Open Fractures of the Hand

Review of Pathogenesis and Introduction of a New Classification System

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KEYWORDS

• Open fractures • Hand • Pathogenesis • Classification system • Infection • Treatment

KEY POINTS

- Open fractures of the hand are commonly encountered, and vary widely in mechanism, location, and severity.
- Current evidence shows that antibiotic use and the extent of contamination are predictive of infection risk, but time to debridement is not.
- Open fractures of the hand are less susceptible to infection than other open fractures.
- The different regions of the hand are unique with regard to the osseous anatomy, blood supply, and soft tissue coverage, all of which factor into the risk of infection after an open fracture.
- Current classification schemas for open fractures are insufficient to describe and indicate treatment of fractures of the hand. A specialized classification is introduced that may better take into account risk factors for infection specific to the hand when determining best treatment of open fractures of the hand.

INTRODUCTION

Fractures of the finger, hand, and wrist constitute a significant disease burden, estimated to comprise up to 1.5% of emergency department visits and constituting 1.4 million cases in 1998 alone.[1] Like all fractures, distal upper extremity fractures range in severity based on several factors, including mechanism of injury, fracture location, fracture pattern, and associated soft tissue injury.

Open fractures of the hand are a common occurrence. A database study in 2001 estimated that 5% of hand fractures are open.[1] Like all open fractures, open hand and finger fractures are at increased risk for infection compared with their closed counterparts. Beginning with anecdotal observations that these fractures were less likely than other open fractures of the body to become infected, several studies have attempted to stratify these injuries by infection risk.

AVAILABLE EVIDENCE ON OPEN HAND FRACTURES

A study by McLain and colleagues[2] examined 208 consecutive patients with open fractures of the hand. Overall, the cohort showed an 11% infection rate. This study had limited subject retention (143 of 208 patients) and excluded both farm injuries and human bite wounds. All injuries were irrigated and debrided in the operating room and received cephalosporin plus/minus penicillin and an aminoglycoside preoperatively.

A similar retrospective analysis of factors correlating with infection in open hand fractures was performed by Swanson and colleagues.[3] These

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investigators showed a 6% incidence of infection in a series of 154 patients, with 35 lost to follow-up. As in the prior study, all patients were treated with prompt intravenous antibiotics and bedside or operative irrigation and debridement.

An in-depth analysis of functional recovery following open fractures in 75 patients performed by Duncan and colleagues[4] showed an infection rate of 6 per 171 fractures (3.5%), all in Gustilo-Anderson type III injuries. This group also underwent standard treatment with antibiotics and urgent irrigation and debridement.

More recent retrospective reviews have varied in the reported incidence of infection in open hand fracture. A 2011 review of 145 cases by Capo and colleagues[5] showed a 1.4% infection rate, even in a series with a high proportion (91 out of 145) of Gustilo-Anderson type III injuries. Similarly, a 2006 review of bone grafting for open fractures of the hand found a 0% infection rate even in more severe fractures.[6] Moreover, a 2010 retrospective review of 432 metacarpal and phalanx fractures requiring internal fixation found no significant difference in infection rates between the open (133 fractures) and closed (299 fractures) injury groups.[7]

These infection rates are significantly lower than that identified in a 2012 meta-analysis of all open fractures, not only hand open fractures, by Schenker and colleagues.[8] That review found an 8% infection rate in Gustilo-Anderson class I and II fractures, and a 12.7% rate in class III fractures. This finding supports the traditional wisdom that the hand is more resilient and less prone to infection after an open fracture than other open fractures of the body.

VARIABLES AFFECTING INFECTION RISK FOLLOWING AN OPEN FRACTURE OF THE HAND

There are several potential variables that may cause an open fracture to be more or less prone to developing an infection. These variables include the local osseous and soft tissue anatomy, the extent of contamination, the integrity of the soft tissue envelope, and the vascularity of the extremity.

Anatomy

Within the hand, distal to the radius and ulna, there are 27 bones that are prone to injury and an open fracture. Each has its unique anatomy, blood supply, and soft tissue coverage. Divided broadly, they can be separated into 3 regions: the phalanges, the metacarpals, and the carpal bones.

The soft tissue coverage of the phalanges consists of skin, tendon, ligament, areolar connective tissue, and nail. The 14 phalanges of each hand are devoid of muscle. As a result, the digits are prone to open injury with minimal amounts of trauma or fracture displacement, especially in the dorsal surface where the fascial layers lack the robustness of the palmar side. Furthermore, these structures do not possess the bulk or vascularity of muscle, potentially limiting their ability to fight infection.

The metacarpals share some morphologic features with the phalanges. Among these are palmar layers of tough fascia and alveolar connective tissue, and a dorsal surface with a thin covering of skin, tendon, and fascia. However, the metacarpals also benefit from the presence of interosseous, thenar, and hypothenar musculature, providing bulky coverage and blood supply. As a result, the metacarpals are vulnerable to dorsal open injuries and wounds but benefit from a robust blood supply.

The carpal bones possess the most dense soft tissue coverage of the osseous regions of the hand. However, they have the most fragile blood supply because of their absence of muscular coverage and otherwise extensive articular nature. Subsequently their blood supply is derived from their ligamentous and capsular attachments, structures that can be readily compromised with trauma. However, these soft tissue attachments, combined with the deep position of the carpus and its highly congruent and strong intercarpal attachments, provide resistance to open fractures in this region.

Vascular Supply

The digits receive most of their blood supply via the palmar digital arteries, with contribution from the dorsal digital arteries. Distally, these palmar arteries anastomose to form the blood supply to the digital pulp.[9] The palmar digital arteries run superficial to the digital nerves and lie directly deep to the skin. As a result of their position, these vessels are easily injured during digital trauma, compromising blood supply and increasing infection risk of the digit. This effect can be mitigated by the arterial anastomoses in the digit, which provide redundant blood supply in case of injury. Degloving, ring avulsion, and other circumferential injuries are a particular risk for dysvascularity, and loss of both radial and ulnar digital arteries can result in an avascular digit.

More proximally, the hand benefits from a robust and redundant vascularity. The vascular supply of the hand is provided by the palmar