

INFOGRAPHIC

# **Fracture-related infection**

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### Background

Fracture-related infection (FRI) carries a substantial burden of disease and socioeconomic costs.<sup>1-3</sup> The incidence of FRI is 1% to 2% in closed fractures and can reach 30% in open fractures.<sup>1</sup> Until recently, amputation and recurrence rates remained high.<sup>2,4</sup> With the publication of international consensus documents,<sup>4,5</sup> an evidence-based overview of diagnosis and management has been provided, which should improve treatment outcomes.

### **Pathology**

The pathology of FRI is multifactorial; bacterial infection and fracture instability are interdependent and fundamental in FRI.<sup>6,7</sup> Biofilm formation, canalicular invasion,<sup>8</sup> intracellular infection,<sup>9</sup> and formation of staphylococcal abscess communities<sup>10</sup> are the key niches occupied by bacteria. A vicious cycle between instability with ongoing soft-tissue trauma, compromised neovascularity, and osteolysis creates a supportive environment for bacteria, promoting the development of FRI or hindering its eradication.<sup>6</sup>

### Diagnosis

Confirmatory criteria include fistula or sinus tract, purulent drainage or pus, microbial growth in two or more deep tissue samples, and histological evidence of pathogens and inflammation in peri-implant tissue.<sup>4,5</sup> Suggestive criteria include clinical signs such as: erythema; swelling; persistent, increasing, or new-onset wound drainage; radiological or nuclear imaging signs; increased serum inflammatory markers; and microbial growth in a single deep tissue sample.<sup>5,7</sup>

#### Management

A consensus-derived management algorithm has been developed and should be led by a multidisciplinary team.<sup>5,7</sup> Based on three basic principles, consisting of exchange, retention, or removal of the indwelling implant, the preferred strategy depends on host physiology, time interval between fracture fixation and FRI manifestation, anatomical localization, and causative pathogen. For implant retention, the stability of the construct and the ability to perform proper debridement are critical, considering the implant type and softtissue conditions.<sup>7</sup>

### **Prevention**

Appropriate use of prophylactic antibiotics is crucial to prevent FRI. In closed injuries, perioperative antibiotic prophylaxis limited to a single dose is recommended. In open fractures, prophylactic antibiotic administration should not exceed 24 hours for Gustilo-Anderson types I and II and 72 hours for Gustilo-Anderson type III fractures.<sup>5,7</sup> Early debridement, soft-tissue management, and stable fracture fixation are cornerstones of management.<sup>5</sup>

Follow-up of FRI should be planned in collaboration with a multidisciplinary team, for a minimum of 12 months after the cessation of surgical and antibiotic therapy.<sup>11</sup>

Future perspectives for prevention and management of FRI include: antimicrobial coated implants; osteoinductive antibioticloaded biomaterials; and bacteriophage and enzybiotic therapy. All these therapies consider the global threat of antibiotic resistance and target mechanisms of antimicrobial tolerance such as biofilm formation.

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# Fracture-Related Infection



### Diagnosis

### **Confirmatory criteria**

- ≥ 1 confirms
- 1. Fistula or sinus tract
- 2. Purulent drainage or pus
- **3.** Microbial growth in 2 deep tissue samples
- **4.** Histology: proof of pathogen and inflammation in peri-implant tissue

### Suggestive criteria:

- Erythema
- Swelling
- Radiological signs





# Prevention

### **Closed fractures**

- Perioperative antibiotics
- Soft-tissue care
- Early stable fixation

# **Open fractures**

Orthoplastic care: Early stable fixation and timely soft-tissue coverage

Antibiotic prophylaxis

- GA type I/II 24 hours
- GA type III 72 hours

# Follow-up

Minimum **12 months** follow-up after cessation of surgical and antibiotic therapy



Multidisciplinary team approach



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- S. Baertl: Designed, created, and revised the infographic.
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