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QUESTION 3: How can superficial surgical site infections (SSIs) be differentiated from deep SSIs (i.e., periprosthetic joint infections (PJIs))?

RECOMMENDATION: There is no single objective clinical test or imaging approach established for the differentiation between a superficial SSI, a deep SSI and a PJI. We recommend that clinical evaluation, workup for infection and early joint aspiration should guide the decision.

LEVEL OF EVIDENCE: Limited

DELEGATE VOTE: Agree: 96%, Disagree: 3%, Abstain: 1% (Unanimous, Strongest Consensus)

RATIONALE

SSIs are infections at the incision site occurring within 30 days after surgery or within one year if implants are left in place [1,2]. The definition commonly used for SSI was specified by the Centers for Disease Control and Prevention (CDC) criteria in 1999 [1]. They are generally categorized into superficial incisional, deep incisional and organ/space SSIs [2,3]. Parvizi et al. proposed a new (2018) definition for PJI (see Question 1, Fig.1) [4]. The new scoring-based definition updated the previous one [5] and is evidence-based with externally validated criteria.

Comparing the aforementioned definitions, CDC criteria for diagnosing SSIs are mainly based on clinical evaluations and histopathology findings, while criteria for diagnosing PJIs also include laboratory results. There is no clinical, laboratory or imaging procedure to reliably allow differentiation between SSIs and PJIs or even between the three different subtypes of SSIs. Furthermore, diagnostic criteria for superficial SSIs, such as tenderness, redness, localized swelling and local heat, have low inter-observer reliability [6]. In the CDC definition, fever above 38° Celsius is considered a clinical sign of a deep incisional SSI [2]. Other wound scoring systems also exist, such as ASEPSIS (Additional treatment, Serous discharge, Erythema, Purulent exudate, Separation of the deep tissues, Isolation of bacteria, and Stay as inpatient prolonged over 14 days). However, neither the CDC definition, nor ASEPSIS differentiate superficial from deep incisional and organ/space SSIs [7]. Additionally, a low-volume knee study demonstrated clinical wound scores (Surgical Wound Aspect Score) with superficial infections having lower scores than deep infection [8]. Despite this finding, the observed difference was not statistically significant [8].

We can assume that PJIs correspond to organ/space SSIs and subsequently, we can attempt to differentiate between superficial SSIs and the organ/space SSIs in a total joint arthroplasty (TJA). A working group of the federal Healthcare Infection Control Practices Advisory Committee completed a comprehensive review of National Healthcare Safety Network (NHSN) SSI definitions in 2011 and 2012. They supported the NHSN adoption of the ICM on PJI's definition of a PJI as the hip and knee arthroplasty "organ/space" SSI [9].

A leaking wound following an arthroplasty can be either the result of a hematoma, seroma, fat necrosis or a sign of deep infection and could also be a risk factor for PJIs (odds ratio (OR) 35.9; 95% confidence interval (CI), 8.3–154.6) [10,11]. Persistent wound drainage may be contaminated and result in a deep infection [12–14]. This knowledge led the 2013 ICM to propose surgical treatment of wound drainage within five days after the index procedure [15]. In a review by Zimmerli, it was proposed that classification of the SSI should guide the selection of the optimal surgical management [16]. An infection occurring within one month of an invasive procedure, such as TJA or arthrocentesis, was classified as an early post-interventional PJI [16]. An acute hematogenous PJI occurs after an uneventful postoperative period with symptoms lasting three weeks or less [16]. Chronic PJI is defined as an infection with symptoms persisting for more than three weeks, or a SSI diagnosed later than one month after implantation [16]. Early post-interventional and acute hematogenous PJIs generally are able to be treated with implant-retaining measures, while chronic PJIs require prosthesis removal due to biofilm formation [16].

A literature review was conducted that revealed no single objective, non-invasive clinical test or imaging approach which can differentiate between a superficial SSI and an early deep PJI. Although several studies address the risk factors for SSI or PJI, none of them differentiated these two conditions [9,17]. We recommend that clinical judgment and early joint aspiration should guide the decision to perform a debridement, antibiotics and implant retention (DAIR) procedure or a superficial debridement. Due to the devastating consequences following PJIs, we recommend that surgeons should have a low threshold for performing a DAIR procedure. Surgeons should also differentiate between stitch abscess, which has only minimal inflammation or discharge from suture points, and superficial and deep surgical site infections. This differentiation can guide the surgeon to perform the needed intervention. Patients in whom the deep space is not involved can be subjected to superficial irrigation and debridement only. In contrast, a DAIR procedure is preferable in patients with deep infections.

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QUESTION 4: How can hip septic arthritis be differentiated from toxic synovitis?

RECOMMENDATION: Currently, there is no single diagnostic test or step that can be performed in order to distinguish a patient with a septic hip from one with toxic synovitis non-invasively. Although algorithms have been created to aid in clinical decision making, there is not enough evidence to support their generalization across all populations, therefore, more research still needs to be conducted before they can be fully validated. Clinical reasoning, evaluation and judgment should still be the standard for which physicians make the distinction between these pathologies as they care for their patients.

LEVEL OF EVIDENCE: Limited

DELEGATE VOTE: Agree: 88%, Disagree: 3%, Abstain: 9% (Super Majority, Strong Consensus)

RATIONALE

Differentiating between a septic hip and toxic synovitis is a balance between the potential morbidity and complications of an undiagnosed, infected hip and unnecessary invasive procedures when conservative management would have sufficed. Clinically, there is major overlap in the presentations of hip septic arthritis and toxic synovitis, and no single variable or laboratory result can sufficiently distinguish the two [1,2]. In fact, laboratory values can all be within normal limits even when hip septic arthritis is confirmed [3,4]. While toxic synovitis is transient, the natural history of an undiagnosed and untreated septic hip can lead to multiple devastating sequelae, such as cartilage damage, osteomyelitis, osteonecrosis and sepsis [5]. Multiple studies have attempted to identify and simplify the diagnostic procedure in order to better guide clinical decision making and treatment.

Although there is no one differentiating factor that can be statistically quantified between hip septic arthritis patients and those with toxic synovitis, Kocher et al. created a clinical algorithm based on four predictive variables [1,5]. These variables include the inability or refusal to bear weight, history of a fever (defined as an oral temperature >38.5˚C), a serum white blood cell (WBC) count greater than 12,000 cells per cubic millimeter (cells/mm3) and an erythrocyte sedimentation rate (ESR) greater than 40 millimeters per hour (mm/hour) [1]. This was carried out retrospectively and then validated later with a prospective study at the same institution [6]. Their results showed a predictive rate of <0.2% and 2.0% without any predictors and up to 99 and 93% when all four predictors were present, in the retrospective and validation study respectively [1,6].

Similar retrospective studies were also carried out at other institutions and included additional diagnostic variables such as C-reactive protein (CRP) and radiographic findings [5,7,8]. Caird et al. found that CRP was a stronger predictor than ESR and in fact was the second strongest predictor behind oral temperature [5]. However, aside from the validation study performed by Kocher et al. at the same institution, the results of that initial predictive model were not reproducible in all populations to the same 99% predictive rate originally described [4].

Another limitation to the current available data lies in the study designs and the statistical analyses used [9]. A systematic review of the literature found that the patient populations did not differ enough to warrant the variance seen in separate studies [9]. The sample sizes of the studies themselves were called into question and even addressed as a weakness in multiple other studies when analyzing the contrast among the studies [5,8–10].

The variability in evidence shows that currently there is no definitive means of distinguishing hip septic arthritis and toxic synovitis non-invasively. Clinicians must continue to use discerning judgment when assessing patients with potentially infected hips through the use of algorithms, imaging and laboratory studies.

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