therefore. For that reason pathogenesis, pathophysiology, treatment and course of prolonged wound leakage after arthroplasty are discussed in this mini-review.

Pathogenesis

Following Winter's original research in 1962, it is now widely accepted that a certain amount of moisture in the wound bed is necessary for optimal healing [10]. The difficulty is determining what that certain amount is and how long it should persist. Inflammation is the body's normal protective response to any injury (including surgery) or foreign bodies.Acute inflammation follows the early stage of the foreign body response (protein adsorption) [11,12]. Chemotactic agents within the provisional matrix play a key role in controlling the migration of neutrophils from the vasculature. The travelling leukocytes surrounding the implant become activated in response to the cytokines released by the platelets e.g PDGF (platelet derived growth factor) and beta-thromboglobulin [13].

After localization and activation of macrophages and neutrophils to the site of injury, enzymes are released and then the neutrophils mediated phagocytosis occurs. Theoretically, the phagocytosis should include the procedures of firstly recognizing and attaching to the foreign materials, then engulfing and degrading them. However, due to the materials size, engulfment and degradation are often not possible,although the process of recognition and attachment occurs. Instead, the implants are coated with opsonins such as complement activated fragments C3b and IgG, which aid the adhesion and activation of neutrophils and macrophages [11]. Macrophages assemble at the implant site, leading to further production of chemoattractive-signalling molecules such as PDGF, tumor necrosis factor (TNF-alpha), interleukin 6 (IL-6), granulocyte-colony-stimulating factor (G-CSF) and granulocyte macrophage colony stimulating factor (GM-CSF), leading to further recruitment of macrophages to the implant site [14]. The foreign body response to bulk implant materials is abberant and prolonged.

At the end-stage of the foreign body response,or when the chronic inflammation occurs, mononuclear cells such as monocytes,

lymphocytes and macrophages can present at the implant site. These macrophages which are added by the production of IL-4, IL-13 from Th2 lymphocytes, can fuse together to form a multinucleated foreign body giant cell (FBGC) at the implant surface [15,16]. Next the infiltrated fibroblasts, macrophages and neovascularisation will present within the newly formed granuloma tissue, which is a precursor for forming a fibrous capsule [17,18]. This capsule may contnue to grow following inflammation due to mechanical motions or chemical leaching exerted in the joint. It was thought that the host response to most bulk biomaterials used in THA was identical and followed these main stages. However, the response to the wear particles released by different biomaterials over time differ greatly [11,18,19]. Alumnium ceramics are the most biocompatible while Cobalt-Chromium and Ultra-High Molecular Weight Polyethylene (UHMWPE) have reduced bioavailability [18].

Pathophysiology

Total hip arthroplasty is a commonly performed operation and yet little information exists about the duration of wound oozing,the factors associated with this and the implications. Wood et al. Studied 62 consecutive patients undergoing total hip arthroplasty (THA). Time to dryness was associated with wound length (p=0,01), body mass index (BMI;p=0,05) estimated volume of blood in dissected tissues (p=0,05) and length of hospital stay (p=0,02). No association was found with duration of surgery or ASA (American Society of Anaesthesiologists) physical grades [20]. Local factors compromising wound healing include extensive scarring, lymphoedema, poor vascular perfusion and excessive adipose tissue. Systemic comorbidities affecting wound healing include diabetes mellitus, rheumatoid diseases, renal or liver disease, corticosteroid medication, poor nutrition HIV and smoking.Since a history of smoking is associated with a statistically significant increased risk of PJI, many centers use formal smoking cessation programs to assist patients in giving up, preferably before surgery [21].

Patel et al. conducted a retrospective study to determine the risk factors associated with prolonged wound drainage after hip and knee arthroplasty [5]. Risk factors included a BMI>40kg/square meter, the use of low molecular weight heparin (LMWH) prophylaxis,and a high drain output after THAs. High drain output was the only risk factor associated with prolonged wound drainage after TKAs. HIV infection is also a risk factor for prolonged wound drainage after TKAs [22]. Obesity is a risk factor associated with prolonged operation times,and a higher rate of early postoperative complications, including excessive wound drainage and infection [23]. However,optimal peri-operative glucose control is an important factor in decreasing wound complications for all patients, including those without diabetes [24]. demonstrated that non-diabetic patients were three times more likely to develop PJI if the fasting blood glucose was > 140 mg/dl on the first postoperative day [25]. Proper selection, dosing and timing of prophylactic antibiotics are critical. Most commonly, a first generation cephalosporin is administered within one hour prior to the skin incision.In patients with allergies to penicillin or cephalosporins, clindamycin is an acceptable alternative. For patients with methicillin-resistant Staphylococcus aureus (MRSA) or coagulase-negative Staphylococcus colonisation, vancomycin is used [26].

Diagnosis and Management

Wound healing problems can range from superficial incisional, to deep incisional (outside the joint space) to involving the joint space. Gaine et al. reported a 10% incidence of superficial wound problems in primary TKAs [27,28]. Patel et al. [5] found that each day of prolonged wound drainage increased the risk of deep wound infection by 25% following TKAs. Drainage from the incision one to three days after surgery should be managed by immobilisation in extension, and application of a foam or rolled gauze compressive bandage over the incision. Use of immobilisation and observation should not exceed three days. Wound drainage that persists greater than three days is considered abnormal and should be treated surgically to decrease the chance of subsequent PJI [2,5,22,29].

Aspiration of the joint is necessary if there is a high level of suspicion. The synovial fluid should be analysed for white blood cell (WBC) count and differential.Cultures should also be obtained. There is some consensus with regard to the cell count. In patients with TKAs, a synovial WBC count>1700 cells/ul or a polymorphonuclear neutrophil (PMN) percentage > 65% is rhe recommended threshold for infection [30-32]. In THAs, the recommended thresholds are a synovial WBC count of > 4200 cells/ul or PMN percentage>80% [33]. During the acute postoperative period,within 6 weeks of surgery, the thresholds are higher with a synovial WBC count>10.000 cells/ul and PMN>89% [34].

Treatment

Prolonged wound leakage after arthroplasty is induced by an inflammatory response , as described above (1,10-16). Conversely, surgical wounds may also show prolonged leakage for other reasons (hematoma,seroma or fatty necrosis) and take longer to heal without development of a PJI. Autoimmune disorders as e.g rheumatoid arthritis and SLE are also associated with prolonged wound leakage [35]. The causes of prolonged wound leakage are poorly understood and studies are scarce and methodologically flaw [6]. However,as expected orthopedic surgeons have been focussed primarily at the association between prolonged wound leakage and PJI.

In the Netherlands,the prevalence of prolonged wound leakage at day 9 after index surgery is about 4% ,2200 patients anually of 55.000 THAs and TKAs. The Dutch Arthoplasty Register reports a total of 3809 THA and 2667 TKA revision surgeries performed in 2015. Revision surgery within 1 year of index surgery was necessary in more than 600 patients and at least 30% of these were PJI related [36]. Persistent wound leakage can be treated

by non-surgical and surgical treatment modalities. Non-surgical treatment can consist of relative rest (no exercise and bed rest), pressure bandages,and wound care with sterile bandages.Hospital admission can be required.

Surgical treatment typically consists of debridement, antibiotics and implant retention (DAIR) [37-42]. A DAIR procedure is meant to clean the prosthesis and wound,including break down of the bacterial biofilm, in order to treat the infection and render further infection. Treatment of persistent wound leakage varies considerably among Dutch orthopedic surgeons, as mentioned above [6]. There was a wide variation in classification, definition, diagnosis and treatment of wound leakage. The survey had only a response rate of 18,1%, suggesting wider variations are possible. More than 30 combinations of treatment modalities were used. Remarkably, 23, 4% of responders used antibiotics in the non-surgical treatment of wound leakage, despite the fact that the efficacy of antibiotic treatment in persistent wound leakage has never been studied. Most respondents (43,8%) convert to surgical treatment if wound leakage is present for ten days after index surgery, implying a non-surgical treatment of 3-7 days. Literature offers litlle guidance but suggests that wound leakage more than 3-5 or 5-9 days after index surgery should be managed by surgical treatment.

Several authors have investigated the effect of DAIR for treatment of wound leakage and reported various results,statements or opinions,generally in favour of early DAIR [2-6], [38-42]. The most recent PJI consensus meetings suggest 5-7 days of wound leakage as the threshold to perform DAIR,but there is no solid evidence for this statement. As early DAiR is hypothesized tobe helpful in treating or preventing infection and salvaging the implant, the Dutch Leak study will be started soon. This is a controlled randomized study, enrolling 388 patients, with prolonged wound leakage after THA or TKA. Patients are randomized for surgical treatment (DAIR at day 9-10 from index surgery) or continued non-surgical treatment. Primary outcome is the percentage of reoperations for PJI within one year of index surgery. Secondary outcomes are self-reported questionnaires regarding quality of life etc at 3,6, and 12 months after index surgery.

Course and Outcomes

There is a lack of data on the long-term outcome of THAs [43]. Short and medium- term THA studies report substantial improvements in the generic health related quality of life (HRQol) [44-48]. Mariconda et al. conducted a follow-up study to evaluate the quality of life and functionality of 250 patients an average of 16 years (11-23 years) after THA using a validated assessment set including the SF-36 questionnaire,Harris Hip Score,WOMAC score,Functional Comorbidity Index and a study specific questionnaire. The authors report that patients who had undergone THA have impaired long-term self-reported physical quality of life and hip functionality but they still perform better than untreated patients with hip osteoarthritis. However, the level of post-surgical satisfaction is high [43].

A considerable proportion of patients report long-term pain after THA or TKA for osteoarthritis.Beswick et al conducted an extensive MEDLINE and EMBASE search of articles published to 2011. Of 1308 articles 115 reported patient-centered pain outcomes. Fourteen articles describing 17 cohorts (6 with hip and 11 with knee replacement) presented appropriate data of pain intensity. The proportion of people with an unfavourable long-term pain outcome in studies ranged from 7% to 23% after hip and 10% to 34% after knee replacements. In the best quality studies,an unfavourable pain outcome was reported in 9% or more of patients after THA and about 20% after TKA [49]. There are no specific short- or long-term studies (>3 years after index surgery) concerning the effect of persistent wound leakage after THA or TKA on quality of life and joint function, whatever the cause or treatment of prolonged oozing.

Conclusion

There is a disconcerting lack of research and knowledge concerning the treatment of prolonged wound leakage after THAs and TKAs, surgeries performed in huge numbers worlwide. With an estimated prevalence of 4%, patients with prolonged wound leakage after arthroplasty also represent a lot of people. Prolonged wound leakage is induced by inflammation, caused by infection immunologic incompability to the implant, autoimmunity as in rheumatoid disorders and SLE, or decreased host defence as in e.g.HIV infection. It will be no surprise that orthopedic surgeons primarily focussed on the association between the low incidence periprosthetic joint infection (1,5%) and prolonged wound leakage after THA or TKA. Recently, a Dutch survey among orthopedic surgeons showed a wide variation in definition, classification, diagnosis and treatment of prolonged wound leakage after arthroplasty.More than 30 combinations of treatment modalities were in use. Remarkably, the unproven use of antibiotics was present in nearly 25% of non-surgical treaments of this issue.

There is no evidence favouring non-surgical treatment above surgical treatment or vice versa. Nevertheless, DAIR (debridement, antibiotics and implant retention) is favoured by most orthopedic surgeons at 3-5,5-7 and 9-10 days after index surgery. This arbitrarily and hypothetical timing of DAIR will be studied in an upcoming Dutch trial, the LEAK trial, randomizing 388 patients with prolonged oozing for DAIR at day 9-10 after index surgery versus non-surgical treatment. It is clear that whatever the cause and treatment of persistent wound leakage,early mobilisation and rehabilitation of the patient as well as the function of the joint are hampered severely. There are no sttudies availabale evaluating this topic. It is evident a lot has to be learned in managing and treating prolonged wound leakage after the most common performed arthroplasties in orthopedics.

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