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Fracture-Related Infection: From Diagnosis to Eradication and Fracture Union A Contemporary Narrative Review and Practical Treatment Algorithm

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Abstract

Fracture-related infection (FRI) remains one of the most demanding complications in orthopaedic trauma because successful treatment requires simultaneous control of infection, preservation or restoration of mechanical stability, fracture union, and recovery of a durable soft-tissue envelope. The international FRI consensus definition has transformed the field by separating confirmatory from suggestive diagnostic criteria and by discouraging the extrapolation of prosthetic joint infection algorithms to the fracture population. Nevertheless, substantial uncertainty persists regarding the optimal use of debridement, antibiotics and implant retention (DAIR), the timing of implant exchange, the role of local antimicrobial therapy, and the duration and composition of systemic treatment. This narrative review integrates current consensus recommendations with recent evidence on diagnostic biomarkers, microbiology, histopathology, biofilm-active therapy and surgical outcomes. A practical treatment algorithm is proposed. The central principle is that elapsed time alone should not determine implant retention. Instead, treatment should be individualized according to fracture healing, fixation stability and reduction, quality of debridement, soft-tissue condition, host status, presumed biofilm maturity, implant type, pathogen profile and previous treatment. When the fracture has healed, implant removal with radical debridement is generally the most direct strategy. In an unhealed fracture, DAIR may be appropriate when stable fixation, acceptable reduction, viable soft tissues and adequate source control can be achieved; otherwise, implant exchange is favored. Local antimicrobial delivery is an adjunct to, not a substitute for, meticulous surgery. Multidisciplinary care and standardized sampling remain essential. The objective is not simply to sterilize an implant but to create the biological and mechanical conditions in which infection control and fracture union can coexist.

Keywords: Fracture-Related Infection, Fri, Biofilm, Dair, Fracture Fixation, Debridement, Implant Retention, Implant Exchange, Local Antibiotics, Rifampicin, Nonunion.

Introduction

Fracture-related infection is not simply an infection occurring in the vicinity of an orthopaedic implant. It is a distinct clinical entity in which infection develops in a biological environment already compromised by trauma, devascularization, fracture instability, soft-tissue injury and, frequently, repeated surgery. These features explain why treatment principles derived from periprosthetic joint infection cannot be transferred uncritically to FRI. In arthroplasty, the implant is intended to remain permanently; in fracture fixation, the implant is a temporary mechanical tool whose purpose is to permit bone healing.

The consequences of FRI extend far beyond microbiological eradication. Persistent infection may lead to delayed union or nonunion, bone loss, soft-tissue breakdown, repeated hospital admissions, prolonged antimicrobial exposure, impaired mobility, loss of employment and, in the most severe cases, amputation. The therapeutic endpoint must therefore be broader than

infection control alone: a healed, stable, functional limb with durable soft-tissue coverage and an acceptable burden of treatment.

The introduction of an international consensus definition represented a major advance. Confirmatory criteria identify infection with high specificity, whereas suggestive criteria indicate the need for further investigation. Subsequent validation demonstrated excellent diagnostic performance of the confirmatory criteria. More recent studies have refined the contribution of histology, tissue culture, sonication and emerging biomarkers, but no single blood test can currently rule FRI in or out [1-5].

This review focuses on the clinically decisive question: once FRI is suspected or confirmed, how should the surgeon choose between implant retention, implant removal and implant exchange? We propose a pragmatic algorithm based on the biology of biofilm, the mechanics of fracture healing and the feasibility of radical source control.

Definition and Diagnostic Framework

The consensus definition divides diagnostic findings into confirmatory and suggestive criteria. A single confirmatory criterion is sufficient to establish FRI, whereas suggestive findings should trigger further investigation. This distinction is clinically useful because postoperative inflammation, pain and radiographic abnormalities are common after fracture surgery and are not specific for infection.

Table 1: Practical summary of confirmatory and suggestive FRI criteria. Interpretation should follow the current FRI consensus framework and the clinical context

Confirmatory criteria	Suggestive criteria
Fistula, sinus tract or wound breakdown communicating with bone/implant	Pain, redness, swelling, local warmth, fever; persistent/new wound drainage
Purulent drainage or pus at surgery	Raised CRP, ESR or WBC count
Phenotypically indistinguishable pathogens from ≥2 separate deep specimens	Single positive deep culture, particularly when clinical context is suspicious
Microorganisms demonstrated in deep tissue by histopathology	Radiological or nuclear imaging features compatible with infection
Quantitative acute inflammation on histology in appropriate chronic/late cases	Delayed union/nonunion, implant loosening, osteolysis or sequestration

Biomarkers, Imaging, Microbiology and Histology

Serum Biomarkers

Routine inflammatory markers are useful as pieces of a diagnostic puzzle, but their limitations must be explicit. CRP, ESR and leukocyte count may rise because of trauma and surgery and may remain normal in chronic or indolent infection. Contemporary reviews therefore regard them as suggestive rather than confirmatory tests. Serial trends can support clinical assessment and may be useful during follow-up, but an isolated normal value must not be used to exclude FRI. Novel blood-based approaches, including inflammatory cell-surface markers, cytokine signatures and omics-derived panels, are promising. Recent prospective data suggest that neutrophil CD64, CD66b and serum thioredoxin may outperform conventional markers in selected postoperative settings. These results are hypothesis-generating rather than sufficient for routine replacement of consensus diagnostics [6-10].

Imaging

Plain radiographs remain essential for assessing union, alignment, implant integrity, loosening, osteolysis and sequestration. CT is particularly useful for defining nonunion, cortical defects and sequestra, while MRI provides detailed soft-tissue and marrow assessment but may be limited by metal artefact. Nuclear techniques, including labelled leukocyte imaging and FDG-PET/CT, can contribute when anatomy and conventional imaging are inconclusive. Imaging should answer two questions: how likely is infection, and what anatomy must be addressed surgically?

Microbiology and Histopathology

High-quality sampling is a surgical act. Multiple separate deep tissue specimens should be obtained before antimicrobial therapy is started whenever the patient is clinically stable. Superficial swabs and samples from sinus tracts are inadequate substitutes. At least five deep samples are commonly recommended, each obtained with separate instruments, with prolonged incubation according to local microbiology protocols. Sonication of removed implants can increase pathogen recovery and may be especially helpful in polymicrobial or previously treated infection.

Histology provides complementary information rather than competing with culture. Quantitative neutrophil assessment is highly specific in chronic or late FRI, whereas culture generally offers greater sensitivity and supplies susceptibility data. A 2026 cohort of 430 consensus-confirmed FRI cases showed that microbiology and host inflammatory response provide independent information; simultaneous culture and histological positivity was associated with a higher risk of treatment failure. The practical implication is that FRI should be interpreted as an interaction between pathogen, biofilm and host response rather than as a culture result alone.

Biofilm and the Biological Rationale for Treatment

Biofilm is central to implant-associated FRI. Once adherent to a surface, bacteria enter a structured community embedded in extracellular matrix, with altered metabolism and markedly reduced susceptibility to antimicrobial therapy and host immune clearance. Biofilm maturity is a continuum, not a switch that occurs on a predetermined postoperative day. This is why rigid time thresholds are clinically attractive but biologically incomplete. The probability of successful implant retention decreases as biofilm matures and as the opportunity for complete debridement diminishes. However, elapsed time must be interpreted together with symptom duration, fixation type, soft-tissue condi-

tion, bacterial phenotype, prior antimicrobial exposure and previous surgery. The surgeon should therefore ask not only 'How old is the infection?' but 'Can I remove the infected and necrotic burden while preserving a mechanically sound construct and viable biology?'

Fundamental Surgical Principles

Successful treatment begins with source control. Radical debridement means excision of nonviable skin, subcutaneous tissue, fascia, muscle and bone until viable, perfused tissue is reached. Necrotic bone, loose implants and foreign material that cannot contribute to stable reconstruction should not be preserved simply to avoid a larger procedure. Mechanical stability is part of infection treatment. Excessive motion impairs vascularity and union and may perpetuate inflammation, while stable fixation supports revascularization and bone healing. Retaining an unstable construct is therefore biologically contradictory even if the implant itself appears technically salvageable[11-15].

Dead space should be anticipated rather than discovered at the end of debridement. Local antimicrobial carriers may deliver high concentrations at the site of infection and simultaneously assist dead-space management. Their role is adjunctive: no carrier can compensate for retained necrotic tissue or inadequate debridement. Definitive soft-tissue closure should be achieved as early as reasonably possible with healthy, vascularized tissue. When local tissues are inadequate, early collaboration with plastic/reconstructive surgery is preferable to repeated temporizing procedures.

DAIR: When Retention is Reasonable

DAIR—debridement, antimicrobial therapy and implant retention—is attractive because it preserves fracture stability and avoids the biological and mechanical insult of fixation exchange. It is not, however, a 'minor' treatment. A successful DAIR requires aggressive exposure, meticulous debridement, high-quality sampling, exchange of removable components when appropriate, irrigation, dead-space management, reliable closure and a coherent biofilm-active antimicrobial plan.

The best candidates have an unhealed fracture with stable fixation and acceptable reduction, a construct that still serves a mechanical purpose, a short or clinically favorable infection history, viable soft tissues that can be definitively closed, and a pathogen for which effective antimicrobial treatment is available. Conversely, gross instability, unacceptable reduction, implant loosening, necrotic bone that cannot be adequately addressed without dismantling the construct, mature or recurrent infection, poor soft tissues, difficult-to-treat organisms and previous failed retention strategies shift the balance toward exchange. Intramedullary fixation deserves particular caution because complete debridement of the medullary canal and the implant surface is inherently difficult while the nail remains in situ. Retention may still be considered in selected situations, but the threshold for exchange should be lower when adequate source control cannot otherwise be achieved.

Implant Removal or Exchange

Healed Fracture

When the fracture is united, the central mechanical reason for retaining fixation has disappeared. Implant removal combined with radical debridement, deep sampling and management of residual bone and soft-tissue defects is therefore generally the most straightforward curative strategy. The surgeon must nevertheless confirm that union is sufficiently robust to tolerate removal and that any resulting defect does not create a new mechanical problem.

Unhealed Fracture

When the fracture is not healed and retention is unsuitable, fixation must be exchanged rather than simply removed. The reconstructive plan should be decided before debridement and should integrate the anticipated bone defect, soft-tissue coverage, local antimicrobial strategy and required mechanical environment.

A one-stage exchange can be considered when radical debridement is achievable, stable definitive fixation can be obtained, the soft-tissue envelope can be closed or reconstructed reliably, and the multidisciplinary team considers the microbial and host situation manageable. A staged strategy is more appropriate when the extent of infection is uncertain, soft tissues are severely compromised, bone loss is substantial, the host requires optimization, or definitive stable reconstruction cannot safely be performed at the index procedure.

Local and Systemic Antimicrobial Therapy

Empirical systemic antibiotics should generally be withheld until deep samples have been obtained, unless the patient is septic or clinically unstable. After sampling, broad empirical coverage should reflect local epidemiology, previous microbiology and individual risk factors, and should then be narrowed promptly once culture and susceptibility results are available. Infectious-disease or microbiology input is particularly valuable in polymicrobial, resistant, culture-negative and recurrent cases.

In retained-implant strategies, biofilm-active therapy is conceptually important. Rifampicin has a major role against susceptible staphylococcal biofilm, but it must not be used as monotherapy because resistance can emerge rapidly. It is usually introduced as part of an active combination after adequate source control and once the wound is controlled. The evidence base specific to FRI remains less robust than that for prosthetic joint infection, so treatment should be individualized rather than copied mechanically from arthroplasty protocols.

For Gram-negative organisms, fluoroquinolones may provide biofilm-active oral therapy when the isolate is susceptible and patient-specific contraindications are absent. Duration is determined by the surgical strategy, implant status, pathogen and host rather than by a single universal number. In selected situations where eradication is not initially achievable but fixation must remain until union, suppressive antimicrobial therapy can be used as a bridge to fracture healing, followed by implant removal and definitive treatment.

Practical Treatment Algorithm

The following algorithm summarizes the proposed decision pathway. It deliberately avoids using elapsed time as the sole determinant of DAIR eligibility. The first decision is fracture healing; in an unhealed fracture, the second is whether the existing construct and biological environment can realistically support both infection control and union.

Table 2: Proposed Decision-Making Algorithm for Fracture-Related Infection (FRI)

SUSPECTED / CONFIRMED FRACTURE-RELATED INFECTION **Apply FRI consensus criteria • define fracture status • assess fixation, bone, soft tissues, host and microbiology**		
↓ IS THE FRACTURE UNITED? ↓ The first therapeutic branch point		
YES — FRACTURE UNITED Fixation has completed its mechanical role		NO — FRACTURE NOT UNITED Stability remains essential for healing
IMPLANT REMOVAL + RADICAL DEBRIDEMENT ≥5 deep samples ± histology/sonication Dead-space management ± local antimicrobials Definitive soft-tissue coverage Targeted systemic therapy		CAN THE CURRENT FIXATION BE SAFELY RETAINED? Stable fixation? • acceptable reduction? • radical debridement feasible? • viable soft tissues? • treatable pathogen? • acceptable host?
GOAL INFECTION ERADICATION + PRESERVED UNION	YES — RETENTION FAVOURABLE Proceed to DAIR	NO — RETENTION UNFAVOURABLE Proceed to implant exchange
DAIR IS NOT A TIME-ONLY DECISION Time modifies probability; mechanical and biological feasibility determine strategy	DAIR Radical debridement Multiple deep samples Irrigation Dead-space management ± local AB Definitive closure Biofilm-active targeted therapy	IMPLANT EXCHANGE Remove failing/inadequately debridable construct Restore mechanical stability Repeat radical source control
RETENTION FAVOURED BY Stable construct • good reduction • viable tissues • first episode • debridement feasible	EXCHANGE FAVOURED BY Loosening/instability • poor reduction • necrotic bone • recurrent infection • failed DAIR • difficult source control	IF EXCHANGE IS REQUIRED: ONE-STAGE OR STAGED? Choose according to debridement completeness, bone/soft-tissue defects, host, pathogen and feasibility of immediate stable reconstruction
AFTER DAIR Monitor infection control + fracture progression → achieve union → consider implant removal after healing	ONE-STAGE EXCHANGE Radical debridement achievable Immediate stable fixation possible Definitive soft-tissue closure possible	STAGED EXCHANGE Uncertain/high infection burden Major bone or soft-tissue defect Host optimization needed Definitive fixation not immediately safe
FINAL OBJECTIVE: INFECTION CONTROL + FRACTURE UNION + STABLE FUNCTIONAL LIMB If curative reconstruction is temporarily impossible: individualized suppression until union or planned salvage; amputation only in exceptional non-reconstructable situations.		

Implant retention should not be determined by elapsed time alone. The feasibility of radical debridement, mechanical stability, fracture reduction, soft-tissue viability, host status, implant type, pathogen profile and availability of an effective antimicrobial strategy must be considered together.

Clinical Interpretation: DAIR is favored when fixation remains mechanically sound and biologically useful, reduction is acceptable, thorough debridement and durable closure are achievable, and effective biofilm-active therapy is available. Implant exchange is favored by loosening or instability, unacceptable reduction, inaccessible or extensive infected interfaces, necrotic bone, compromised soft tissues, recurrent infection, difficult-to-treat organisms, or previous failed retention. In every branch, surgical source control, fracture stability and soft-tissue reconstruction should be planned as a single strategy [16-20].

Table 3: Factors That Should Lower the Threshold for Implant Exchange

Domain	Favors retention	Favors exchange/removal
Mechanical	Stable construct; acceptable reduction	Loosening, instability, implant failure, unacceptable reduction
Biological	Viable bone and soft tissue; achievable debridement	Necrotic bone, inaccessible infected interface, major dead space
Soft tissue	Early durable closure possible	Persistent drainage, exposed implant, major defect requiring complex reconstruction

Infection	Short/favorable history; first presentation	Chronic/recurrent infection; mature biofilm likely; previous failed DAIR
Microbiology	Susceptible organism with active regimen	Difficult-to-treat/resistant organism or no reliable biofilm-active option
Implant	Accessible plate/screws with debridable interface	Intramedullary construct or implant configuration limiting source control
Host	Optimizable host	Severe uncompensated comorbidity, poor vascularity or inability to tolerate repeated failure

Failure, Recurrence and Suppressive Strategies

Treatment failure should trigger a structured reassessment rather than automatic repetition of the previous operation. The team should revisit the diagnosis, sampling quality, organism and resistance profile, fracture stability, union progression, adequacy of prior debridement, residual dead space, soft-tissue coverage and host factors. Repeating DAIR in a setting in which the reasons for the first failure remain unchanged is unlikely to convert an unfavorable biology into a favorable one.

Suppressive therapy is not synonymous with therapeutic failure. In a carefully selected patient with an unhealed fracture and a construct that must temporarily remain for stability, suppression may be a deliberate staged strategy: control clinical infection, achieve union, then remove the implant and perform definitive source control. The endpoint and contingency plan should be explicit from the beginning.

Multidisciplinary Care and Referral

Complex FRI is best treated by a dedicated multidisciplinary team including orthopaedic trauma or limb-reconstruction surgeons, microbiology/infectious-disease specialists, plastic surgeons when required, radiology, and rehabilitation. The value of the team is not administrative; it lies in coordinating sampling, antimicrobial stewardship, reconstruction, host optimization and follow-up. Recurrent infection, segmental bone loss, major soft-tissue defects, resistant organisms and failed previous treatment should prompt early referral to a center experienced in bone and joint infection [21].

Discussion

The central tension in FRI treatment is unique: the foreign body that supports biofilm may simultaneously be the device that permits the fracture to heal. Removing every implant at the first sign of infection may compromise stability; retaining every stable implant may preserve a biofilm that cannot be eradicated. The correct strategy lies between these extremes.

This explains why a time-only classification is insufficient. Early presentation is favorable mainly because biofilm is less mature, tissues may be more salvageable and the original construct may still be mechanically sound. Conversely, some later infections can still be treated with retention when the biology and mechanics are favorable, while some very early infections require exchange because fixation is unstable or source control is impossible. Time modifies probability; it does not replace surgical judgment.

Recent diagnostic evidence reinforces the same multidimensional approach. Conventional blood markers have limited stand-alone accuracy. Culture, histology and sonication each interrogate a different aspect of the disease. The 2026 data linking combined microbiological and histological positivity with poorer outcome suggest that the intensity of both pathogen burden and host inflammatory response may contain prognostic information. Future FRI stratification may therefore evolve from a binary infected/not-infected framework toward biologically meaningful phenotypes.

Antimicrobial therapy should likewise be subordinated to source control rather than used to compensate for inadequate surgery. Rifampicin is an important example: its anti-biofilm properties make it valuable in susceptible staphylococcal implant-associated infection, but its effectiveness depends on combination therapy and adequate surgical control. The same principle applies to local antibiotics: high local concentrations are attractive, particularly in dead space, but they cannot sterilize retained necrotic tissue.

Ultimately, successful FRI care is the restoration of a functional biological system. Eradication, union, stability and soft-tissue integrity are not separate endpoints. They are components of the same reconstructive objective.

Conclusions

FRI should be approached as a combined infectious, biological and mechanical failure. Diagnosis rests on consensus confirmatory and suggestive criteria, with microbiology and histology at the center and serum biomarkers playing a supportive role. Surgical source control remains the cornerstone of treatment. Once fracture healing is established, implant removal usually simplifies eradication. In an unhealed fracture, implant retention is appropriate only when fixation is stable, reduction is acceptable, radical debridement and durable soft-tissue closure are feasible, and an effective antimicrobial strategy exists. When these conditions are absent, exchange fixation should be favored. The decision must not be reduced to a rigid time threshold. The most useful question is whether the chosen construct, host and biological environment can simultaneously support infection control and fracture union.

Key Clinical Messages

- FRI is a distinct entity; PJI algorithms should not be copied uncritically.
- A confirmatory consensus criterion establishes infection; suggestive criteria require further investigation.
- CRP, ESR and WBC cannot independently rule FRI in or out.
- Obtain multiple deep samples before antibiotics whenever the patient is stable.
- Debridement quality and fracture stability are inseparable components of source control.

- DAIR is not defined by time alone.
- Local antibiotics complement but never replace radical debridement.
- Rifampicin should never be used alone for staphylococcal implant-associated infection.
- If the fracture is healed, removal of unnecessary fixation generally improves the possibility of eradication.
- If the fracture is unhealed and fixation cannot safely be retained, exchange must restore stability while enabling source control.
- Early definitive soft-tissue coverage and multidisciplinary management are central to success.

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