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Complications of Proximal Humeral Fractures

Abdelsalam Eid Abdelsalam, Mohamed Hegazy Abdelhamid Ahmed, Hany Mohamed Abdelfatah Bakr, Salah Mahmoud Abdelkader

Department of Orthopedic Surgery, Zagazig University Hospital, Egypt.

Corresponding Author: Mohamed Hegazy Abdelhamid Ahmed

Email: Mohamed_hegazy2017@hotmail.com

Abstract

Background: Proximal humerus fractures are common, representing 5% of all adult fracture. The majority are stable injuries, occurring in elderly osteoporotic individuals after low-energy trauma. A conservative treatment in a sling followed by functional rehabilitation under supervision yields satisfactory results in minimally displaced fractures. In 15% to 20% of displaced proximal humerus fractures where there is significant displacement or comminution going to surgical fixation is preferred for better results.

Keywords: Fifth Metatarsal Fractures

Introduction

There is much complication to proximal humeral fractures which contributed too many factors some related to the nature of the fracture, bad selection of technique or bad operative technique**[1]**

Avascular Necrosis:

Osteonecrosis, avascular necrosis, and ischemic necrosis are terms to describe the loss of circulation to the terminal articular surface. This is followed by collapse in most cases **[1]**

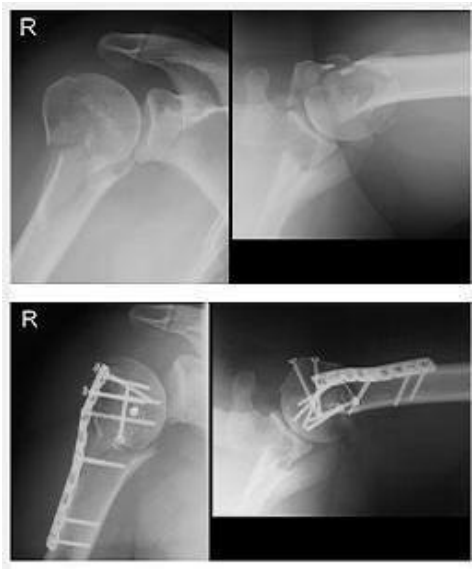

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Figure (1): Rigid fixation of a fracture **[1]**

Although rare, it can occur after two-part and minimally displaced fractures. It occasionally occurs with a three part displaced proximal humeral fracture, but most commonly occurs after four part fractures or fracture dislocations **[1]**

Although the amount of osteoporosis and severity of the trauma may play a role, occlusion of the arcuate artery of Laing in the proximal portion of the biceps groove, which provides vascular supply to the anterior and superior two thirds of the humeral head, is a major contributing factor **[1]**

The timing of replacement if this complication occurs is important. The longer one waits, the more likely it is that both sides of the joint will need to be replaced **[2]**

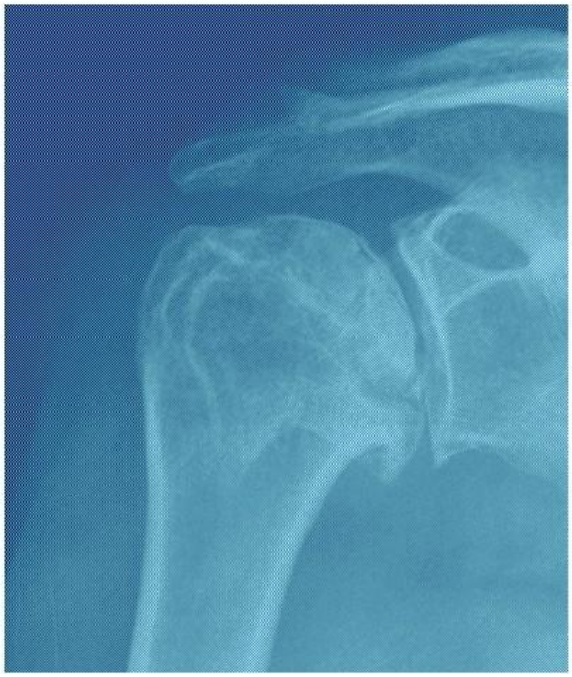

Figure (2): Avascular necrosis **[2]**

In those patients in whom posttraumatic arthritis has developed from avascular necrosis, humeral head replacement may suffice. If the glenoid surface is significantly damaged, and in particular if the concavity of the glenoid is altered with bony erosion at one edge, a total shoulder is preferred. Prosthetic arthroplasty reliably provided pain reduction, restoration of range of motion, and improvement in function**[3]**

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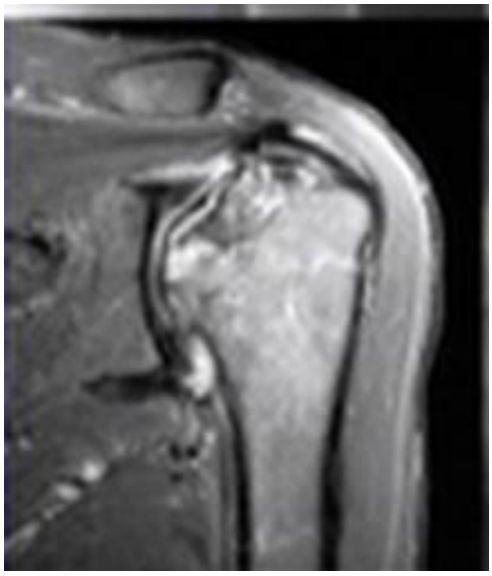

Figure (3): Magnetic resonance (MRI) scan **[2]**

Metal Failure

As the use of locked plates has expanded and the numbers of fractures fixed with these plates have increased, clinical failures have been seen. The plates can fail when physiological loads are outside plate design parameters.

Causes:

- The locked screws can disengage from the plate secondary to failure of the screw to seat into the plate properly as a result of cross-threading (where the screw threads and the plate threads are not collinear) or when insufficient screw torque is used to engage the screw threads into the plate threads**[4].**
- Additionally, like all mechanical devices, the screws can break or disengage from the plate under excessive cyclical loading.
- Despite an excellent feel in the operating theater, locked plates may cease providing fragment fixation as a result of exceedingly poor bone quality.
- Nonunion and malunion can still occur with the use of locked plates**[4].**

Fracture reduction can be challenging with locking plates because the locking screws do not pull the plate to the bone in the manner of conventional screws. Therefore, it is essential that the surgeon have a preoperative plan for fracture reduction effective use of locking plates requires an understanding of the potential Pitfalls of usage **[4].**

Locking holes offer minimal opportunity for screw angulation. More than 5° of angulation between the screw and the locking hole can cause the screw to eventually fail. Careful technique is necessary to ensure that the screw is perfectly lined up with the axis of the screw threads in the plate. Malaligned screw threads can lead to lose screws and loss of reduction **[4].**

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The weakest part of the combi locking plate (e.g., the LCP) is the dynamic compression unit. This is the part of the plate that should be used for bending, if required, and it is the part that breaks when there is increased stress concentration and strain on the plate.

For this reason, when a bridge plate is used to fix a comminuted fracture, at least three or four plate holes should be left empty at the level of the fracture, in order to achieve a larger area of stress distribution on the plate **[5].**

In contrast to conventional plates, which fail at the interface between the screws and the plate often leading to breakage of conventional screw heads the interface of the locking head screw with the threaded locking hole is the strongest part of the locking plate system **[5].**

Locking head screws can break in cases of chronic instability and increased Strain as a result of rotational forces **[5].**

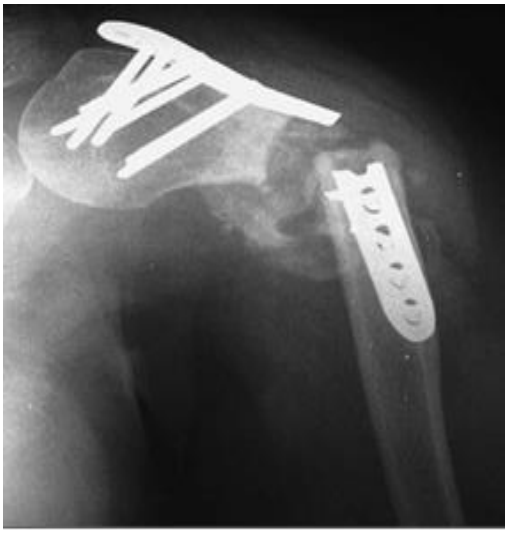

Figure (4): Metal failure**[4]**

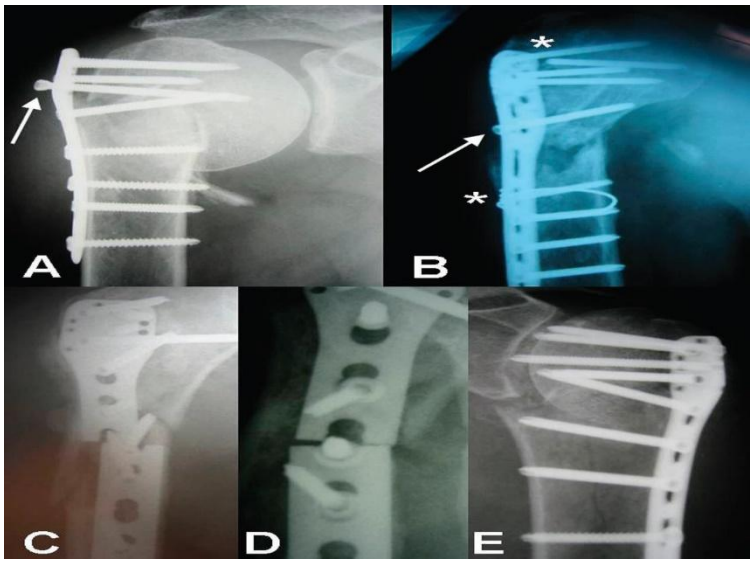

Figure (5): Typical patterns of failure of locking plate fixation of proximal humeral fractures.**(6).**

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Posttraumatic Shoulder Stiffness:

The causes of posttraumatic shoulder stiffness are often multifactorial: Although capsular contracture is usually the main cause of refractory stiffness, other factors may include fracture malunion, complex regional pain syndrome, and mechanical impingement of implants. These factors are poorly described in the contemporary literature but may nevertheless be contributory to persistent stiffness after fracture **[7].**

The most characteristic finding is of restriction of movement in a capsular pattern, with generalized stiffness but selectively greater loss of shoulder abduction and external rotation. The initial treatment is non- operative with shoulder rehabilitation to attempt to regain movement by selective stretching exercises. Most patients improve to a degree on this regime, and recovery of movement is often protracted over the first year after injury**[8].**

A plateau in recovery is usually heralded by the presence of a firm woody feel on terminal stretching exercises, suggesting a mechanical block to movement. Distension arthrography is useful in stretching and rupturing the capsule in idiopathic adhesive capsulitis, but it is the author's experience that this procedure is less effective in the post-traumatic shoulder **[8].**

In patients with refractory posttraumatic stiffness without malunion, treatment with manipulation under anesthesia is usually performed. This procedure is contraindicated in patients with uncertain fracture healing and in patients with severe osteoporosis, where there is a substantial risk of humeral shaft fracture during manipulation. If manipulation is unsuccessful in regaining sufficient movement, this should be followed by arthroscopic release of capsulitic tissue from the rotator interval, circumferential intra- articular capsular releases, subacromial decompression, and removal of impinging metalwork **[8].**

Infection:

Infection post-surgery for proximal humeral fracture is relatively rare in the shoulder even after surgical repair using open methods. This is because of the rich vascularity to this region and the good soft tissue coverage. The risk is likely to be increased in patients with a more severe soft tissue injury and grade of fracture, prolonged surgery time, poor surgical technique. It is important to distinguish superficial from deep infections**[9].**

Malunion:

Proximal humerus malunion results from either inadequate reduction of the displaced fragments or loss of fixation following closed reduction, with or without percutaneous pinning, or ORIF. They occur more commonly after nonoperative treatment **[10].**

Occasionally, a malunion occurs because the treating physician failed to appreciate the extent of displacement either due to lack of experience or inadequate or incomplete imaging studies **[10].**

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The malunion seen after internal fixation usually is secondary to inadequate fragment fixation obtained in the poor cancellous bone of the proximal humerus. This can result in post-operative loss of fracture reduction. It can also occur due to inadequate fragment reduction at the time of surgery **[10].**

Other contributing factors in proximal humerus malunion include inadequate immobilization, or soft tissue interposition at the fracture site. Excessively aggressive rehabilitation can result in loss of fracture reduction or fixation **[10].**

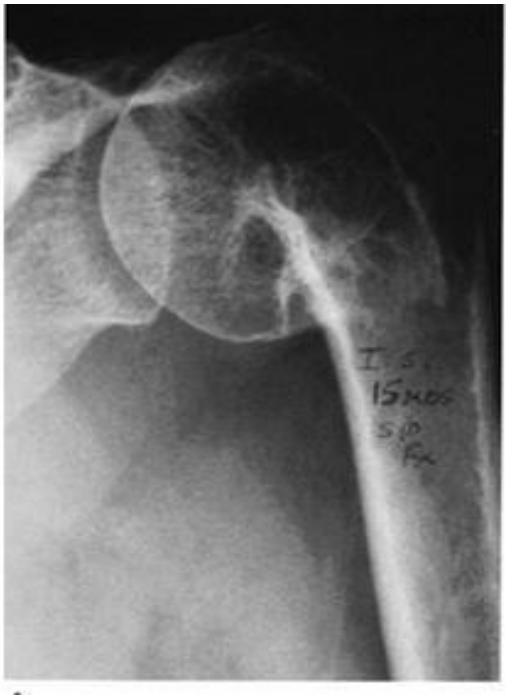

Figure (6): Malunited proximal humerus **[10].**

Nonunion:

Fractures of the proximal humerus are nondisplaced in the vast majority of cases and typically heal uneventfully with closed treatment. Unfortunately, a small percentage of these fractures treated nonoperatively or operatively, develop nonunion **[10].**

Patients that develop this complication are often debilitated with persistent pain and limited function. The technical challenges of previous scarring, poor bone stock, and prior hardware can make these cases extremely challenging, but successful treatment of proximal humerus nonunion resulting in relief of pain and restoration of function can be extremely rewarding**[10].**

The incidence of proximal humerus fracture nonunion suggests that they are a rare phenomenon. However, nonunion may be commonly seen after displaced two-part surgical neck fractures or after cases in which inadequate open reduction and internal fixation was used as the primary treatment **[10].**

In fact, up to 23% of surgical neck fractures may go on to nonunion. Other fracture patterns that have a higher incidence of nonunion include those with displaced

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tuberosities, which are treated closed, and four-part fractures, which are treated either closed or open**[11].**

Factors contributing to the development of nonunion may be attributed to either patient factors, fracture site factors, sequelae of inappropriate primary fracture treatment, or any combination there**[11].**

Fracture site factors also contribute to the development of nonunion. Soft tissue interposition between the proximal and distal fracture fragments can prevent adequate contact of opposing bone surfaces, impeding callous formation and fracture reduction. The long head of the biceps tendon, the deltoid muscle, or the rotator cuff tendons are potential impediments **[12].**

Additionally, the dense cortical bone in the distal fragment of a surgical neck fracture and the generalized decreased quality of metaphyseal bone in the proximal humerus seen in this elderly population predispose to poor bone healing **[12].**

Surgical neck fractures are also subject to the deforming forces of surrounding musculotendinous units. The pectoralis major pulls the proximal shaft anteromedially and the rotator cuff tendons rotate and abduct the proximal head or tuberosity fragments. These forces can prevent adequate reduction of the fracture segments and contribute to nonunion **[13].**

Lastly, synovial fluid from the adjacent joint can dilute the fracture hematoma and inhibit callous formation.

Finally, proximal humerus nonunion can result as the iatrogenic sequelae of inadequate primary fracture treatment. Closed treatment can be problematic as the weight of the arm causes distraction across the fracture site, which can be accentuated with the use of a hanging arm cast**[12].**

Attention to detail is necessary when treating these fractures with immobilization; the arm must be immobilized across the front of the body to neutralize the medial pull of the pectoralis, and the elbow must be kept in front of the midline in the coronal plane to prevent apex anterior angulation and loss of reduction **[12].**

Inadequate operative treatment can also contribute to nonunion, especially if severe osteopenia is overlooked at the time of initial intervention. Epidemiologic studies have linked proximal humerus fractures with fractures of the distal radius and proximal femur, especially in elderly women. This factor should not be overlooked as plate and screw fixation can be problematic in osteopenic bone, leading to inadequate fixation, hardware loosening, fracture site motion, and nonunion **[12].**

Lastly, premature institution of range-of-motion exercises following closed or open treatment contributes to nonunion.

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It is imperative that the fracture parts have consolidated and move as a unit prior to the initiation of shoulder motion **[12].**

Patient factors that predispose to proximal humerus fracture nonunion include osteoporosis, metabolic bone disease, diabetes, drug or alcohol addiction, nutritional deficiency, smoking, and general non-compliance **[14].**

Secondary to preexisting glenohumeral stiffness, either from rheumatoid arthritis or prior glenohumeral fusion. In this situation, humeral motion occurs in the fracture site rather than the glenohumeral joint, leading to nonunion **[14].**

Its believe that osteosynthesis with preservation of the humeral head is worth considering when adequate reduction and stable conditions for revascularization can be obtained. If osteosynthesis is not feasible, especially in older, incompliant patients with osteopenic bone and/or comminuted fractures, hemiarthroplasty seems to be a viable alternative to osteosynthesis, offering comparable final outcomes to osteosynthesis **[15].**

Rotator Cuff Tears:

All fractures dislocations in which either or both tuberosities are significantly displaced will, by necessity, have a longitudinal split in the rotator cuff often along the rotator interval between the supraspinatus and the subscapularis. In these cases, rotator cuff repair should be performed at the time open reduction and internal fixation is performed. Therefore, the elderly are particularly susceptible to this injury, because their already degenerated cuff tendons are less able to withstand the high-tensile forces generated during dislocation **[16].**

Neurologic Injuries:

Injury to peripheral nerves and the brachial plexus accompany fractures dislocation of the shoulder, with a reported incidence of between 2% and 30%. This variability partly reflects age differences of the groups studied, as well as the method of assessing neurologic deficits by physical examination versus EMG **[17].**

The axillary nerve, which courses anteriorly along the anterior border of the subscapularis, is especially vulnerable to traction injury during an anterior dislocation. It is the most commonly injured nerve in shoulder fractures dislocations, and the likelihood of injury increases with the age of the patient, the duration of the dislocation, and the severity of the trauma producing the injury **[17].**

This nerve can also be damaged with surgical approaches that split the deltoid fibers beyond 5 cm from the acromial edge or with percutaneous pins inserted from an inferior starting point **[10].**The vast majority of these injuries represent traction neurapraxias and usually resolve spontaneously within 5 months. Other isolated nerve injuries to the musculocutaneous, radial, and ulnar nerves are much less common **[17].**

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When a neurologic injury is suspected, the initial physical examination findings should be carefully documented. An EMG should be obtained 3 weeks following the injury for further documentation. This is a much more sensitive method of assessing a neurologic injury than the physical examination **[18].**

The difficulties in assessing axillary neuropathy based on physical examinations are twofold. First, dermatomal sensory testing is very unreliable. Second, the high incidence of rotator cuff tears associated with these shoulder injuries confuses the picture somewhat. Clinical findings of weak abduction can be due to rotator cuff tear, axillary neuropathy, or the acute pain associated with the injury **[18].**

Surgical options include neurolysis, nerve grafting, and neurotization. In most series, the majority of patients who required surgery underwent a nerve grafting procedure. The results of nerve grafting have been encouraging, due to the relatively short distance from the lesion to the motor end-plate and the monofascicular nature of the proximal portion of the axillary nerve **[18].**

Vascular Injuries:

Fractures dislocations of the shoulder can, on rare occasions, be associated with vascular injuries usually involving the axillary artery, its branches, or the axillary vein. These injuries can occur either during the injury itself or during the reduction maneuver. Attempted reductions of chronic dislocations are particularly risky and should be discouraged. Invariably, too many attempts are made and too much force used **[19].**

Vascular damage most frequently occurs in elderly patients with fragile, atherosclerotic vessels and in patients whose soft tissues (and vessels) may be adherent as a result of previous dislocations or other injuries when a vascular injury occurs; it is an emergency and must be addressed immediately. These injuries are associated with a mortality rate of up to 50% **[19].**

Different types of arterial damage can occur, including intimal damage followed by thrombosis, avulsion of a large arterial branch, and laceration of the artery itself **[19].**

As discussed earlier, anatomic studies have shown the axillary artery to be relatively fixed at the lateral border of the pectoralis minor muscle. The artery becomes taut with abduction and external rotation of the arm and is vulnerable to injury when the humeral head dislocates anteriorly and the pectoralis minor acts as a fulcrum over which the artery is stressed. There is the further risk with fractures dislocations that the artery can be impaled or lacerated by the fracture fragments **[19].**

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