Review Article

# Periprosthetic Infection in the Setting of Periprosthetic Total Hip Fractures: Evaluation and Management

Navin Fernando, MD, FRCS(C)
Steven Yacovelli, BS
Javad Parvizi, MD, FRCS

## ABSTRACT

The incidences of periprosthetic fracture and periprosthetic joint infection after total hip arthroplasty are expected to increase exponentially over the coming decades. Epidemiologic data suggest that many periprosthetic fractures after THA occur concurrently with a loose femoral implant. Recent studies suggest an approximately 8% incidence of indolent infection in cases of suspected aseptic loosening. The available data, therefore, suggest that periprosthetic fracture and infection may coexist, and this possibility should be considered, particularly in patients with a loose femoral stem and high pretest possibility. Although currently limited, the available literature provides some guidance as how to manage this complex issue.

The incidence of total hip arthroplasties performed in North America is estimated to increase exponentially in the coming decades, with epidemiologic data suggesting an annual demand of 635,000 primary total hip arthroplasties in the United States by 2030.[1] The incidence of periprosthetic fracture will also likely increase proportionately with the number of total hip arthroplasties performed.[2,3] This increased rate of fracture can be largely explained by the diminished bone quality more commonly seen in an aging patient demographic, increased rates of falls associated with a rising comorbidity burden (eg, dementia, obesity, and deconditioning), and a concurrent increase in the rate of revision total hip arthroplasties performed.[2-4] A challenging scenario encountered by orthopaedic surgeons involves the management of a periprosthetic fracture with a concurrently loose femoral implant. This may in fact represent two distinct clinical scenarios: (1) patients with Vancouver B2/3 traumatic injuries in which the fracture pattern results in femoral implant loosening or (2) a progressively loose femoral implant (due to osteolytic wear or infection) in a patient who subsequently sustains a fracture (with or without significant trauma). A study by Lindahl et al[5] to this latter point identified preexisting loosening of the femoral implant in 70% of patients with periprosthetic femoral fractures after total hip arthroplasty (THA). It should be noted that 47% of these patients had unknown loosening before periprosthetic fracture (qualified based on retrospective radiologic interpretation), and as such broad

From the University of Washington, Seattle, WA (Fernando), and the Rothman Orthopaedic Institute at Thomas Jefferson University, Philadelphia, PA (Yacovelli, and Parvizi).

None of the following authors or any immediate family member has received anything of value from or has stock or stock options held in a commercial company or institution related directly or indirectly to the subject of this article: Fernando, Yacovelli, and Parvizi.

*J Am Acad Orthop Surg* 2022;30:957-964

DOI: 10.5435/JAAOS-D-21-00083

conclusions about incidence of clinical loosening should be interpreted with caution.

In a study by Parvizi et al,[6] it was further suggested that in cases of revision total hip arthroplasty due to presumed aseptic loosening, the incidence of concurrent periprosthetic joint infection (PJI) may be as high as 8% if adequately investigated. The authors recommend to routinely rule out infection through preoperative serologic markers, with the addition of synovial fluid analysis (based on abnormal serologic results or high pretest probability) have been made in all cases of revision total hip arthroplasty for suspected aseptic loosening.[6] Of note, an important limitation of this study was that the diagnosis of periprosthetic infection was made before Musculoskeletal Infection Society (MSIS) adopted criteria, a refinement that may have affected the diagnostic accuracy of infection based on current standards. This study cohort was also limited to those patients undergoing revision due to radiologic and/or clinical evidence of loosening in the absence of acute trauma.

At least two studies have evaluated inflammatory marker kinetics after hip fracture and suggest that elevation may be significant secondary to trauma itself, thereby limiting their value in the preoperative evaluation of postsurgical complication such as infection.[7,8] These findings therefore suggest that conflation could exist when interpreting inflammatory markers in the context of periprosthetic fracture and thus requires refinement for this specific patient population.

## Classification

### Periprosthetic Fracture

The Vancouver classification is by far the most commonly cited, likely due to its strength both as a classification tool and its ability to guide appropriate management[9] (Table 1). Vancouver A fractures refer to peritrochanteric fractures in which the femoral implant is considered well fixed and typically can be treated nonsurgically. Vancouver B1 fractures denote a stable implant that can typically be treated with internal fixation without revision. Vancouver B2 refers to a fracture with a concurrently loose stem, and B3 refers to a fracture with a loose stem and diminished bone stock that may compromise reconstruction. Both B2 and B3 fractures typically require femoral revision with supplemental internal fixation. Vancouver C fractures are those distal to tip of the stem with a stable implant and may be treated with isolated open reduction and internal fixation.

Several studies have evaluated the diagnostic validity of the Vancouver classification, with values approximating 80% for type B fractures.[10,11] These studies furthermore suggest that up to 20% of fractures radiographically identified as stable were found to be loose intraoperatively (on mechanical testing of the stem), and therefore a high level of suspicion should exist before classifying and treating femoral implants as well fixed.

### Periprosthetic Joint Infection

The benchmark for the diagnosis of PJI was recently established by the International Consensus Meeting (ICM) in 2018, offering both high sensitivity (97.7%) and specificity (99.5%).[12] The criteria are evidence based and rely on a combination of clinical findings, serologic markers, synovial fluid analysis, results of frozen section, and isolation of organisms from preoperative or intraoperative cultures (Table 2).

## Epidemiology

The epidemiology of periprosthetic fracture and PJI existing independently after total hip arthroplasty has been reported in several large series and joint registries, although the concurrent incidence of fracture and infection remains poorly elucidated. Data analyzed from the National Inpatient Sample database in 2014 demonstrated that 17.6% of revision total hip arthroplasties in the United States were performed for periprosthetic fractures, representing a 74.7% increase from 2006.[13] Abdel et al[15] reported results from the Mayo Clinic Joint Replacement Database from 1969 to 2011, with a postoperative periprosthetic fracture rate of 3.5% from 32, 644 primary total hip arthroplasties,[14] and a rate of 11% from 5,417 revision total hip arthroplasties

Several large population studies have reported on the overall infection burden in the United States after elective primary total hip arthroplasty. Kurtz et al[16] examined the overall rate of infection over 15 years using the Nationwide Inpatient Sample database, demonstrating a rate of periprosthetic infection of 0.88% in total hip arthroplasty.[16] Ong et al[17] examined the Medicare database with a cumulative patient population of 39,929 primary total hip arthroplasties between 1997 and 2006.[17] The overall infection rate was determined to be 2.22%, with 73.3% of infections occurring within the first 2 years.

No large registry studies that we are aware of have specifically attempted to identify and quantify the

**Table 1.** **Vancouver Classification of Postoperative Periprosthetic Fractures of the Femur After Total Hip Arthroplasty**

| Type of Fracture | Description |
|---|---|
| Type A | |
| AG | Fracture involving the greater trochanter |
| AL | Fracture involving the lesser trochanter |
| Type B | |
| B1 | Fractures at or around the distal aspect of the femoral implant with a well-fixed implant |
| B2 | Fracture at around the distal aspect of the femoral associated with femoral loosening. Good residual bone stock of femoral reconstruction |
| B3 | Fracture at around the distal aspect of the femoral associated with femoral loosening. Poor residual bone stock of femoral reconstruction |
| Type C | Fracture distal to the femoral implant without femoral loosening |

Adopted from Duncan CP, Masri BA. Fractures of the femur after hip replacement. *Instr Course Lect*. 1995;44:293-304. Adaptations are themselves works protected by copyright. So in order to publish this adaptation, authorization must be obtained both from the owner of the copyright in the original work and from the owner of copyright in the translation or adaptation.

concurrent rate of periprosthetic infection and periprosthetic fracture. The current literature is limited predominantly to institutional database studies, which by nature of their design may have intrinsic flaws that prohibit the ability to accurately report incidence. Further epidemiologic evaluation of existing large databases or preferably prospective multicentered efforts are likely necessary to provide accurate data.

## Evaluation

The largest study evaluating the concurrent existence of periprosthetic fracture and infection was conducted by Chevillotte et al,[18] examining the Mayo Clinic institutional clinical patient database and total Mayo Clinic Joint Replacement Database. Two-hundred four patients were identified who underwent surgical management for fracture after total hip arthroplasty between 2000 and

**Table 2.** **The ICM (International Consensus Meeting) 2018 Definition of Periprosthetic Hip and Knee Infection**

| Major Criteria (At Least One of the Following) | Decision | | | |
|---|---|---|---|---|
| Two positive growth of the same organism using standard culture growth methods | Infected | | | |
| Sinus tract with evidence of communication to the joint or visualization of the prosthesis | | | | |
| | Threshold | | | |
| Minor Criteria | Acuteα | Chronic | Score | Decision |
| Serum CRP (mg/L) or<br>D-dimer (μg/L) | 100<br>Unknown | 10<br>860 | 2 | Combined preoperative and postoperative score<br>≥ 6 infected<br>3–5 inconclusive[a]<br><3 not infected |
| Elevated serum ESR (mm/hr) | No role | 30 | 1 | |
| Elevated synovial white blood cell (μg/L) or<br>Leukocyte esterase or<br>Positive alpha-defensin (cutoff/signal) | 10,000<br>++<br>1.0 | 3,000<br>++<br>1.0 | 3 | |
| Elevated synovial polymorphonucleocyte (%) | 90 | 70 | 2 | |
| Single positive culture | | | 2 | |
| Positive histology | | | 3 | |
| Positive intraoperative purulence[b] | | | 3 | |

α These criteria were never validated for an acute infection. β No role in suspected adverse tissue reaction. *Consider further diagnostic testing such as Next-Generation Sequencing.

2006. Based on the Vancouver classification, there were 12 types $A_G$, 1 type $A_L$, 6 types B1, 105 types B2, 35 types B3, and 1 type C fracture. True infection (as determined by either two or more intraoperative tissue or the intraoperative fluid aspiration positive for growth on bacterial culture) was identified in 21 of 204 fractures, accounting for 11.6%. Aspiration was only performed in 41 patients based on surgeon discretion, and as such, the value of synovial fluid analysis was not investigated. They demonstrated elevation of C-reactive protein (CRP) (<10 mg/mL) in 83.3% of infected patients, as opposed to 43.5% in noninfected patients, an elevated erythrocyte sedimentation rate (ESR) (>30/hr) in 50% of infected patients as opposed to 33.3% in noninfected patients, and a peripheral WBC count elevated ($>10.5 \times 10^9$ C/L) in 23.8% of infected patients as opposed to 16.2% of noninfected patients. Based on these values, no specific metric (or combination of metrics) was determined sufficiently accurate to be considered a useful diagnostic test. As in studies by Lindahl[5] (70% implant loose), Belthea[19] (52% loose implant), and Beals[20] (57% implant loose), the predominance of periprosthetic fractures was classified as having a loose femoral implant at the time of revision (Vancouver B2 or B3). Importantly, no study was able to conclude that implants were definitely loose before the trauma, or if loosening occurred as a consequence of the injury, and as such, definite conclusions regarding epidemiology may be difficult to make.

Shah et al[21] identified 121 patients with periprosthetic fracture using their institutional database. Using the Musculoskeletal Infection Society (MSIS) classification of PJI, 14 (11.6%) patients were found to have concomitant infection.[22] Given that cultures could potentially delay fracture treatment, they focused on serum ESR/CRP and synovial fluid white blood cell (WBC)/polymorphonucleocyte (PMN)%. Overall, they found that synovial WBC count (sensitivity 100% and 65% specificity) and differential (sensitivity 100% and specificity 63%) were the best diagnostic tests (area under the curve of 0.84) with optimal cutoffs of 2707 WBC/μL and 77% PMNs. These values are similar to current validated cutoffs in the ICM 2018 criteria for evaluation of chronic infection.[12] Although less accurate than synovial tests, serum ESR and CRP remained relatively sensitive at standard cutoffs of 30 mm/hr and 10 mg/L, respectively.

A recent study by van de Kieboom et al[23] evaluated 144 periprosthetic fractures (101 hips and 43 knee), among whom 41 (34 hip, 7 knees) were diagnosed with a concurrent periprosthetic infection based on the MSIS criteria. Similar to the finding by Shah et al,[21] they found that ESR and CRP had reasonable sensitivity (87% and 95%, respectively) but poor specificity. They also found that synovial WBC count (sensitivity 86% and specificity 85%) was the most accurate diagnostic test (area under the curve of 0.9) with an optimal cutoff of 4552 WBC/μL. Although they suggested an optimal cutoff of 79.5% for PMN differential (sensitivity 73.7% and specificity 63.2%), the diagnostic accuracy of this test (area under the curve [AUC] 0.70) was poorer than that shown by Shah et al.

In the aforementioned studies by Chevillotte et al,[18] Shah et al,[21] and van de Kieboom et al,[23] no treatment protocols for patients with concurrent infection were explicitly described, nor were long-term follow-up or outcomes reported. Although these were primarily diagnostic studies, an improved understanding of complications and outcomes in future studies would provide better guidance with respect to treatment. It may in fact be difficult to apply the same diagnostic MSIS criteria to this specific population until an analysis on clinical outcomes is performed. The incidence of concurrent periprosthetic infection identified with periprosthetic fracture (11.6% in both studies by Shah et al and Chevillotte et al; 28% in the study by van de Kieboom et al) seems to be higher than that of revision total hip arthroplasty for aseptic causes (1.35% to 8.3%).[22,24] To our knowledge, no published guidelines currently exist to suggest appropriate management of these patients.

Given a potentially higher rate of concomitant infection, we recommend that an evaluation for PJI should be routinely done for periprosthetic fracture in which there is a high pretest probability of infection or there is high suspicion of a chronically loose implant before femoral fracture (Vancouver B2 or B3). Evaluation should follow the 2018 ICM criteria, including preoperative serologic testing and joint aspiration. In cases in which the chronicity of loosening cannot be determined, we recommend serologic testing routinely due to reliability of a negative predictive value. Synovial fluid analysis may be performed based on pretest probability of infection. In most hospital systems, urgent synovial fluid analysis may yield preliminary analysis within 1 hour but often requires coordination on the part of surgeon and microbiology laboratory. If this cannot be expediently organized, we do not recommend a delay in surgery but suggest obtaining intraoperative fluid for definitive analysis and microbiology testing and continuing antibiotics until results are reported. Based on the previously noted studied by Shah and van de Kieboom, synovial WBC and

PMN% may have the highest diagnostic accuracy toward the identification of PJI in the setting of periprosthetic fracture, although given limited data the optimal cutoffs remain poorly defined.

If pretest probability of infection is low, we do not recommend delaying surgery for synovial fluid analysis but recommend ESR and CRP as a screening test and routine intraoperative fluid and tissue culture evaluation. If fluid or tissue cultures return a positive culture(s), consultation with infection disease services may be made. Although evidence is very limited, short-term or chronic antibiotic suppression may be considered given the morbidity involved in treating a recurrent or persistent clinical infection.[25,26]

In cases in which the implant is not loose and the predicted fracture management is open reduction and internal fixation only (Vancouver A, B1, or C), patients should be stratified based on clinical suspicion (low or high). Although risk factors for this specific patient population have not been evaluated, extrapolations can be reasonably made based on the PJI literature. Patients in whom a high clinical suspicion exists may include (but is not limited to) any patient with one or more of the following risk factors: (1) a history of perioperative surgical site infection involving the primary hip surgery, (2) physical findings of incisional drainage after the primary hip surgery, (3) history of metachronous joint infection, and (4) radiologic evidence of early osteolysis or loosening (<5 years). Patients with severe persistent or progressive pain (ie, limiting weight bearing) about their hip before periprosthetic fracture may be triaged for risk based on further host factors, including body mass index >40 kg/m$^2$, active smoking, cirrhosis or hepatitis C, HIV or other immunosuppressive diseases, and uncontrolled (HBA1C > 8) diabetes.[27] If clinical suspicion for infection is low, no additional workup for infection is necessary. If clinical suspicion for infection is high, screening can begin with serologic CRP and ESR. Both Chevillotte et al[18] and Shah et al[21] noted negative predictive values for CRP and ESR of greater than 89% each (despite poor overall accuracy). Given these results, if both CRP and ESR are negative, infection is unlikely. If one or two of the screening serologic markers are positive, we recommend that aspiration should be performed preoperatively to determine synovial fluid WBC and PMN% before management. It should be noted that a bloody aspiration may be common depending on the location of fracture. Using an adjusted correction formulas as proposed by Ghanem et al[28] may be of value in this circumstance $\{WBC_{adjusted} = WBC_{observed} - ([WBC_{blood} - RBC_{fluid}/RBC_{blood}])_{predicted}\}$.

Munoz-Mahamud et al[29] evaluated the reliability of frozen sections in the context of Vancouver B2 periprosthetic infection. They demonstrated that 6 of the 11 patients had greater than five neutrophils per high powered field, despite only two of those six patients having positive intraoperative cultures. This resulted in a false-positive rate of 66.6%, suggesting that neutrophil proliferation due to trauma may imitate the inflammation seen in infection. Given the small sample size of this study, however, the utility of frozen sections in the context of periprosthetic fracture likely remains uncertain.

D-dimer elevation has not been evaluated in the context of concurrent periprosthetic fracture and infection, and such warrants further investigation as to its utility as a screening tool. As a nonspecific inflammatory marker, false-positive elevation would likely be high in the context of trauma. Similarly, alpha-defensin and leukocyte esterase have not been evaluated in the context of periprosthetic fracture, and as such, further studies are recommended to determine clinical utility.

Based on the limited studies that have evaluated these metrics (ESR, CRP, synovial fluid analysis, and frozen sections), it seems that their accuracy in comparison to the primary diagnosis of periprosthetic infection in the absence of fracture is diminished. Given that studies evaluating the native inflammatory response in the context of primary hip trauma demonstrate baseline elevation, a reasonable postulation can be made that a higher percentage of false-positive infections may be diagnosed if standard diagnostic criteria are rigidly applied. Given the limited current evidence evaluating traditional metrics for PJI in the setting of periprosthetic fracture, continued caution should be considered in their interpretation until more robust evidence exists.

## Treatment

Patients in whom preoperative/intraoperative testing confirms infection may be treated with an antibiotic impregnated spacer, with removal of implants and concurrent surgical fixation of the fracture as necessary. The use of custom mobile articulating spacers has been advocated by several authors in the past given the theoretical advantages they offer, including easier mobilization through restoration of leg length, soft-tissue tension, and hip motion.[30,31] A recent multicentered randomized controlled trial by Nahhas et al[32] provided evidenced-based support for their use over static spacers, revealing that the use of articulating spacers was associated with

shorter hospital stays at both the first and second stage or reconstruction (of note, this study did not evaluate patients who had concurrent periprosthetic fracture, only isolated chronic periprosthetic injection). Their data also favor a decreased rate of recurrent infection (25% in the static group versus 15% in the articulating group) and dislocation (10% in the static group and 5% in the articulating group), although neither finding was statistically significant. This latter finding in particular should be weighed against several studies that have suggested that articulating hip spacers may have a higher rate of dislocation in comparison to static spacers, and caution is advised particularly in cases of severe abductor insufficient or acetabular deficiency.[33,34] Although the use of implants in the context of persistent infection is undesirable, fracture union is unlikely without sufficient stabilization. The use of a long-stemmed articulating femoral spacer, bypassing the fracture site by at least two cortical diameters, may allow adequate mechanical support with the addition of cerclage cable fixation (thereby mitigating the need for plate fixation). Plate fixation, however, may remain necessary depending on the fracture pattern, presence of comminution, and achieved mechanical stability. In the context of a well-fixed stem with a low virulence or antibiotic susceptible organism, fracture fixation and implant retention could be considered. The morbidity of explantation (i.e., need for femoral osteotomy for removal) should be weighed again the possibility of persistent infection and/or fracture nonunion.

Static (nonarticulating) spacers may be considered a safer option to an articulating spacer in circumstances where the probability of post-operative instability is considered excessive, or in cases where residual bone stock is inadequate to support an antibiotic coated implant. Of note, although static spacers are effectively Girdlestone hips without a functional articulation, dislocation of the cement spacer from the acetabular cavity or femoral canal into the surrounding tissues may still occur.

For patients who present with periprosthetic fracture, and in whom conflicting or equivocal data from an investigation of periprosthetic infection result, determination of treatment may be based on clinical suspicion and weighed against morbidity involved with staged treatment. Recommendations remain to obtain multiple tissue cultures at the time of surgery. Should one or multiple tissue cultures return positive for bacterial growth, consultation with infection diseases specialists may be considered to determine the role for prolonged antibiotic treatment. As previously noted, evidence regarding specific protocols (eg, duration and route) is limited.[25,26]

For patients with a periprosthetic fracture of an noncemented prosthesis within 3 months of their index surgery, and for whom a low clinical suspicion for infection exists, the most likely cause of femoral loosening if identified is probable insufficient time to allow for osteointegration. Despite this, we recommend that inflammatory markers be obtained and considered in the context of the ICM criteria for early postoperative infection (CRP >100 mg/L or 10 mL/dL).[35] If a high clinical suspicion for infection exists, consideration for preoperative or intraoperative synovial fluid analysis may be made and also interpreted as per the ICM criteria for early postoperative infection (WBC >10 K, PMN% >90%).[35] No literature we are aware of, however, has specifically addressed this scenario to provide normative values, and as such, results should be interpreted with caution. Multiple intraoperative tissue cultures should also still be obtained at the time of revision.

The final scenario potentially encountered would be an acute hematogenous infection or early postoperative infection (<3 months of index surgery)[36] with concurrent periprosthetic fracture, in which the stem appears to be well fixed (Vancouver A, B1, or C). In this situation, surgical fixation of the fracture if indicated (Vancouver A fractures rarely require surgical fixation; B1 and C fractures typically do), with irrigation and débridement, implant retention, and exchange of modular implants and antibiotics (DAIR) may be a reasonable approach. As in most DAIR scenarios, however, onset of symptom to surgical management, bacterial virulence, and host factures all influence probability of success. Patients with onset of infectious symptoms beyond 3 weeks treated with DAIR have rarely been shown in the literature to have successful long-term outcomes, presumably due to biofilm formation.[36,37] Suboptimal factors in any category may theoretically result in successful fracture healing, but also in recurrent or persistent joint infection, potentially necessitating further surgical management.

Evaluation and management of periprosthetic femur fractures after cemented total hip arthroplasty should follow similar principles, although the radiologic evaluation of loosening differs. Although for both cemented and noncemented femoral implants the only definitive evidence of loosening is subsidence or change in position of the implant, attention to radiolucencies at the bone-cement and implant-cement interfaces and the integrity of the cement mantle should be observed (ideally in

comparison with prefracture images).[38] Cemented stem fixation is also more sensitively influenced by stem design after periprosthetic fracture (ie, taper-slip stems versus composite beam stem). Maggs et al[39] have suggested that cement-in-cement revision technique may be an acceptable option for tapered polished stem in which the stem loosens after periprosthetic fracture, but the cement mantle remains well fixed to bone.

## Summary

Both periprosthetic fracture and periprosthetic infection are looming issues on the horizon of total joint arthroplasty. If epidemiologic trends continue as predicted, the incidence of periprosthetic infection and fracture, both independently and concurrently, will likely increase dramatically in the coming decades. Although large-scale studies are scarce, the best current evidence suggests an increased risk of recurrent infection after revision total hip arthroplasty for periprosthetic fracture, greater in comparison to revision hip arthroplasty for other indications. Data currently are sparse to confidently direct management, although the existing literature does provide some guidance. We recommend that further multicentered studies be performed to better refine appropriate parameters to deal with this complex issue.

## References

References printed in **bold type** are those published within the past 5 years.


**1. Sloan M, Premkumar A, Sheth NP: Projected volume of primary total joint arthroplasty in the U.S., 2014 to 2030. *J Bone Joint Surg Am* 2018;100:1455-1460.**

2. Tsiridis E, Pavlou G, Venkatesh R, Bobak P, Gie GA: Periprosthetic femoral fracture around hip arthroplasty: Current concepts in their management. *Hip Int* 2009;19:75-86.

3. Della Rocca GJ, Leung KS, Pape HC: Periprosthetic fractures: Epidemiology and future projections. *J Orthop Trauma* 2011;25:s66-s70.

**4. Giaretta S, Momoli A, Porcelli G, Micheloni GM: Diagnosis and management of periprosthetic femoral fractures after hip arthroplasty. *Injury* 2019;50(suppl 2):S29-S33.**

5. Lindahl H, Malchau H, Herberts P, Garellick G: Periprosthetic femoral fractures classification and demographics of 1049 periprosthetic femoral fractures from the Swedish National Hip Arthroplasty Register. *J Arthroplasty* 2005;20:857-865.

6. Parvizi J, Suh DH, Jafari SM, Mullan A, Purtill JJ: Aseptic loosening of total hip arthroplasty: Infection always should be ruled out. *Clin Orthop Relat Res* 2011;469:1401-1405.

7. Beloosesky Y, Grinblat J, Pirotsky A, Weiss A, Hendel D: Different C-reactive protein kinetics in post-operative hip-fractured geriatric patients with and without complications. *Gerontology* 2004;50:216-222.

**8. Lim SJ, Choi KH, Lee JH, Jung JY, Han W, Lee BH: Different kinetics of perioperative CRP after hip arthroplasty for elderly femoral neck fracture with elevated preoperative CRP. *Biomed Res Int* 2018;2018: 2140105.**

9. Duncan CP, Masri BA: Fractures of the femur after hip replacement. *Instr Course Lect* 1995;44:293-304.

**10. Lee S, Kagan R, Wang L, Doung YC: Reliability and validity of the Vancouver classification in periprosthetic fractures around cementless femoral stems. *J Arthroplasty* 2019;34:S277-S281.**

11. Naqvi GA, Baig SA, Awan N: Interobserver and intraobserver reliability and validity of the Vancouver classification system of periprosthetic femoral fractures after hip arthroplasty. *J Arthroplasty* 2012;27: 1047-1050.

**12. Shohat N, Bauer T, Buttaro M, et al: Hip and knee section, what is the definition of a periprosthetic joint infection (PJI) of the knee and the hip? Can the same criteria be used for both joints? Proceedings of International Consensus on Orthopedic Infections. *J Arthroplasty* 2019;34:S325-S327.**

**13. Schwartz AM, Farley KX, Guild GN, Bradbury TL Jr: Projections and epidemiology of revision hip and knee arthroplasty in the United States to 2030. *J Arthroplasty* 2020;35:S79-S85.**

14. Abdel MP, Watts CD, Houdek MT, Lewallen DG, Berry DJ: Epidemiology of periprosthetic fracture of the femur in 32 644 primary total hip arthroplasties: A 40-year experience. *Bone Joint J* 2016;98-B: 461-467; Erratum in: *Bone Joint J*. 2020;102-B:1782

15. Abdel MP, Houdek MT, Watts CD, Lewallen DG, Berry DJ: Epidemiology of periprosthetic femoral fractures in 5417 revision total hip arthroplasties: A 40-year experience. *Bone Joint J* 2016;98-B:468-474.

16. Kurtz SM, Lau E, Schmier J, Ong KL, Zhao K, Parvizi J: Infection burden for hip and knee arthroplasty in the United States. *J Arthroplasty* 2008;23:984-991.

17. Ong KL, Kurtz SM, Lau E, Bozic KJ, Berry DJ, Parvizi J: Prosthetic joint infection risk after total hip arthroplasty in the Medicare population. *J Arthroplasty* 2009;24(6 suppl):105-109.

18. Chevillotte CJ, Ali MH, Trousdale RT, Larson DR, Gullerud RE, Berry DJ: Inflammatory laboratory markers in periprosthetic hip fractures. *J Arthroplasty* 2009;24:722-727.

19. Bethea JS III, DeAndrade JR, Fleming LL, Lindenbaum SD, Welch RB: Proximal femoral fractures following total hip arthroplasty. *Clin Orthop Relat Res* 1982;170:95-106.

20. Beals RK, Tower SS: Periprosthetic fractures of the femur. An analysis of 93 fractures. *Clin Orthop Relat Res* 1996:238-246.

21. Shah RP, Plummer DR, Moric M, Sporer SM, Levine BR, Della Valle CJ: Diagnosing infection in the setting of periprosthetic fractures. *J Arthroplasty* 2016;31(9 suppl):140-143.

22. Kosashvili Y, Backstein D, Safir O, Lakstein D, Gross AE: Dislocation and infection after revision total hip arthroplasty: Comparison between the first and multiply revised total hip arthroplasty. *J Arthroplasty* 2011;26: 1170-1175.

**23. van den Kieboom J, Tirumala V, Xiong L, Klemt C, Kwon YM: Concomitant hip and knee periprosthetic joint infection in periprosthetic fracture: Diagnostic utility of serum and synovial fluid markers. *J Arthroplasty* 2021;36:722-727.**

24. Badarudeen S, Shu AC, Ong KL, Baykal D, Lau E, Malkani AL: Complications after revision total hip arthroplasty in the Medicare population. *J Arthroplasty* 2017;32:1954-1958.

25. Saleh A, Guirguis A, Klika AK, Johnson L, Higuera CA, Barsoum WK: Unexpected positive intraoperative cultures in aseptic revision arthroplasty. *J Arthroplasty* 2014;29:2181-2186.

26. Jacobs AME, Bénard M, Meis JF, van Hellemondt G, Goosen JHM: The unsuspected prosthetic joint infection : Incidence and consequences of positive intra-operative cultures in presumed aseptic knee and hip revisions. *Bone Joint J* 2017;99-B:1482-1489.

**27. Cizmic Z, Feng JE, Huang R, et al: Hip and knee section, prevention, host related: Proceedings of International Consensus on Orthopedic Infections. *J Arthroplasty* 2019;34:S255-S270.**

28. Ghanem E, Houssock C, Pulido L, Han S, Jaberi FM, Parvizi J: Determining "true" leukocytosis in bloody joint aspiration. *J Arthroplasty* 2008;23:182-187.

29. Munoz-Mahamud E, Bori G, Garcia S, et al: Usefulness of histology for predicting infection at the time of hip revision for the treatment of Vancouver B2 peri- prosthetic fractures. *J Arthroplasty* 2013;28:1247.

30. Younger AS, Duncan CP, Masri BA, McGraw RW: The outcome of two-stage arthroplasty using a custom-made interval spacer to treat the infected hip. *J Arthroplasty* 1997;12:615-623.

31. Biring GS, Kostamo T, Garbuz DS, Masri BA, Duncan CP: Two-stage revision arthroplasty of the hip for infection using an interim articulated prostalac hip spacer: A 10- to 15-year follow-up study. *J Bone Joint Surg Br* 2009;91:1431-1437.

**32. Nahhas CR, Chalmers PN, Parvizi J, et al: Randomized trial of static and articulating spacers for treatment of the infected total hip arthroplasty. *J Arthroplasty* 2021;36:2171-2177.**

33. Wentworth SJ, Masri BA, Duncan CP, Southworth CB: Hip prosthesis of antibiotic-loaded acrylic cement for the treatment of infections following total hip arthroplasty. *J Bone Joint Surg Am* 2002;84-A(Suppl 2):123-128.

34. Leunig M, Chosa E, Speck M, Ganz R: A cement spacer for two-stage revision of infected implants of the hip joint. *Int Orthop* 1998;22:209-214.

**35. Chotanaphuti T, Courtney PM, Fram B, et al: Hip and knee section, treatment, algorithm: Proceedings of International Consensus on Orthopedic Infections. *J Arthroplasty* 2019;34:S393-S397.**

36. Volpin A, Sukeik M, Alazzawi S, Haddad FS: Aggressive early debridement in treatment of acute periprosthetic joint infections after hip and knee replacements. *Open Orthop J* 2016;10:669-678.

37. Tsang SJ, Ting J, Simpson AHRW, Gaston P: Outcomes following debridement, antibiotics and implant retention in the management of periprosthetic infections of the hip: A review of cohort studies. *Bone Joint J* 2017;99-B:1458-1466.

38. Gruen TA, McNeice GM, Amstutz HC: Modes of failure of cemented stem-type femoral components: A radiographic analysis of loosening. *Clin Orthop Relat Res* 1979;141:17-27.

**39. Maggs JL, Swanton E, Whitehouse SL, et al: B2 or not B2? That is the question: A review of periprosthetic fractures around cemented taper-slip femoral components. *Bone Joint J* 2021;103-B:71-78.**