Key Clinical Topics in Trauma

Edited by
Keith Porter • Ian Greaves
Derek Burke

Contents

Abnormal fracture healing

Key points

- Abnormal fracture healing remains a problem in trauma patients despite advances in initial and definitive management
- Both delayed and nonunion of fractures have significant direct and indirect costs to health care providers and the patient
- Promoting smoking cessation, dietary supplementation and early weight bearing will reduce the risk of fracture nonunion

Epidemiology

Despite advances in orthopaedic practice and a greater understanding of fracture biology, the incidence of fracture nonunion remains a concern. Fracture nonunion has direct cost implications for the health services and indirect costs for patients through loss of earnings and additional social care. In the United Kingdom, there are approximately 850,000 new adult fractures each year. In Scotland between 2005 and 2010, the overall incidence of nonunion was 18.4/100,000 per annum, with a peak at 30–40 years of age and a higher incidence in males.

Nonunion in children is considered a rare complication. The immature skeleton has a robust periosteal layer, sufficient vascularity and enhanced healing potential. The risk of nonunion per fracture is low throughout childhood with a risk of approximately 1 in ≤500 per fracture in boys aged under 14 years and in girls of all ages; however, nonunion risk increases to approximately 1 in 200 fractures for older teenage (15–19 years) boys.

Definitions

A fracture is considered to have healed when the patient is pain free during physiological loading of the injured bone; there is no translation at the fracture site on examination and there is adequate consolidation on radiographs. The majority of fractures are expected to unite within 3–4 months of injury in adults. Delayed union is when the fracture heals at a slower rate than expected for a particular bone.

A nonunion exists when repair is not complete within the period expected for a specific fracture, cellular activity at the fracture site ceases and there are no visible progressive signs of healing. According to the United States Food and Drug Administration, a nonunion is established when a minimum of 9 months has elapsed since injury and the fracture show no visible progressive signs of healing for 3 months. It is impractical to apply this to every patient, and the clinician must judge each case individually. Delayed union, nonunion and pseudoarthrosis represent part of the spectrum of bone repair. It is difficult to predict which patient or fracture type will progress to nonunion and an even greater challenge to prevent it.

Diagnosis

- Clinical examination is the cornerstone of diagnosis and will identify tenderness at the fracture site, persistent movement at the fracture site and pain while applying physiological stresses or while weight bearing
- Plain radiographs provide additional information (**Figure 3**). Assessing for fracture consolidation involves evaluating the extent of bridging callous across the

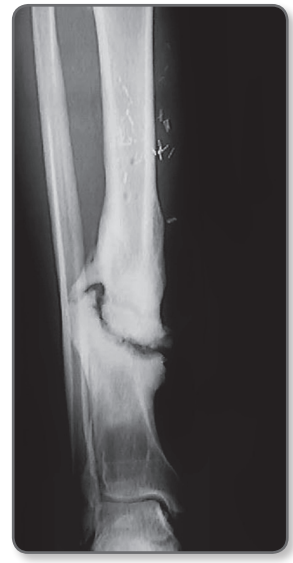

Figure 3
Nonunion of a tibial fracture.

fracture site in two orthogonal views and demonstrating diminishing fracture lines
- High-resolution CT is more sensitive and is used in cases where nonunion is suspected
- Dual-energy X-ray absorptiometry scanning is useful for distinguishing between hypervascular and atrophic nonunion in the early phase
- Diagnostic adjuvants have been developed, such as the orthometer or strain gauge bar, but they are not yet universally used
- Recently, attention has been drawn to biochemical markers. The majority of studies are laboratory based with extensive work carried out on animal models, but encouraging data has been reported on the predictive value of transforming growth factor and alkaline phosphatase

Classification

Weber introduced a classification system based on biological activity at the fracture site. The system is widely used and has stood the test of time. Other classifications exist that are specific to a particular fracture site or injury type.

According to Weber, fracture nonunions are divided into hypervascular (hypertrophic) or avascular (atrophic) types. The hypervascular group has adequate vascularity and biological activity to progress to union but union is limited by bony stability and is evident on radiographs with excessive callus in response to motion at the fracture site. Excessive interfragmentary motion has a detrimental effect on healing. Weber further subdivided this group by radiological appearance (elephant foot, horse hoof and oligotrophic).

Avascular nonunions lack vascularity and biological healing potential and show no evidence of healing. Avascular nonunion can be further subgrouped by the fracture pattern into torsion wedge, comminuted, defect or atrophic fractures. The classification systems are useful in treatment planning. It is important to consider the possibility of infection in all cases, particularly in high-energy open fractures.

Risk factors

Nonunion occurs when there is failure of biology (high-energy injuries with devascularisation), failure of host (nicotine consumption, vascular disease and other comorbidities), failure of mechanics (improper stabilisation) or treatment failure (iatrogenic devascularisation). If the original injury was open, or there has been previous surgery to the limb, there should always be the suspicion of infection as the cause of nonunion. The aetiology is often multifaceted, requiring a host of patient and treatment factors to be managed simultaneously. **Table 2** summarises the common factors associated with nonunion which have been the literature to date.

Bones with a tenuous or precarious blood supply such as the scaphoid or talus are prone to nonunion after fracture, and even conservatively managed patients must be closely followed up. Maintaining vascularity to the fractured bone ends is vital, particularly during surgery. Trauma patients with high-energy mechanisms sustain soft tissue injuries

Table 2 The risk factors for fracture nonunion

Patient factors	Fracture-specific factors
Increasing age	High-energy trauma or injury severity score
Smoking	Soft tissue injury. Larger zone of injury and high Gustilo–Anderson grade
Poorly diabetic control	Large interfragmentary gaps
Osteoporosis	Biomechanical instability
Vitamin D, calcium, or protein deficiency	Infection
Increased alcohol consumption	Prolonged immobilisation
Reduced muscle mass and mechanical stimuli	Perioperative or prolonged nonsteroidal anti-inflammatory drugs use
Postmenopausal females	Complex or comminuted fractures
	Diaphyseal fractures
	Large fracture haematoma

with periosteal stripping. A larger zone of injury is associated with a greater risk of nonunion.

In addition to maintaining vascularity, a favourable local environment at the fracture site is crucial to the proper progression of healing. This is achieved by minimising interfragmentary gaps through accurate reduction and adequate stability through fixation or casting (**Figure 4**). Mechanical stability is required to prevent further insult to callus and to modify osteogenic behaviour to promote healing. Bone cells are sensitive to their environment and mesenchymal cells alter differentiation according to local forces. Hydrostatic forces promote a chondrogenic pathway; however, sheer or tensile forces have a detrimental effect resulting in fibrogenesis. Achieving mechanical stability in osteoporotic bone is challenging; technology such as locking screws where the screw engages into an osteosynthesis device has resulted in increased stiffness of fixation.

Smoking and dietary insufficiency are detrimental to bone healing and potentially modifiable to minimise risk of nonunion. Not only does nicotine cause tissue hypoxia through induced vasoconstriction, it also has inhibitory effects on biological activity and cell maturation required during the early phases of fracture healing.

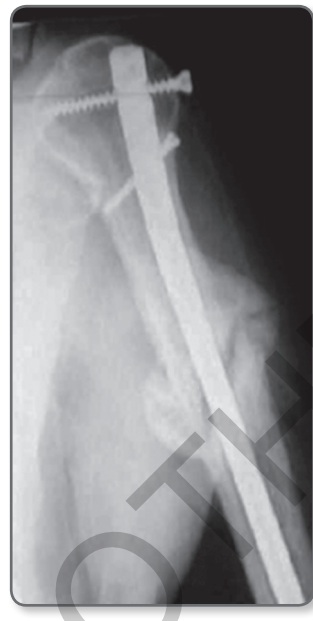

Figure 4 Humeral nonunion treated by nailing.

Conclusion

Nonunion remains a significant problem and socioeconomic burden. Early recognition and appropriate management of patients failing to progress are important; however, there is an increasing focus on modifiable risk factors to minimise risk.

Further reading

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Weber BG, Cech O. Pseudarthrosis, pathophysiology, biomechanics, therapy, results. Bern: Hans Huber, 1976.

Related topics of interest

- Fracture classifications (p. 107)
- Fragility fractures (p. 110)
- Metabolic response to trauma (p. 182)

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ISBN 978-1-909836-08-2
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