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Non-union after plate fixation

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KEYWORDS

Non-union
Revision surgery
Fracture healing

ABSTRACT

Approximately a third of patients presenting with long-bone non-union have undergone plate fixation as their primary procedure. In the assessment of a potential fracture non-union it is critical to understand the plating technique that the surgeon was intending to achieve at the primary procedure, i.e. whether it was direct or indirect fracture repair. The distinction between delayed union and non-union is a diagnostic dilemma especially in plated fractures, healing by primary bone repair. The distinction is important as non-unions are not necessarily part of the same spectrum as delayed unions. The etiology of a fracture non-union is usually multifactorial and the factors can be broadly categorized into mechanical factors, biological (local and systemic) factors, and infection. Infection is present in ~40% of fracture non-unions, often after open fractures or impaired wound healing, but in 5% of all non-unions infection is present without any clinical or serological suspicion. Methods to improve the sensitivity of investigation in the search of infection include the use of; sonication of implants, direct inoculation of theatre specimens into broth, and histological examination of non-union site tissue. Awareness should be given to the potential anti-osteogenic effect of bisphosphonates (in primary fracture repair) and certain classes of antibiotics. Early cases of delayed/non-union with sufficient mechanical stability and biologically active bone can be managed by stimulation of fracture healing. Late presenting non-union typically requires revision of the fixation construct and stimulation of the callus to induce fracture union.

Introduction

Fracture non-union is a rare but clinically challenging condition. A population-based analysis found an overall risk of non-union per facture to be 1.9%, rising to 9% in the peak age group of 25–44 years [1]. Approximately a third of patients presenting with long-bone non-union have undergone plate fixation as their primary procedure [2].

In the assessment of a potential fracture non-union it is critical to understand the plating technique that the surgeon was intending to achieve at the primary procedure [2] (i.e. rigid fixation and primary bone healing or biological fixation and secondary bone healing). Primary plate fixation can be achieved using a number of different techniques, depending on the fracture location, morphology, and indication for surgery [3]. The rigid fixation techniques include: 1) tension band plating; 2) Buttress or antiglide, e.g. for AO B-type fractures [4]; 3) neutralisation plates, e.g. for fixation of rotational fractures of the fibula [5]; and 4) compression plating. All of these rigid plating and primary bone healing techniques usually require a certain degree of soft tissue stripping which can have a major bearing on the treatment if the fractures becomes a non-union. In contrast, biological or bridge plating is indicated for fixation of comminuted diaphyseal fractures to minimise soft tissue stripping and used as "an internal external-fixator" to induce secondary bone healing [6]. The key to these internal fixators is the locking mechanism of the screw in the implant, which provides angular stability [7].

Diagnostic dilemma

The distinction between delayed union and non-union is a diagnostic dilemma [8], especially after a fracture has been plated. Radiographic techniques are commonly used to infer biological activity at the fracture site and indicate if there is fracture union or non-union. The radiographic grading systems include the Weber and Cech system [9] and the Callus Index [10]. More recently classifications such as the RUST scale have been described and validated for intramedullary fixation of tibia [11,12]. All of these classifications have in common, the detection of callus on plain radiographs as the key diagnostic feature. However, callus formation is not seen in during primary bone healing, i.e. rigid plating and even with 'biological' plating, as, unfortunately, plates often obscure both periosteal and endosteal callus. Clinical assessment of mobility at the fracture union has also been described as a surrogate of healing potential [13] but is not useful for determining the progression of healing of a plated fracture.

As a consequence of the lack of a robust diagnostic technique, there are many definitions for fracture non-union, which include:

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1. Fracture not healed in the expected time with no progression on plain radiographs,
2. Cessation of fracture healing process
3. Not healed within nine months
4. Need for additional procedures to achieve union.

The first three of these criteria rely on radiographic features, such as bridging callus [12] to determine if fracture healing is progressing. Yet, as mentioned above, plated fractures undergoing direct bone healing, often do not have any visible callus. Thus, in plated fractures, the requirement for further surgery (e.g. re-fixation or bone grafting) has been used as a diagnostic criterion for non-union. However, this can be erroneous, as failure to hold a fracture to union depends on: 1) the fatigue strength of the implant (which is dependent on geometry and type of material of the plate); 2) the rate of healing of the fracture; and 3) the amount of load (both magnitude and number of cycles) taken by the implant and bone fragments, which depends on the technique of fracture fixation. The amount of load on a plate has been measured using implantable telemetric devices fixed onto plates [14] and this may be a method for detecting failure of the fracture repair process.

If the fracture has progressed to non-union, the plate will fail eventually, and additional procedures will be needed. If, however the fracture is healing slowly (i.e. a delayed union), the plate may fail before union and would therefore be classified as a non-union even though the healing processed has not ceased (Fig. 1). This would be more likely with a delayed union in a heavy individual with a relatively small plate. From the patient's perspective, further treatment is needed in both the 'true' non-union and the delayed union where the plate has failed, but as the repair process has ceased in the established non-union the patient may benefit from additional stimulation of the fracture.

Etiology

The etiology of a fracture non-union is usually multifactorial, which can be broadly categorized into mechanical factors, biological (local and systemic) factors, and infection. It has been shown that a high percentage of fracture non-unions have more than one cause, with infection presence in ~40% of cases. Of note, 5% of all non-unions were found to have the presence of infection despite a lack of clinical or serological suspicion [2]. This is of particular importance in non-unions following plating especially when an open plating procedure has been performed. The diagnostic difficulty of detecting infection clinically, serologically, or through traditional microbiological methods, such as direct culture is related to the presence of the bacterial biofilm. The formation of a biofilm by bacteria is an adaptive state following adherence to host tissue and biomaterials, characterised by the production of an exopolysaccharide matrix, known as a glycocalyx, and transition to a sessile phenotype [15]. These adaptations allow the bacteria to survive in low nutrient environments and evade host immune defenses but present an obstacle in the diagnosis of infected non-unions. The glycocalyx produces a zone of relative immune deficiency as the cellular components of the host immune system are unable to penetrate the extracellular matrix thus minimising any evidence of an immune response to the infection. The bacterial adherence and glycocalyx reduce the yield of swabs and samples obtained for culture, with the sessile phenotype associated with poor growth rates [16]. Methods to circumvent these issues and improve sensitivity of bacterial culture include the use of sonication to liberate a greater proportion of organisms adherent to tissue and implant samples [17] and direct inoculation of theatre specimens into broth to drive the bacteria to a more metabolically active planktonic state [18]. A further diagnostic adjunct is histological examination of non-union site tissue. The presence of >1 neutrophil polymorph seen per high power field on average, after examination of at least ten high power fields has been shown to have 100% positive predictive value for the diagnosis of infected fracture non-unions [19]. Although more recently in the arthroplasty field has been updated to more than 5 neutrophils per high power field [20].

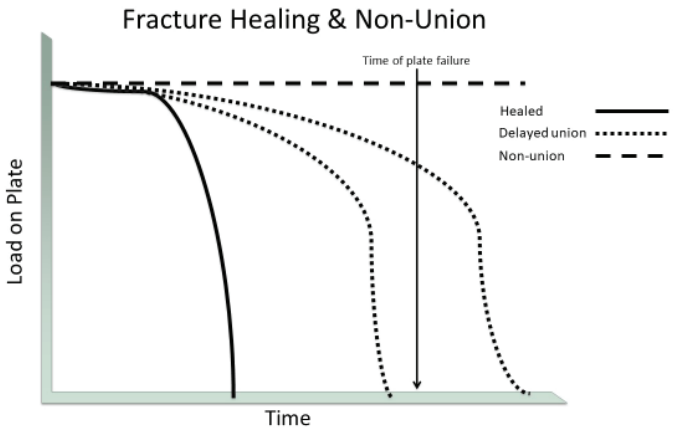

Fig. 1. Plate failure according to time from fixation and progress of fracture union.

Mechanical factors can influence fracture non-union in a variety of ways depending on the original plating technique. Primary bone healing plating techniques require absolute stability and thus mechanical factors need to be addressed if stability has been lost in order to prevent (or treat) a hypertrophic union. The biological plating techniques require relative stability and so mechanical factors can influence fracture healing in two ways: 1) Excessive motion resulting in a hypertrophic non-union; 2) Excessive rigidity resulting in a lack of stimulus for callus formation. This is primarily dependent on the working length, plate material and plate geometry. Attention to the working length of a plate, when used in a biological fashion, is crucial for allowing the optimal levels of inter-fragmentary motion for fracture union [21,22]; too small a working length will result in non-union and early plate failure. Working lengths of under 30 mm are unlikely to have sufficient micro movement at the fracture site to induce fracture callus with stainless steel plates. Working lengths of 50 mm and 70 mm typically need weight bearing of 200 N and 300 N to provide a good mechanical environment for healing [22]. Loss of screw fixation following plate fixation (all techniques) must always be addressed due to the loss of both axial, angular and rotatory stability; unlike loss of screw fixation with intramedullary devices where only rotatory stability is lost.

Biological failure can be a result of both local factors, such as avascular bone with a gap (local), and modifiable and non-modifiable systemic host factors, such as medications and age, respectively [23]. Of particular relevance to plating are bisphosphonates which have been shown to inhibit primary fracture repair and consequently patients on bisphosphonates who sustain a fracture that has been rigidly fixed are prone to non-union [24].

Treatment algorithm (Fig. 2)

1) General considerations when formulating a treatment strategy for fracture non-union following plate fixation

a) Host factors

Modifiable host risk factors for non-union should be addressed in the pre-operative period [25–27]. Chronic diseases (e.g. diabetes mellitus, chronic renal failure, diabetes mellitus, hypothyroidism, anaemia, peripheral vascular disease) should be optimised before any non-union surgery and the use of medications (Non-steroidal anti-inflammatory medication (NSAIDs) and corticosteroids) alcohol consumption and tobacco should be stopped if at all possible