SVOA Orthopaedics

ISSN: 2752-9576

ScienceVolks Open Access

Review

Management and Complications After Paediaric Fracture

Nguyen Ngoc Hung[1*], Hoang Hai Duc[2], Le Tuan Anh[2], Phung Công Sang[2] and Nguyen Vu Hoang[2]

[1]Surgical Department, General Hospital HongPhat, 219 Le Duan Street, Hanoi City, Vietnam.

[2]Paediatric Orthopedic Department, Vietnam National Hospital for Paediatrics, Vietnam.

***Corresponding Author:** Nguyen Ngoc Hung, Surgical Department, General Hospital HongPhat, 219 Le Duan Street, Hanoi City, Vietnam.*

***Received:** November 24, 2022* ***Published:** December 23, 2022*

Abstract

Fractures in children are a common emergency and often leave many complications. Complications have early complications and late complications. Early complications such as compartment syndrome, damage to blood vessels, nerves... Late complications such as infection, displacement after fracture… Fractures represent many types of injuries that share common features of fractures. These categories range from penetrating wounds from the inside out to more extensive wounds, representing high energy trauma; Significant damage to surrounding tissues (skin, subcutaneous layer, muscle structures, tendons, and nerve vessels) must be observed. Fracture management involves a series of principle-based steps to guide initial emergency management, followed by chiropractic management, and finally rehabilitation. Recognizing and implementing the management principles of Improving Life After Trauma is important to integrate into the approach. Ultimately, the goal is to stop the development of infection, heal fractures, and restore function of the limbs. Clinical with fracture management principles based on a combination of historical and traditional experience, basic science, and clinical research evidence. Advances in post-traumatic musculoskeletal care have led to evolution over time in what is accepted as the standard of care and rehabilitation of the limb. Advances in antibiotics, implantation and immobilization methods, reduce the incidence of nosocomial infections. Cost-effective care has created the need for a reassessment of standards practice fracture.

***Keywords:** Compartment syndrome, Vascular injury, Nerve damage, Pathological ischemia, Osteomyelitis.*

Introduction

Most bone injuries heal normally. But some patients experience complications during the healing process.

Complications of fractures fall into two categories: early and late:

1. Early complications include: fat embolism, compartment syndrome, deep vein thrombosis, Vascular trauma, Nerve damage, Fat embolism syndrome, and post-fracture infection.

2. Delayed complications include: Pin line infection, Fixed hypercalcemia, Myelitis, delayed association, No nunion, Remetastasis, and Cubitus varus.

COMPARTMENT SYNDROME

Acute compartment syndrome (ACS) has been recognized since 1881 when Volkmann first described spasticity of the hand due to compartment syndrome [1]. The first report on the treatment of acute limb compartment syndrome was by Petersen in 1888 [2]. The diagnosis and management of ACS in adults is well known, but only a few authors have reported on pediatric populations.

Compartment syndrome in pediatric patients most commonly occurs after limb trauma, especially after fracture. The most common site of compartment syndrome in children is the forearm, Foot, and humerus (Figures 1).

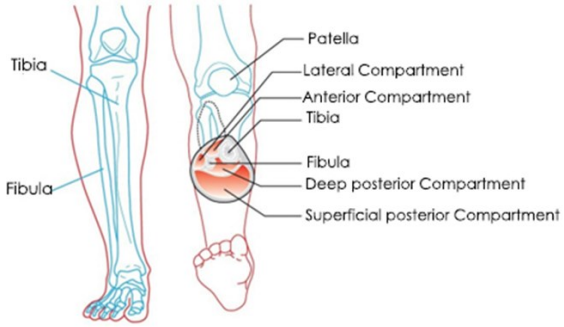


Figure 1: *Major anatomical structures of the leg.*

This condition is caused by swelling and increased pressure in an enclosed space, such as an enclosed space, but it can also occur from stretched skin or a cast. If not treated in time, it will lead to complete death of internal structures and cause ischemia of Volkmann spasticity. Compartment syndrome occurs in the medial compartments of the hands and feet, the sternum and dorsal cavities of the forearms, thighs, and all four compartments of the legs (Figure 2). Trauma and compression are classic causes, but compartment syndrome is more common with fractures, severe impacts, drug overdoses causing extremities, burns, and vigorous movement. In children, compartment syndrome may be accompanied by vascular damage or bone resorption, especially in the proximal tibia. Multiple trauma predisposes children to compartment syndrome because other high-risk factors such as hypotension, vascular trauma, and high-energy trauma increase tissue necrosis.

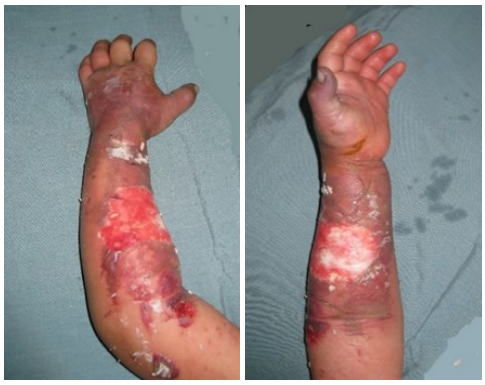


Figure 2: *Photographs showing that the elbow and forearm on presentation were red, hot, and diffusely swollen. Anterior, posterior, and mobile wad compartments were tight with palpation.*

The appropriate therapy for ACS, after removal of any external source of compression (peripheral dressings, casts, etc.), is to decompress all involved compartments using the open technique. Skin incision alone reduces ICP by 5-9 mmHg. The subcutaneous technique, even successfully used in chronic compartment syndrome, does not appear to be sufficient in ACS [4]. The lateral incision decompresses the anterior and lateral compartments, whereas the medial incision decompresses the superficial and deep posterior compartments (figures. 3). A complete fasciotomy is of utmost importance (Figure 4) . Incomplete fasciotomies do not adequately release a muscular compartment, contribute to continued compartment syndrome, and lead to worse outcomes.

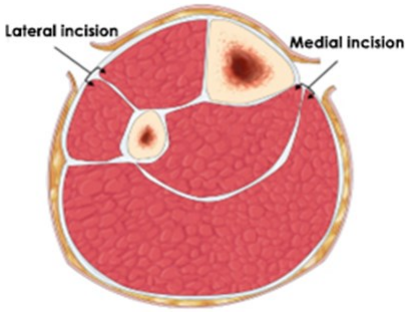


Figure 3: *Four-compartment fasciotomy of the right leg through two incisions. The lateral incision decompresses the anterior and lateral compartments, and the medial incision decompresses the superficial and deep posterior compartments.*

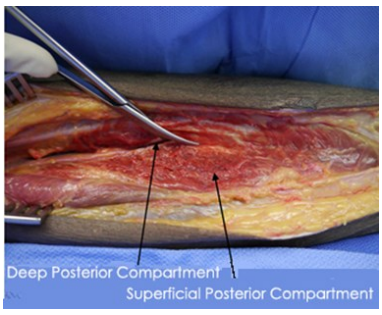


Figure 4: *Left leg fasciotomy,medial incision. The superficial compartment is decompressed with a fascial incision, made about two fingerbreadths posterior to the tibia. The deep posterior compartment is decompressed through a fascial incision just behind the edge of the tibia.*

Skin defects during an open plantar fasciectomy should not be closed in the same session. To avoid skin grafting, the Berman ligation technique can be used successfully to close the secondary wound [5], but secondary wound healing, secondary skin sutures, skin grafting, or topical application can be used. mask. covered by cap. For our patient, in most cases, a secondary direct plantar fasciectomy could be performed (87.5%).

Treatment of compartment syndrome includes emergency plantar fasciectomy to relieve pressure in the affected compartment and re-establish perfusion to the involved tissues. Permanent ischemic damage to nerves and muscles can occur as early as 6 to 8 hours after compartment syndrome develops. Delays in treatment can lead to loss of limb function and spasticity.

VASCULAR INJURIES

The potential for serious vascular injury in fractures is related to the location and mechanism of bone injury. The blood vessels in the extremities are prone to rupture, mainly because of the proximity of the blood vessels to the bones, the fixation around the joints, and their surface position. Therefore, vascular trauma should be anticipated with fractures and/or dislocations in vulnerable anatomical regions, such as the knee, elbow, and shoulder. Arterial-related fractures carry a high risk of muscle necrosis, partial or total amputation, or loss of extremity function.

Fifty-eight percent of neurological injuries in children are related to orthopedic trauma. Usually, the artery involved is located near the fracture; For example, the common femoral artery is frequently associated with internal fractures and dislocations of the hip, and the superficial femoral artery and femoral artery are often associated with medial and inferior axial fractures. The femoral artery can be damaged by rupture of the vessel leading to a fracture of the upper femur. Injury to the tibial artery or a combination of the anterior and posterior tibial arteries is commonly associated with distal femoral fractures, proximal femoral fractures, or dislocations of the tibia or proximal knee joints (Figure 6) 32% to 64% and fracture was reduced and fixed with cross pins; Early growth arrest then occurs. [6].

Common signs of vascular injury are (1) absence of blood vessels at the distal end, (2) lower skin temperature, and (3) poor skin circulation with decreased capillary and venules at the distal end of the wound. Children are particularly susceptible to ischemia and gangrene due to vasospasm, a rare problem in adults. Patients should be evaluated further if pulse does not return after reduction of fracture or dislocation (Figure 7B). It is not enough to observe that the leg does not have a warm rhythm after a knee dislocation. Typically, these patients have good capillary flow because the amount of flow required to maintain skin and subcutaneous tissue viability is much less than is required by the muscle. In these cases, Green and Allen reported that 90% of the limbs eventually had to be amputated or had fibrosis and spasticity or spasticity.

In general, indications for saving the arm are expanded in children because of a higher chance of cure; however, there are no data to establish rescue limits. In all limbs, 90% can be saved if circulation is re-established within 6 hours, while revascularization after 8 hours of injury can result in amputation rates ranging from 72% to 90%. In children, natural vein grafts are preferred over synthetic or bovine materials. Peeling off the ends of the blood vessels allows a longer suture to be created to accommodate future vascular enlargement without being too tight. In contrast to proximal trauma, single vascular injuries distal to the elbow or knee can sometimes be treated with vascular ligation.

Claffey. In 1980 [7] found that complications with initial lower extremity fixation were less frequent than with internal fixation. Indoor arterial and venous shunts may be helpful in certain circumstances to reduce the risk of vascular damage and compartment syndrome. Similarly, temporary shunt placement may provide a satisfactory solution to the clinical question of whether an ischemic limb should be reperfused prior to immobilization of the fracture. Bone shortening surgery can facilitate vascular repair and can subsequently resolve leg length differences.

Vascular injury associated with Supracondylar of the Humerus, Femur fractures

Vascular damage is the most serious complication associated with supracondylar fractures; Fortunately, it is not common [8]. If a child has non-vascular quadriplegia, immediate fracture reduction is needed to try to restore blood supply and avoid compartmental ischemia [9] (Figures 5).

Campbell et al. [10] found brachial artery damage in 38% of patients with severe supraclavicular fractures. Because children are very susceptible to vasospasm, the pulse may not return to normal and Doppler waveform analysis may be helpful. These techniques are noninvasive and safe to assess brachial artery protection, but they need further study to evaluate their applicability in clinical practice. Children can have very good secondary capillary perfusion, which can lead to the misconception that the vessel is intact.

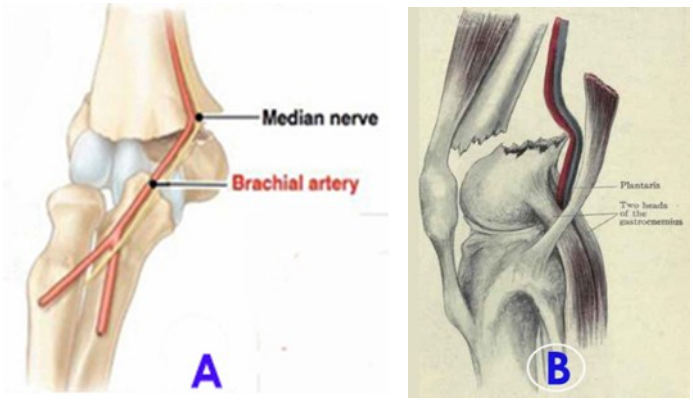


Figure 5 A-B: ***A****. Vascular injury in paracondylar humerus fracture.*
B*. Vascular injury in paracondylar femoral fracture*

Likewise, children should have their pulse checked periodically after signs of brachial artery stenosis and tear have subsided. Blakemore and colleagues found that one-third of children with upper displacement fractures associated with lateral forearm (floating elbow) fractures develop compartment syndrome.

About 10% to 20% of supracondylar fractures have altered vascularity. The radial impulse was reported to be absent before decreasing from 7% to 12% in all fractures and up to 19% in displacement fractures.

The absence of radial pulses after crack closure was described in 3.2% of cases. Non-arrhythmic hand alignment and median nerve palsy strongly predict the need for exploratory surgery.

Hands are the best indicator of vascular health. According to the presence of pulse and perfusion of the extremities, three situations were noted: 1) good pulse and well perfused extremities (warm, pink, capillary filled <3 s with distal pulse detected. by eco-Doppler); 2) the so-called avascular hand when there is no pulse but the hand is well perfused (warm, pink, capillary filled <3 s, distal pulse unchanged); 3) the so-called cold hand when there is no pulse and the hand is poorly perfused (pale, cold and capillary recovery > 3 s). The treatment algorithm is based on these three scenarios (Figure 6).

Blakey et al [11] note the importance of monitoring pain and signs of progressive neuropathy in the setting of pinky hands, as these patients may be susceptible to surgical visits. probe. Controversy continues about the long-term consequences of poor vascularization, such as cold intolerance, exercise-induced ischemic symptoms, and differences in limb length in some diseases. core. Carbonell et al looked at 14 adults with a median age at the most recent follow-up of 20 years who had a grade III ultrasound fracture without pink pulse at an early age (mean age at fracture). is 7 years old). 12 years old]. The patient was managed with fracture closure, percutaneous nailing and close vascular monitoring without surgical revascularization.

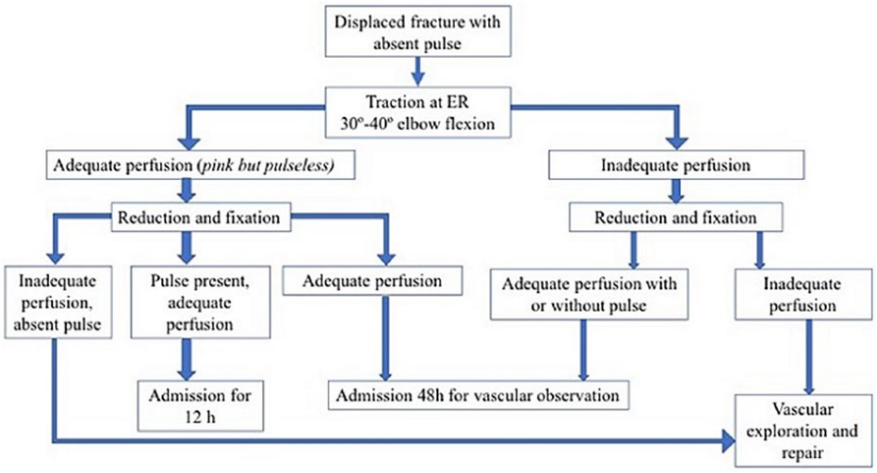


Figure 6: *Algorithm for the management of vascular injury in the context of a supracondylar fracture.*

Vascular exploration

Urgent pulse exploration is indicated in patients with cyanosis and palpitation. This should be done by a surgeon with experience repairing small blood vessels or in conjunction with a vascular surgeon. First, attempts should be made to reduce adduction and immobilize the fracture to see if this restores perfusion to the limb.

The management of patients with pink, perfused, and pulseless hands after correction and fixation remains controversial. These patients should be closely monitored postoperatively. Nonsurgical management has been shown to yield acceptable results. [13] Some patients with no pulse, with immobilized arm may continue to be ischemic, and this group of patients requires urgent pulse exploration [13].

NEUROLOGICAL INJURY

Neurological evaluation must be performed carefully in children. If the record is relevant, it should be carefully evaluated and documented by documenting the onset, extent and possible progression of the neural structures involved as well as the organs and supplies. grant. patient level.

An article published in 2013 titled Anatomical basis of anterior cruciate nerve palsy secondary to maxillary fractures in children concluded that the mechanism of injury leads to the development of selective AIN paralysis. secondary to upper elbow fracture in children can be the result of two factors: direct contact of the posterior aspect of the median nerve, and thus the AIN nerve, by the proximal segment; The tension of the AIN is located in Zone 1, where there is less tension than the median nerve and its other branches because the AIN is fixed in Zone 2. A 2008 study mentioned that the prevalence of cord injury traumatic neuropathy and traumatic nerve damage in children with mandibular fractures. 12-20% and 2-6% respectively. They found that the median nerve, specifically the anterior intercostal nerve, accounted for 52 percent of the injuries and the afferent nerve for 32 percent of the injuries.

McGraw et al. [14] identified an association between posterior displacement and median nerve injury, and postoperative displacement (Figure 7) with equal rates of radial, medial, and ulcerative nerve injury.

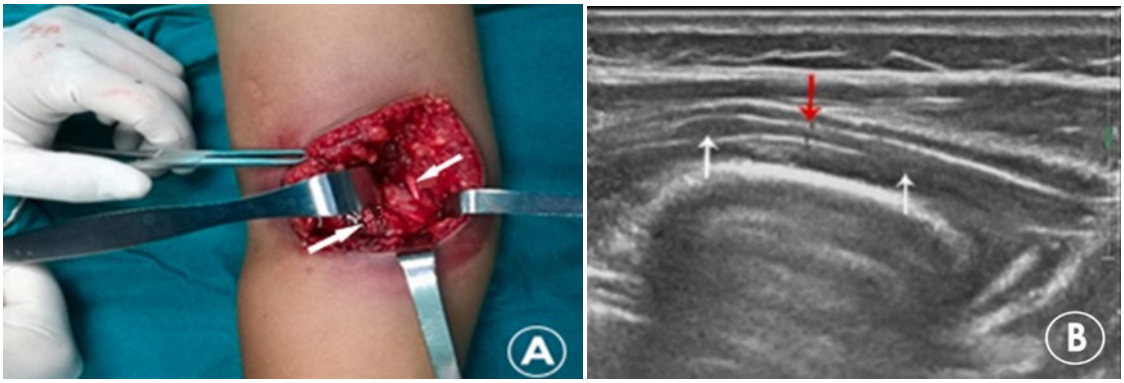


Figure 7: *A-B.* ***A****. Another SCHF with an injured median nerve trapped posterior to the proximal fracture end. (The left arrow shows the fracture end, and the right arrow marks the trapped median nerve.).*
B. *Showed the narrowed part of the trapped radial nerve (red arrow) in ultrasound.*

Nerve damage can occur in 6.5% to 19% of displaced fractures, and they are very specific in nondisplaced fractures. They may appear before surgery (primary injury) or after correction and immobilization of the fracture (secondary injury).

In fact, Valencia et al. found that more than half of their ulcerative nerve injuries were immobilized with lateral staples alone [15]. The authors hypothesized that nerve damage from ulcers may reflect the severity of the injury or the difficulty of reducing fractures by self-contained measures. Most of these lesions are neurological and resolve spontaneously; Therefore, surgical exploration of the nerve is rarely indicated. In our opinion, when a midpin is present in a patient with an ulcerative nerve injury, the median pin should be resected (Figure 8). However, Lyons et al. [16] reported complete recovery in 17 patients with postoperative ulcerative nerve palsy and central pin fixation despite only 4 cord resection.

Valencia et al. [15] reported in their series that 100% of radial nerve injuries, 87.5% of median nerve injuries, and 25% of ulnar nerve injuries were recovered if carefully managed during long-term follow-up. Median recovery time was 3 months for the afferent nerve, 2.5 months for the median nerve, and 5 months for the optic nerve.

The rate of primary nerve damage reported in displaced supraclavicular fractures is up to 20%. The incidence of iron-induced nerve damage has been reported to be 2-3%. However, even in the case of incomplete preoperative data, this number increases to 10%. In this study, the rate of iron-induced nerve damage was 6.0%, which is high compared to the literature. However, we would like to emphasize that in this percentage, there were 10 cases of unsatisfactory reduction or vascular injury.

When these fractures are closed, the ulnar nerve is at risk because of the location of the median Kirschner nerve near the ulnar tunnel. The median nerve may be directly punctured or constricted by the cuboidal tunnel, or the motor nerve outside its groove in the epicardium may be injured. Possible mechanisms for late ulcerative neuropathy are nerve constriction with edema or stretching of the ulcerated nerve through a pin. Delayed ulnar nerve palsy may persist in the medial crooked position because displacement of the triceps mediates the extrusion of the ulnar nerve. One of the methods used to avoid damage to the ulcerated nerve by the Kirschner nerve is the use of parallel bilateral pins. Results using this method were comparable to cross-stapling, although other methods failed to reproduce the reduced risk of iron-induced nerve damage and described increased rotational instability.

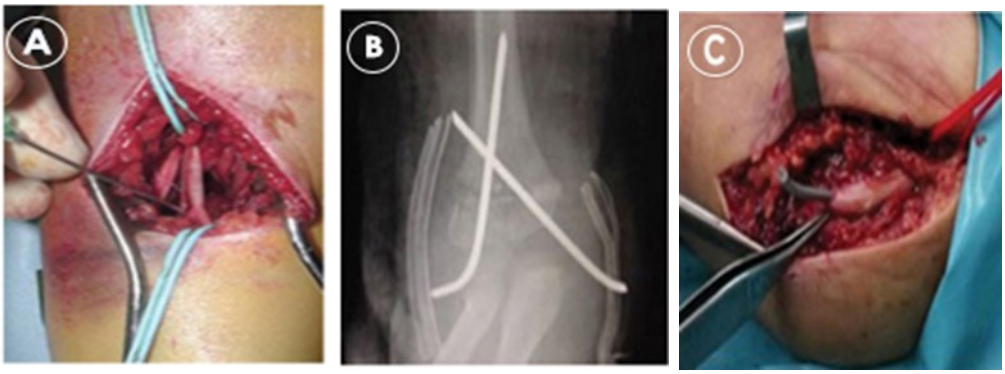


Figure 8: *A-C.* ***A****. Exploration of the brachial artery and median nerve for neurovascular injury;* ***B.*** *Fracture fixated with crossed pins percutaneously;* ***C****. Ulnar nerve was traversed by medial pin and post-operative palsy was present.*

Brown and Zinar reported that there was no nerve damage from iron ulcers after using a small incision in the middle epigastrium [17]. Now we also follow this process. Iron-induced ulnar nerve injury in our study was mainly seen in the early stages of this study, when a small open approach to the medial supracondylar process was not used.

In difficult fractures, fractures with impaired neurocirculation and reduced clearance, and unsatisfactory pinning should be used. It is said that this can be done without increasing the risk of complications, but in this study, one afferent and one median nerve were found after shrinking the opening. In these two cases, additional damage was caused by the discovery of orbital fossils. We think that the altered anatomy of a dislocation fracture may increase the risk of further complications. Like many authors, we took a cautious approach to the associated nerve palsy. We will explore the nerve in the absence of evidence of clinical or electrophysiological improvement at 4 months postoperatively [17].

FAT EMBOLISM SYNDROME

Fat embolism (FE) and fat embolism syndrome (FES) are a clinical phenomenon characterized by the prevalence of systemic fat embolism in the circulatory system [18]. The resolution of the fat embolism disturbs the capillary layer and affects the microcirculation, causing systemic inflammatory reaction syndrome. This activity reviews the pathophysiology, diagnosis, and presentation of fat embolism syndrome and emphasizes the role of the interdisciplinary team in management (Figure 9).

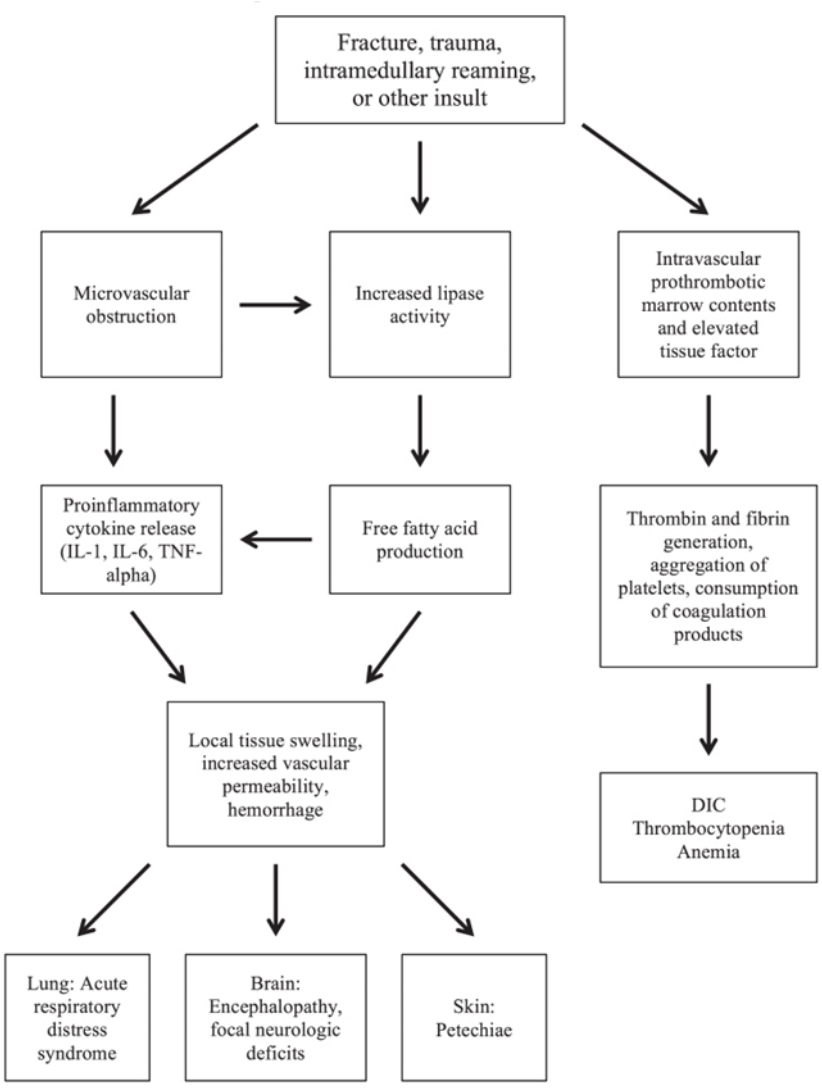


Figure 9: *Flowchart showing proposed pathophysiologic mechanisms for the clinical manifestations of fat embolism syndrome.*

There are two theories

Mechanics Theory. The mechanical theory holds that external mechanical forces, such as trauma or invasive surgery, cause FE from bone marrow or adipose tissue to enter the venous circulation. These emboli are first trapped in the pulmonary microvasculature, [19] leading to pulmonary dysfunction, but can subsequently also embolize micro-vessels in other organ systems, causing pulmonary dysfunction. multi-organism.

It remains unclear how FE has access to the arterial circulation. One theory is that the increased pressure on the heart forces fat particles to pass through the capillary membranes and into the arteries, leading to systemic circulation and thus organ dysfunction. The use of echocardiography supports this theory, allowing visualization of embolic material entering the left atrium. [20].

Theoretical biochemistry. Although FES occurs most frequently in patients with prolonged mechanical trauma, cases of FES have been reported without mechanical force acting, suggesting a biochemical component to the pathophysiology. of this syndrome. Once FEs enter the venous circulation, they cause an inflammatory and thrombotic episode that leads to rapid fibrin production and platelet aggregation. The release of free fatty acids (FFAs) into the circulation also stimulates the release of inflammatory mediators, such as C-reactive proteins, which induce FFA aggregation and, as a result, the formation of globules. fat. . This leads to symptoms of acute respiratory distress syndrome (ARDS), cardiac contractility dysfunction, elevated plasma lipase, and ultimately multiple organ failure and dysfunction [19].

Diagnosing fat embolism syndrome can be difficult because the signs and symptoms can be vague. There are no widely accepted diagnostic criteria. Several authors, based on experience and research, have established diagnostic criteria for fat embolism syndrome [21]. The most important finding in the laboratory was a decrease in oxygen tension in the arteries. Testing for lipids in urine and sputum is of little value compared with more modern diagnostic methods. Recently, bronchoalveolar lavage to detect fat cells and retinal examination for cotton spots and retinal hemorrhages have been reported to be useful in early diagnosis. Classic chest radiograph shows interstitial edema and increased peripheral vascular signs. If left untreated, fat embolism can be fatal; however, early diagnosis and prompt management can often maintain the patient until the problem is clear.

Treatment / Management

Pharmacotherapy

There is no therapeutic effect on fat or obstructive syndromes. Aptomat about testing methods, there is a method that uses dextrose converted to FFA to work. Ethanol is also used as an inhibitor of lipolysis. In clinical practice, no benefit has been demonstrated.

The use of heparin in animal experiments was thought to be beneficial but was not used in practice because of the potential bleeding risk. There is no evidence that there is a clinical benefit to using heparin in fibrotic syndrome.

Corticosteroid therapy has been proposed for the treatment of herds based on the effects of corticosteroids:

• Inhibition of active adjuvants of leukocytes

• FFA level limit

• Mature driving

• Inferior Vena Cava filter

Here is the output as a rule for the processor block. As a therapeutics project for lipodystrophy syndrome, the following set of venous filtration systems has not been fully studied.

Operational measures

Early decompression and internal fixation are highly recommended in cases of long fractures. The incidence of fat embolism syndrome is higher in patients with long fractures who are treated conservatively.

The use of internal fixatives in the treatment of long fractures significantly reduces the incidence of fat embolism syndrome.

During surgical immobilization of long fractures, care must be taken to limit intramedullary pressure, as high pressures can increase the amount of fat embolism entering the circulatory system.

Some of the techniques used in orthopedic surgery to reduce embolism include:

• Bone marrow expansion before fixation
• Femoral ventilation
• Drill small holes in the cortical bone to relieve pressure in the marrow

Some of the techniques used in orthopedic surgery to reduce embolism include:

• Bone marrow expansion before fixation
• Femoral ventilation
• Drill small holes in the cerebral cortex to relieve pressure in the medulla oblongata

None of these manipulations have been shown to reduce fat embolism syndrome.

SPONTANEOUS DEEP VEIN THROMBOSIS

This complication is very rare in children: there are only sporadic reports in the literature. In general, the clinical manifestations are similar to those in adults and include local discomfort, pain and heat, and often swelling of the extremities. Deep vein thrombosis should be confirmed by appropriate noninvasive testing and possibly venography.

Most children with thrombophlebitis or pulmonary embolism have hereditary or congenital thromboembolic disease (Figure 10). Antithrombin III activator protein deficiency antithrombin III, fibrinolysis, fibrinolysis, protein C deficiency, protein S deficiency and factor V Leiden are the more common conditions associated with an increased incidence of obstructive inflammation. veins in children.

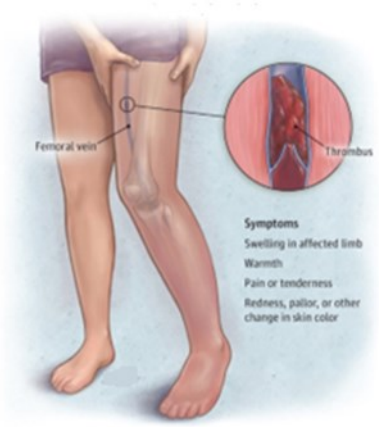


Figure 10: *Deep Vein Thrombosis at femoral vein*

When identifying children with this disease, family members should be screened because children may also have the disease and need preventive treatment. Serum lipoprotein (a) (Lp[a]) levels greater than 30 mg/dL are an important risk factor for thromboembolism in children. Children with VTE should have their serum Lp levels checked (a). It is likely that many cases go undetected. Most children respond to usual treatment, similar to adults. Initial treatment consists of heparin, followed by warfarin (Coumadin) for an appropriate time.

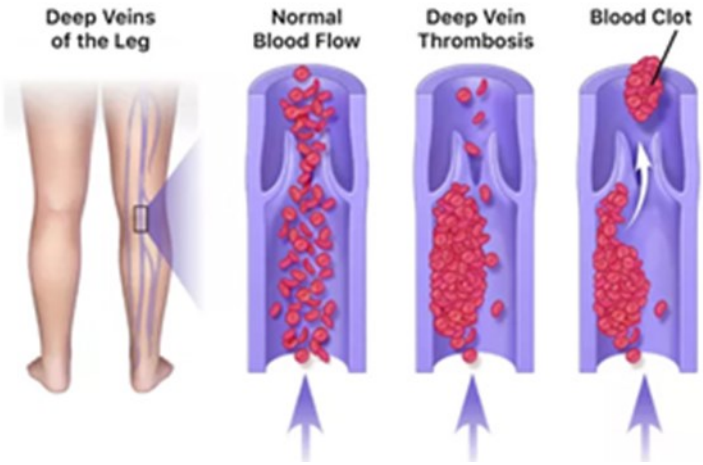


Figure 11: *Formation of a blood clot in the femoral vein.*

Pediatric venous thromboembolism (VTE) is rapidly becoming a well-recognized cause of morbidity and mortality from burns in children. Most children diagnosed with VTE have a serious underlying disease such as cancer, chronic total parenteral nutrition (TPN) dependence, or congenital heart disease. Infants and adolescents are most at risk for VTE, and the most significant risk factor is the presence of a central venous line (CVL). VTE rates vary widely according to study design and diagnostic tests used to detect thrombosis (Figures 11). Venous angiography remains the gold standard diagnostic test, although ultrasound is increasingly used due to its noninvasive nature, despite concerns about sensitivity in upper systemic VTE. Treatment of uncomplicated VTE in children initially consisted mainly of unfractionated heparin (UFH), followed by oral anticoagulation or low molecular weight heparin (LMWH) for 3 months. LMWH offers several advantages over UFH due to its longer half-life, increased bioavailability, and ease of administration and monitoring in children. Acute complications of VTE in children are numerous and include pulmonary embolism (PE), chylothorax, and superior vena cava syndrome. Long-term morbidity includes recurrent VTE, post-thrombotic syndrome, repeated general anesthesia for CVL insertion, and ultimately destruction of the superior venous system in children with repeated CVL-associated VTE. Death from VTE is rare and is mainly due to PE.

Clinical features

History and physical examination are not reliable ways to diagnose DVT [22]. Lower extremity DVT may or may not be symptomatic. Patients with lower extremity DVT usually do not have erythema, tenderness, heat, swelling, or tenderness. Patients with symptoms of proximal DVT may present with lower extremity pain, calf pain, and lower extremity swelling. The sign of Homans can be demonstrated in DVT. Most of these features lack specificity; therefore, clinical evaluation often implies the need for further evaluation. The left leg is the most common site of venous thrombosis in pregnancy and acute major venous thrombosis. This may be due to compression of the left iliac vein by the right iliac artery (May-Thurner syndrome) [23].

The treatment

The goals of DVT treatment are to prevent the spread of thrombosis, acute PE, recurrent thromboembolism, and the development of late complications such as pulmonary hypertension and post-thrombotic syndrome. Initial treatment usually includes achieving therapeutic doses of UFH or LMWH, or with fondaparinux.

Studies have shown that the effectiveness of heparin treatment is primarily dependent on the ability to achieve a critical therapeutic rate within the first 24 hours of treatment, achieving an activated partial thromboplastin time (aPTT). 1.5 times the mean of the control value or above the limit of the normal aPTT interval (aPTT ratio) from 1.5 to 2.5. This level corresponds to a blood heparin concentration of 0.3 to 0.7 U/mL by the amidolytic anti-factor Xa assay [24]. Heparin is used initially along with warfarin and discontinued after a minimum of 4 to 5 days, at which point the international normalized ratio (INR) should be between 2.0 and 3.0 (range) treatment) [24]. Long-term anticoagulation with LMWH is more effective than warfarin in preventing recurrent venous thrombosis in cancer patients without a statistically significant bleeding risk [25].

Thrombolytic therapy

Catheter-guided thrombolytics (CDTs) can be used to treat DVT as an adjunct to medical therapy [26]. Current evidence suggests that CDT can reduce clot burden and recurrence of DVT and thus prevent the formation of post-thrombotic syndrome compared with systemic anticoagulants. Pharmacological CDT is now commonly used in some centers for the treatment of acute thoracic DVT. Appropriate indications may include young adults with proximal thrombosis, long life expectancy, and relatively few comorbidities. Thrombosis that threatens the limb can also be treated with CDT, although subsequent mortality remains high.

Absolute contraindications to anticoagulants include central nervous system (CNS) bleeding, excessive gastrointestinal bleeding, retroperitoneal bleeding, massive hemoptysis, brain metastases, and stroke brain blood. growth, central nervous system damage, and significant thrombocytopenia (50,000/μL) [27].

INFECTIONS AFTER FRACTURE

Most fractures (fractures) do not lead to infection. When infection occurs after a fracture, treatment and recovery can be lengthy and complicated.

Pin site infection is always a possible complication when treating patients with maxillary fractures. The reported frequency of pin-site infection varied from 3.6 to 77.0% [28]. In Oetgen [29] only 3 (0.4%) patients out of 709 had sepsis and all were type III fractures. However, in our study, out of 125 staplers used on 106 patients, only two patients (1.6%) developed an infection, with no significant difference between the two patients. fracture.

Cause

Infection usually occurs after a broken bone because bacteria enter the body during the injury. Although uncommon, bacteria can also enter the body during surgery to place a broken bone or later, after the wound has healed.

Open fracture

When a bone breaks in such a way that bone fragments protrude from the skin or the wound penetrates the bone, it is called an "open" or complex fracture (Figures 12). The skin normally acts as a barrier to external contaminants, including bacteria. When the skin is broken, bacteria can easily travel down the broken bone and this can lead to an infection.

Surgery

During surgery to immobilize the fracture, your doctor will cut through the skin and soft tissue to access the broken bone. The risk of developing an infection in this setting is quite low, usually less than 1% in healthy individuals, although this rate varies depending on the wound and the surgery performed. Preventive antibiotics are used before surgery to reduce the risk of infection.

Risk factors

Chronic diseases that lower your immune system can put you at a higher risk of infection after a fracture. Consists of:
- Diabetes
- Immunodeficiency (such as HIV)
- Rheumatoid arthritis

You may also be more at risk due to your lifestyle choices. These include, first and foremost, smoking and the use of nicotine products. Other factors include obesity, poor nutrition or poor hygiene.

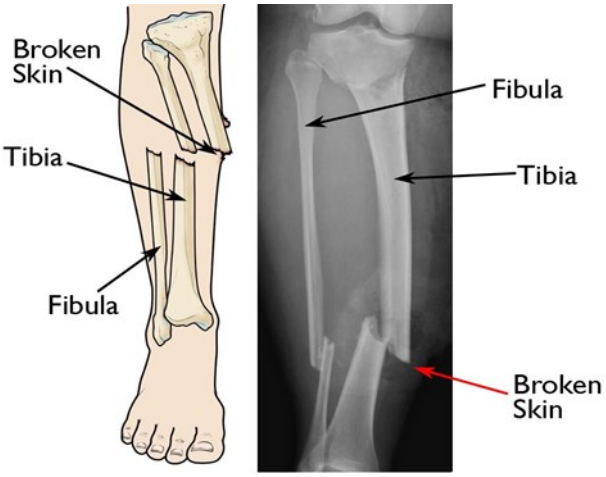


Figure 12: *Illustration and x-ray show an open fracture. The broken end of the tibia (shinbone) has torn through the soft tissues and is sticking out through the skin.*

Symptom

Infection after a fracture often causes pain, heat, redness and swelling around the affected area – more than usual (Figures 13).

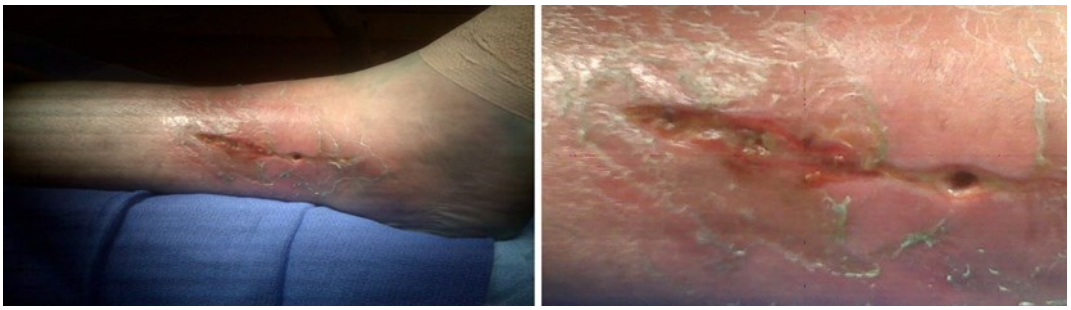


Figure 13: *(Left) This infected wound on the outside of the ankle is red and swollen. (Right) A close-up image clearly shows a moist area that appears to be draining pus.*

Test

Even if the infection seems obvious, your doctor may order an X-ray if you haven't had a recent infection. Blood tests may also be used to help diagnose an infection. They may include:
- WBC (white blood cell count)
- ESR (stability rate)
- CRP (C-reactive protein)

The treatment

A special drainage tube may be placed in the wound to help drain the pus. Antibiotic delivery systems, such as "antibiotic beads", can also be used to deliver higher concentrations of antibiotics.

Once the bacteria are identified, the doctor can choose the most effective antibiotic to treat the infection. An infectious disease specialist can work with your doctor to determine the appropriate antibiotic. Most patients will need to take antibiotics for 6 to 12 weeks.

PIN TRACT INFECTION

Percutaneous splints and external fixators are used in many cases where bone stabilization is required. In children, a plain Kirschner (K-string) is an effective immobilization method for many wounds and can even be placed on a growth plate when needed. Whenever a device protrudes through the skin, there is always concern about infection.

Some of the main factors that cause battery tracking infections:

• Open fracture

• Fixed inside

• Surgical wound

• Pin Sites

Studies that have described infection rates have shown prevalence rates to range from 0.5% to 100%, largely depending on the definition of infection used in the study. Severe infections, as described by Green [30], include those requiring hospitalization and parenteral antibiotics. It is thought that minor infections are reported less frequently, while large infection rates are more accurately reported [30].

Clinical features

- New or increasing pain
- Heat
- Red
- Swelling
- Rust/Discharge is green or cloudy
- Tender

Any orthopedic protrusion through the skin raises concerns about infection. External staples and cords, K-strings, and Steinman pins are all devices that can be used through the skin. In children, it is quite common practice to use a percutaneous K-wire to stabilize fractures. The prevalence of staple groove infection varies from less than 1% to 21% [31].

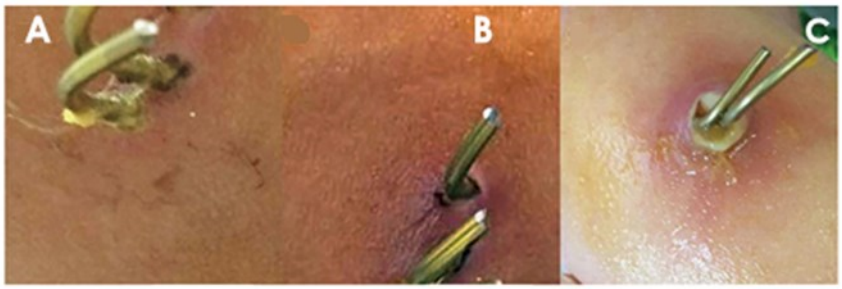


Figure 14 A-C: *The distribution of pin site condition. A. Normal skin. B. Slight redness as grade I. C. Purulent discharge as grade II.*

Green's study [30] showed an infection rate of 0.5% for staples and patches and 8.3% for external fixators. A review of the literature by Green [28] in 1983 noted that studies published before 1950 had an average infection rate of 4.2%, while those published after 1950 had an average infection rate of 4.2%. The infection rate was 4.2%. 5.8% when all percutaneous fixation methods are included.

Herstik et al. [31] found that hospitalization was necessary to treat a stapled groove infection in 0.46% of cases involving Steinman and K-string staples. Studies in adults have documented increased rates of infection. K-string in the hand was 2.2% [32] and the infection rate was 21% with small external fixations around the wrist. Mild infections may not be reported because they do not change treatment (Figures 14).

Treatment

The literature has reported rates of major infections ranging from 1% to >25%. [32]. Most infections are superficial and resolve with staples and oral antibiotics [30]. In rare cases where the infection is deep or involves the joint, drainage, sterilization, and intravenous therapy usually resolve the infection without significant sequelae.

HYPERCALCEMIA OF IMMOBILIZATION

Many children develop hypercalcemia after lying immobile due to fractures. Cristofaro and Brink reported that 7 out of 20 children had an elevated serum calcium level of 10.7 to 13.2 dL (normal is 8.5 to 10.5 dL). Urinary calcium excretion peaks about 4 weeks after inactivity and may return to normal with activity.

Intravenous fluids and corticosteroids have been reported to be successful in lowering serum calcium levels until mobilization. Usually, a low calcium diet is recommended. Plicamycin (also known as mithramycin) also effectively reduces calcium by directly antagonizing bone resorption or by interfering with parathyroid hormone metabolism.

The homeostasis of calcium is complex because the bones, gastrointestinal tract, and kidneys all affect the balance of calcium. Alterations in calcium homeostasis from any of these organ systems can lead to changes in serum calcium. Therefore, hypercalcemia can be divided into three categories: increased bone calcium reabsorption (hypercalcemia reabsorption), increased gastrointestinal calcium absorption (hypercalcemia reabsorption) and enhanced resorption of calcium. renal calcium absorption (hypercalcemia reabsorption) (Figure 15).

Immobilization-induced hypercalcemia may be present in patients with spinal cord injury, multiple fractures, or Landry -Guillain-Barre Syndrome. It is thought to be due to increased bone resorption and decreased bone formation, clinically characterized by increased serum calcium levels, hypercalciuria, increased risk of urolithiasis, and renal failure. Traditional treatments can affect the depth of therapy delivered in a comprehensive rehabilitation program.

Since the initial description by Albright et al in 1941 of "acute bone atrophy" complicating immobilization in a 14-year-old boy, it has been accepted that long-term immobilization can lead to mineralization. osteomalacia, hypercalcemia, and sometimes decreased renal function [33]. However, studies of renal function have only been performed in a limited number of patients with immobility-induced hypercalcemia [34].

Albright et al's original patient was a hyperactive adolescent boy who had previously developed bone atrophy, hypercalcemia, and decreased renal function during immobilization due to a femoral fracture. Two exploratory neck surgeries revealed no parathyroid gland abnormalities.

The patient's symptoms disappeared after exercise. In 1952, Dodd et al. [33] described a 12-year-old boy with osteoporosis, hypercalcemia, renal failure, hypertension, and complex encephalopathy immobilized by multiple fractures. During his period of high hypercalcemia (14.9 mg/100 ml), the patient's urea clearance decreased to 43.5% of normal. He became normal and his urea clearance returned to normal after exercise [33]; Halvorsen, 1954, described a similar case of a 9-year-old boy with a fracture of his right femur immobilized for 5 weeks. Immobility-induced hypercalcemia has been described in another young male [35] in a young female and in two adults with Paget's disease who developed immobility-induced hypercalcemia due to fracture.

Therapy

Different approaches to the treatment of hypercalcaemia of nonparathyroid origin have been described.

Intravenous fluids and corticosteroids have been reported to be successful in lowering serum calcium levels until mobilization. Usually, a low calcium diet is recommended. Plicamycin (also known as mithramycin) also effectively reduces calcium by directly antagonizing bone resorption or by interfering with parathyroid hormone metabolism. In addition, calcitonin has been reported to effectively reduce serum calcium levels by inhibiting bone resorption.

The important role of furosemide in the treatment of episodes of hypercalcemia secondary to malignancy or vitamin D toxicity has been demonstrated [36]. Although the patient was given furosemide, other forms of complementary therapy were used; therefore, it is not possible to assess the specific beneficial effects of furosemide. The use of oral phosphates to reduce serum calcium has been used successfully and may be of long-term benefit in the treatment of hypercalcemia.

However, Hulley et al. have shown that oral phosphate therapy leads to negative calcium balance only during the first [37] week of immobilization. Then the calcium balance is negative in noni. it does not reduce bone resorption in this state. Intravenous phosphate therapy should be used with caution because of the potential for unwarranted metastatic calcification and severe acute hypocalcemia associated with hypotension. The use of steroids (25 to 40 mg/m2/day of prednisone for one to two weeks) is considered to be one of the more successful methods of treating hypercalcemia due to malignancy and vitamin D toxicity.

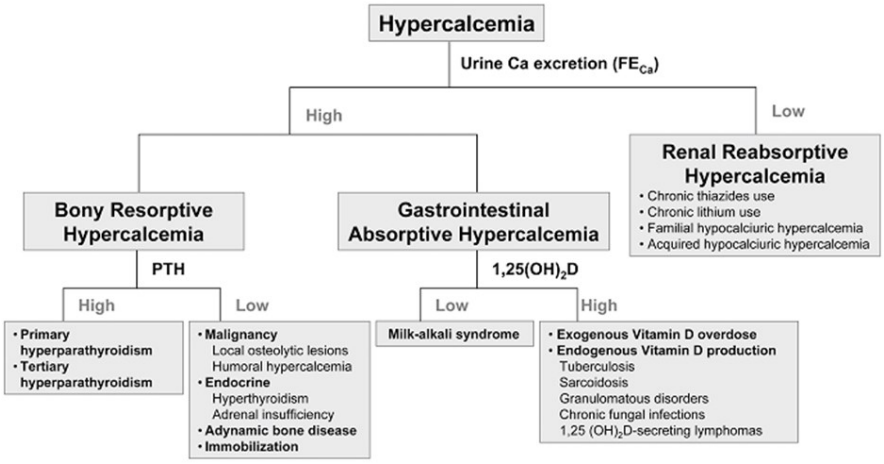


Figure 15: *Common causes of hypercalcemia.*

OSTEOMYELITIS

Acute hematogenous osteomyelitis mainly affects children because their supraclavicular regions are highly vascular and susceptible to even mild trauma. More than half of cases of acute hematopoietic osteomyelitis in children occur in patients younger than 5 years of age [38].

Posttraumatic osteomyelitis may occur in 26% of open fractures. However, acute hematopoietic osteomyelitis following closed fracture is an uncommon complication in adults, with clavicle involvement being extremely rare. Reports in the literature agree that early and aggressive treatment is strongly indicated.

The case presented describes a unique example of acute osteomyelitis with rapid progression, serious complications, and death after a missed clavicle fracture. Furthermore, a brief literature review is presented of previously reported articles on acute osteomyelitis following closed fracture.

The case presented demonstrates the importance of early diagnosis of acute osteomyelitis and the appropriate treatment of closed fractures, as well as the importance of prophylactic treatment of skin and soft tissue injuries, which can be fatal. potential risk of local and systemic infections. This is especially important in chronically ill or immunocompromised patients, who are at high risk of developing these complications.

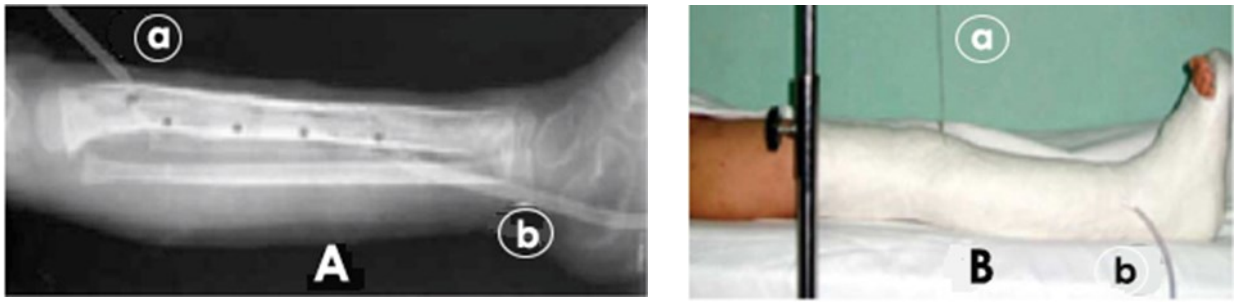
Waldvogel et al [39] described clavicle involvement in an infectious process as uncommon and often secondary to spread of infection from adjacent areas and often associated with other infections. Predisposing factors such as head and neck surgery, radiation therapy, subclavian veins. catheterization or immunosuppression in transplant patients. Concurrent with this statement, various authors have reported rare cases of clavicle inflammation following central line placement. Swan–Ganz catheterization [40] is traumatic [41] and is a complication after head and neck surgery. Our brief literature review revealed that Staphylococcus aureus appears to be the most commonly isolated pathogen in the reported cases of hematopoietic osteomyelitis following closed fractures.

Regarding the underlying conditions that may have contributed to this devastating clinical course of hematopoietic osteomyelitis in the patient, there are several factors that can be considered. One most likely factor is the patient's immunity, which may have been impaired due to his long history of chronic alcoholism. E. coli has been described in the literature as Gram-negative bacilli commonly isolated in sepsis E. coli and S. pneumoniae are pathogens commonly isolated in sepsis cases in chronic alcoholics than the general population. The most common foci of bacteremia caused by E. coli are urinary tract infections; however, Gram-negative bacilli in acute, debilitated, alcoholic patients; Diabetics and people with chronic bronchitis can also develop pneumonia caused by E. coli.

In addition to severe anemia, concurrent S. pneumoniae bacteremia and right lower lobe pneumonia showed a poor immune status of the patients in this study. To search for the probable primary focus of E. coli bacteremia, several different smears were performed and E. coli was isolated from a deep skin wound on the left fourth toe of the patient. All other tests were negative (pleural fluid sample, Sanford urinalysis). This finding highlights the importance of skin wound prophylaxis in immunocompromised patients, especially in specific body regions with a greater bacterial burden. Preventing infection requires gentle wound care to prevent further injury, immersion, or alteration of the normal microbiome. If the wound becomes infected, it is important that it be diagnosed and treated specifically.

Imaging techniques play an important role in early diagnosis and monitoring of trauma. In cases where osteomyelitis develops, early diagnosis is crucial, as prompt antibiotic therapy and surgical intervention can prevent bone necrosis. Incomplete or late diagnosis significantly reduces cure rates and increases the severity of complications and morbidity.

In many cases, although osteomyelitis was detected as soon as the patient came to the hospital for the second time, the disease was already in a severe stage. despite the prompts treatment with antibiotics and surgery, the infection developed rapidly and aggressively, and the patient's condition deteriorated. Early diagnosis of acute osteomyelitis and appropriate treatment of closed fractures are crucial for a successful treatment outcome. Early diagnosis is important, as late diagnosis can lead to serious complications, such as complicated osteomyelitis and sepsis, which can even be fatal.



Figures 16 A-B: A*. System of irrigation, (a) catheter was placed in medullary canal and (b) drainage tube was placed at the inferior border of the tibia.* ***B****. System of irrigation with gentamycin (a, catheter with a 8 Fr, b, drainage tube with a 16 Fr and it is connected with vacuum-associated closure).*

The patient had acute post-traumatic osteomyelitis with cortical perforation with continuous antibiotic irrigation, to mediate blood (Figures 16) [41]. This is especially important in patients with chronic diseases or immunocompromised conditions. Although uncommon, osteomyelitis should be considered as a possible cause of clinical deterioration in patients with a history of recent trauma.

DELAYED UNION

It is estimated that there are 100,000 non-healing fractures each year in the United States. [42]. The reported incidence and prevalence of non-healing conditions vary considerably based on the anatomical region and the criteria used to define the non-healing condition. This variability reflects the overall complexity of understanding the epidemiology of disunity. Risk factors for uncoordinated status can be described as patient-dependent or patient-independent. Identified patient-dependent risk factors include advanced age, various medical conditions, sex, smoking, use of non-steroidal anti -inflammatory drugs, various genetic disorders, metabolic disease metabolism and nutritional deficiencies [43]. Patient-independent factors include fracture type, location and displacement, severity of soft tissue injury, degree of bone loss, quality of surgical treatment, and presence or absence of infection. [44].

Slow healing of broken bones

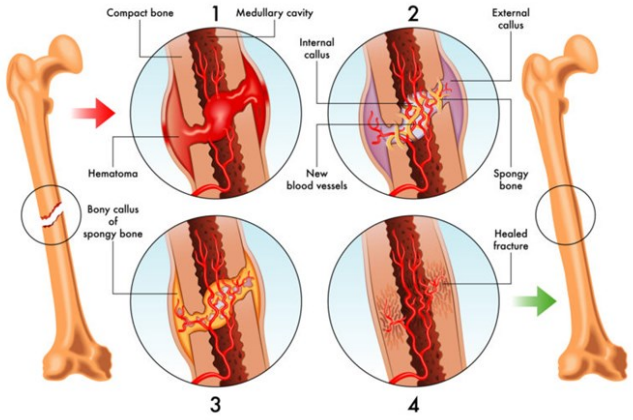
However, the healing process may not take place in some special cases as we expect, leading to serious health problems. About 10% of fractures will take longer to heal because they may not bond together due to various factors. These healing complications are called dislocation fractures, dislocation fractures, and delayed fractures, which sometimes require additional treatment to correct.

What is the delay matching rift?

Late link fractures are sometimes confused with non-union fractures; Nonunion fractures are the result of delayed fusion. A fracture is considered a "non-bonding" fracture if it does not heal or requires further surgery to heal completely.

Delayed healing time of conjugate fractures

While "Delayed Union" is a fracture in which the broken bone is lost or takes longer to heal than expected. Most of the time, however, it is likely to heal on its own without additional surgery (Figures 17).



Figures 17*. Union of bone takes place in several stages: 1. hematoma formation & inflammation; 2. Cellular proliferation; 3. Callus formation & Consolidation, 4. Remodeling*
Risk factors for bone retardation include open fractures, high-energy fractures with bone resorption, severe soft tissue trauma, bone loss, and advanced age.

The cause of the alliance breakup was delayed

The healing process takes place in different stages, including bone formation, soft scar tissue formation, hard bone formation and finally regeneration. However, this process is disturbed because there is no wound on the X-ray or the broken bone is unstable for a period of 4 to 6 months after the injury.

Some other factors that can lead to a delayed union breakup are as follows:

- Old
- Malnutrition
- Metabolic diseases such as diabetes
- Smoke
- Alcohol consumption
- General health
- Use of NSAIDs
- Loss of blood supply

Pain from slow fracture

In most patients, long bone fractures will usually heal without any complications. A slow rate of conjugate fractures is observed in a small percentage of fractures, about 2 to 10%. Treatment of previous fractures should be continued. Damaged pulp wrappers are replaced if necessary. Traction may be reduced. Functional bone splints can be performed to promote bone fusion. Additional nonsurgical treatments include electrical stimulation, extracorporeal shock wave therapy, and ultrasound. Surgical treatment includes - sterilization and hardware removal, osteogenesis, intramedullary nailing and external fixation.

Delayed alliance error management

Patients need emotional support from family members to be admitted to the hospital as soon as possible to reduce the risk of late involvement. An appropriate treatment modality, surgical or conservative, is suggested to correct the problem. During treatment, bone remodeling alternatives such as bone grafts and PRP can also promote faster wound healing and reduce infection. For more information or if you feel discomfort even after treatment, consult Dr. Vasudeva Juvvadi, one of the best orthopedic surgeons for the treatment of delayed fractures in Hyderabad, Gachibowli.

NONUNIONS

With modern treatments, most fractures (fractures) heal without problems. After the broken bone is processed, new bone tissue begins to form and connect the broken bone pieces.

Several authors have conducted studies and learned about the general and local risk factors that can lead to the development of pseudogout. However, we must consider whether there are mathematical models and/or algorithms to help us understand this pathology. During the 1990s, authors such as Kenwright [45], Lavini and Renzi Brivio, Caon and Caraffa, Marsh, published important research evidence on the post-fracture bone repair process. These authors derived their concepts and intuitions from researchers such as Pauwels (1940s), who reversed the "biological incompetence" concept of pseudoarticular disease, by demonstrating that in Given the right mechanical conditions, the bone tissue can complete the bio-bonding process, identifying compressive agents as favorable versus unfavorable forces under stress and torque. Judet in 1958 [46] pointed out the importance of angiogenesis in the prosthetic environment. He also showed that osteogenesis imperfecta, which can be thought of as opacity of bone fragments in X-rays, is due to an unidentifiable strong osteogenic reaction (hypertrophy) and not to necrosis as one would expect. still believed at the time. 4° Muller [47], the father of the AO School in 1966, applied Pauwels' concepts of mechanical stability. He defined delayed bone healing as the absence of radiographic evidence of wound healing after 4 months and pseudogout after 8 months [48]. Weber and Cech [49] (1976) developed a classification system for prosthetics that is commonly used to this day in the clinical setting.
The Anglo-Saxon School, with KenwMght, in 1991 reported a monitoring system for fracture alignment processes, introducing the Angleometer (radiometer) as an instrument for this type of survey, applied used for single-axis external fixators [45].

Bone healing

For bone healing to take place, the bone needs to be stable and have an adequate blood supply. Good nutrition also plays a role in bone healing.

• The stability. All fracture treatment follows one basic principle: the broken pieces must be put back in place and not moved out of place until they have healed.

• Blood supply. Blood provides the ingredients needed to heal fractures.

Nutrition

Broken bones also need adequate nutrition to heal. Eating a healthy and balanced diet that includes protein, calcium, vitamin C and vitamin D is the best way to ensure adequate nutrition.

Risk factors

Several factors increase the risk of disunity.

• Tobacco or nicotine use of any kind (smoking, chewing tobacco, and the use of nicotine gum or patches) interferes with bone healing and increases the likelihood of smoking cessation.
• Old
• Severe anemia
• Diabetes
• Low vitamin D levels
• Hypothyroidism
• Poor nutrition
• Medicines include anti-inflammatory drugs such as aspirin, ibuprofen and prednisone. Physicians and patients should always discuss the risks and benefits of using these drugs during fracture healing.
• Infection
• An open or complex compound interrupt

Blood supply

Flight is more likely if the injured frame has a limited supply of blood.

• Some frames, such as the leg bones, have inherent stability and an excellent blood supply.
• Some bones, such as the upper femur (head and neck femur) and the small wrist (backbone), have a limited blood supply.
• Some bones, such as the bony plate (cheekbone), have a moderate blood supply, however, an injury can break it.

Symptom

To combat nonadherence, doctors use imaging studies that provide detailed images of the bone and surrounding soft tissues. Depending on the panel involved, these tests may include X-rays, computed tomography (CT), and magnetic resonance imaging (MRI). Imaging studies allow the doctor to see the broken bone plate and monitor its healing progress.

Nonunion may be suspected if your doctor finds one or more of the following:

• Where not at the partion

• Can't extend the time the board slides over the sliding positions

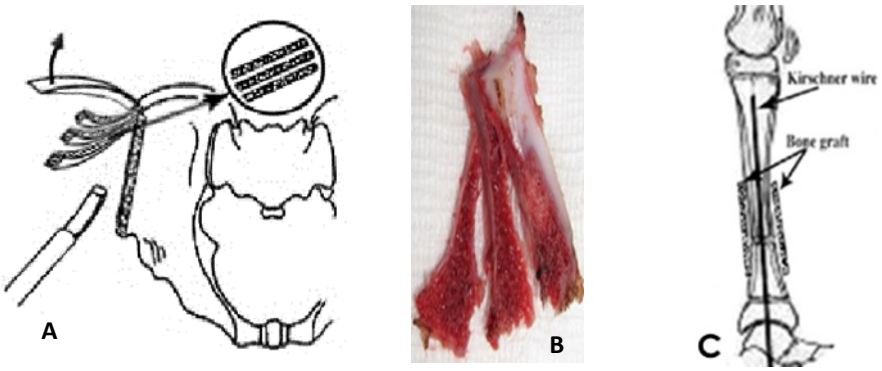
• No growth in healthy course when comparing repeated imaging studies over several months

• Insufficient healing for a sufficient amount of time to restore normal health

If the doctor suspects no planets, they may order blood tests to investigate the cause. These screening tests may reveal an infection or other medical condition that could delay a too benign program, such as anemia or diabetes.

Event

Non-surgical and surgical treatments for astronauts both have advantages and disadvantages. More than one option may be appropriate.



Figures 18 A-C: *A-B. Cortical cortical cancellous bone graft is harvested from Ilium. C: for Tibial pseudarthrose.*

Non-surgical treatment

Some pilots can be handled in a non-combat manner. The most common non-surgical treatment is a bone stimulator. This small device emits ultrasonic or pulsed electromagnetic waves that stimulate the healing process. The patient places the stimulator on the child's skin from 20 minutes to several hours a day. This provision must be applied daily to be effective.

Surgical treatment

Frame grafting. During this process, the frame from another part of the body at the site of the frame "starts" too heals (Figures 18) [50]. The framework provides a new teacher with which to develop the framework. Bone grafts also provide healthy bone cells and natural chemicals that the body needs to heal bones.

Allograft (corpse bone graft). Allogeneic (cadaveric) bone grafting avoids the removal of bone from the patient, and thus, reduces pain in non-graft treatment. Like traditional bone grafting, this method provides a scaffold for the patient's bone to heal over the graft-free area. Over time, the patient's bones will replace the cadaver's bones. Although there is a theoretical risk of infection, bone grafts from cadavers are processed and disinfected to minimize this risk.

Depending on the type of phinion, any of the above materials or a combination of materials may be used to immobilize the phinion. Bone grafting (or bone graft substitutes) alone does not provide stability to the fracture site. Unless the uncombined part is inherently stable, you may also need additional surgical procedures (internal or external fixation) to improve stability.

Fixed outside. External fixation also stabilizes the injured bone. The surgeon attaches a rigid frame to the outside of the injured arm or leg. The frame is attached to the bone with wire or pins. External fixation may be used to increase stability of the fracture site if instability helps to cause disintegration. External fixation can treat casts in patients who also have bone loss and/or infection.

RE - DISPLACEMENT

Fractures of the distal radius and forearm are among the most common injuries to orthopedic surgeons and the most common bone trauma requiring surgical care in children. Distal radius fractures account for 2% of all fractures in children. Of these, 60% of fractures occurred at the osteoarticular level and 14% affected the distal radius, accounting for 39% of all bony injuries in children. Metaphyseal injuries are further subdivided into toroidal ('lock') fractures; greenstick fracture, with subclassification of tension; Compression fractures and bilateral ('complete') fractures.

According to Oetegen [51], because of the gradual ossification of the distal humerus, younger patients with a greater proportion of the distal humerus by cartilage may have less stable fixation by pins penetrating the skin leading to a loss of subsequent fracture reduction.

It has been reported that up to 34% of distal radius fractures in children can be displaced soon after correction. Factors associated with childhood orthodontics include initial displacement (bayonet placement, displacement greater than 50% and angle greater than 30°), isolated distal radius fracture , related ulnar fractures of the same degree, inadequate initial closure correction, poor cast technique, muscle atrophy, and initial resolution of soft tissue swelling with cast.

Given the proximity of these cracks to the distant radial physics, the possibility of remodeling is very high. According to several studies, physical fractures with an inclination angle of less than 15° and shortening up to 1 cm will show complete regeneration without functional impairment in skeletally immature patients. The dorsal-volar angle of 20 to 25° and the radial-pillar deviation of 10° may be developmental in young patients. The remodeling potential in the longitudinal plane is 0.9° per month and in the coronal plane is about 0.8° per month [52].

Recent studies suggest that an angle of inclination greater than 10° may not reproduce satisfactorily in patients over 6 years of age. Malrotation will not be modified [53]. Late re-manipulation increases the rate of growth arrest [54]. This rate is as high as 4–5% and is associated with displaced distal ulnar fractures; The risk is approaching 50%. Therefore, accurate early correction is necessary and equally important to maintain manipulation until the fracture heals.

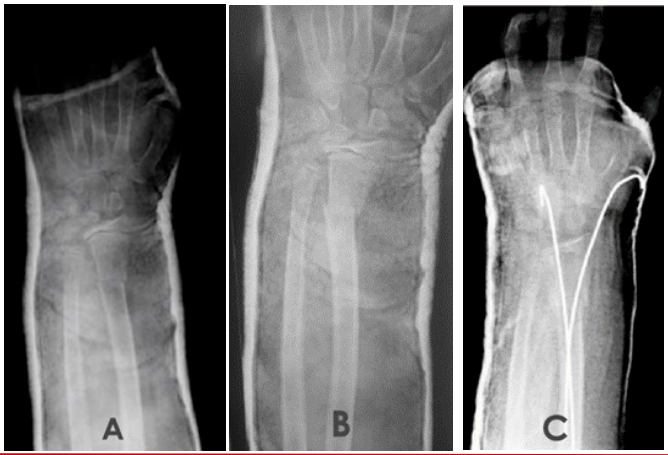
Several studies have attempted to identify risk factors associated with loss of fat loss. These include complete initial displacement (bayonet placement, >50% displacement and >30° angle), isolated distal radius fracture, related ulnar fracture of the same degree, initial closed manipulation inadequate cast technique, muscle atrophy, and soft tissue deformity initially with swelling, during cast.

Mani et al. [55] classified the initial displacement as 'Severe' with radial displacement of more than half the diameter of the bone on both radiographs. They have a 60% or higher probability of failure. The 'Mild' category with radial displacement of less than half the diameter of the bone on both radiographs, has a failure probability of 8%. Proctor et al. [56] identified two factors that increase the chances of re-shifting, the presence of complete initial displacement and the failure to achieve a perfect reduction.

Likewise, the quality of fracture reduction and casting is also more important. In several prospective randomized trials, the risk of incapacitation was higher with incomplete reduction and when the actor index was greater than 0.7 [53].

McLauchlan et al [57] performed a prospective, randomized clinical trial in 68 children treated with closed and fixed manipulation compared with immediate percutaneous staples and fixed casts. found the rate of joint loss in 21% of the cast group and 0% of the cast group. Friberg [52] similarly performed a prospective, randomized trial of closed manipulation and cast fixation versus staple fixation in 34 patients over 10 years of age with severe distal radius fractures. Thirty-nine percent of cast patients lost their ability to reduce the need for repeat manipulation.

In fractures of the diaphragm joint, the pinning technique can be difficult due to the oblique direction and proximity to the growth plate. It should be positioned obliquely at the edge of the vertebrae, not only to avoid physis and reduce the risk of growth arrest, but also to provide a support in the back to reduce the risk of displacement (Figures 19).

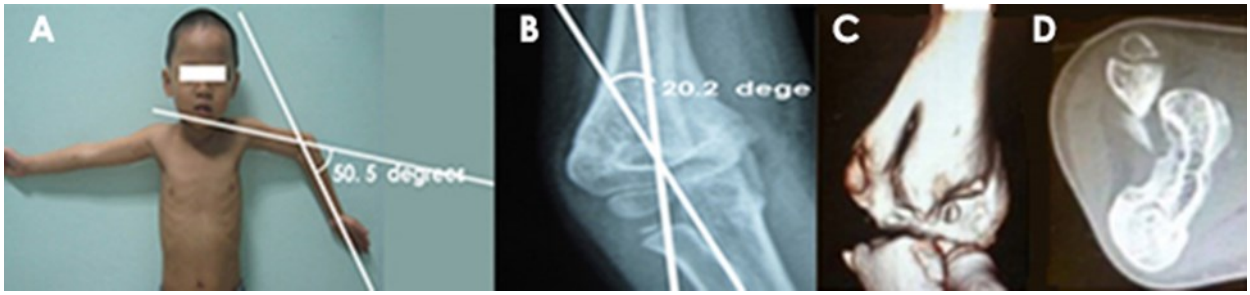


Figures 19 A-B. *Case report of an obese patient. A. Post-reduction casting X-ray; B. One-week postreduction X-ray with loss of reduction; C. Surgical treatment of the fracture.*

In adolescents who have less than 2 years left for bone growth, a formal laparotomy and volar plating can be performed. Open manipulation may also be indicated in cases of open fractures and intractable fractures where soft tissue interlacing precludes the ability to achieve an acceptable closed correction. Also, distal ulnar fractures are associated with a higher risk of displacement despite good cast technique. Although appropriate reduction in radius fractures also often reduce ulnar fractures, intervention is recommended for displacements greater than 50% or 20° angulation [58].

CUBITUS VARUS

The cause of cubitus varus remains uncertain. Most authors believe that the medial curvature is a consequence of fracture misalignment rather than growth arrest. Angular and rotational distortions are believed to be the cause of internal curvature (Figures 19). Posterior displacement for a higher Baumann value indicates an internal curvilinear mass deformation, while posterior posterior displacement for a lower Baumann value indicates cubitus valgus [59].



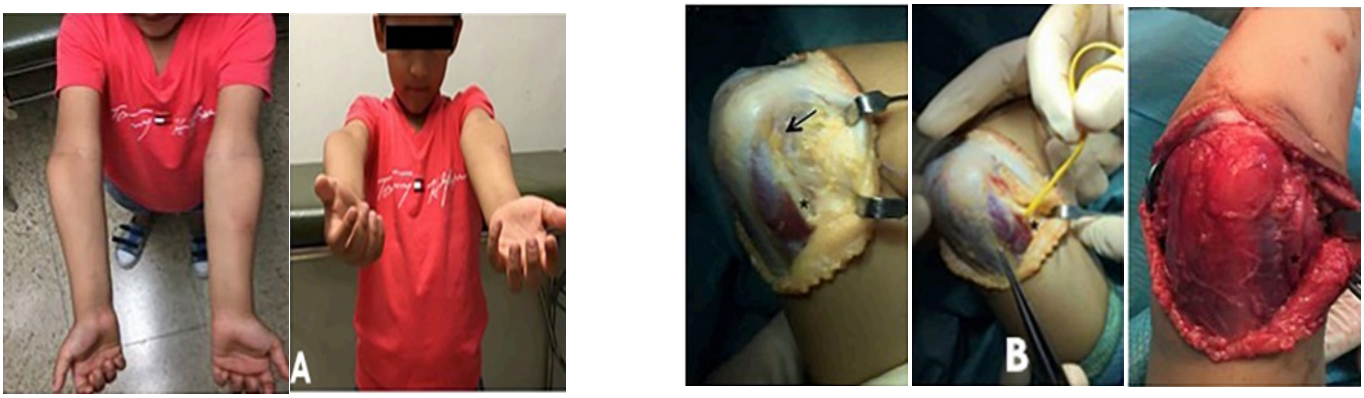
Figures 20 A-D: *Cubitus varus deformity. A. Clinical, B. Roentgenographic, C. MRI, and D. CT scan.*

In a study published by Moraleda et al [60] the incidence of scoliosis in untreated extension type II fractures was reported to be as high as 26.1%. For that reason, the best way to avoid humerus in the humerus seems to be to achieve and maintain anatomical fracture reduction, with particular attention to reconstructing contralateral rotation of the humerus.

O'Driscoll et al. [61] assert that the medial torticollis leads to two biomechanical disorders: 1) an inward displacement of the mechanical axis of the upper extremity and, consequently, the load-bearing lateral ligament complex. pull increases and becomes weakened; and 2) the triceps are displaced inward and the force vectors of the triceps are displaced resulting in the external (supine) moment arm above the ulna. Late ulnar nerve palsy with anterior dislocation of the nerve has been described (Figure 20) Fracture of the medial triceps may result from inward displacement of the triceps as well as medial rotation of the distal humerus.

Various osteotomy methods have been proposed for the treatment of scoliosis. We have designed a modification of stepwise osteotomy (stepped osteotomy) to achieve more deformity correction effect.

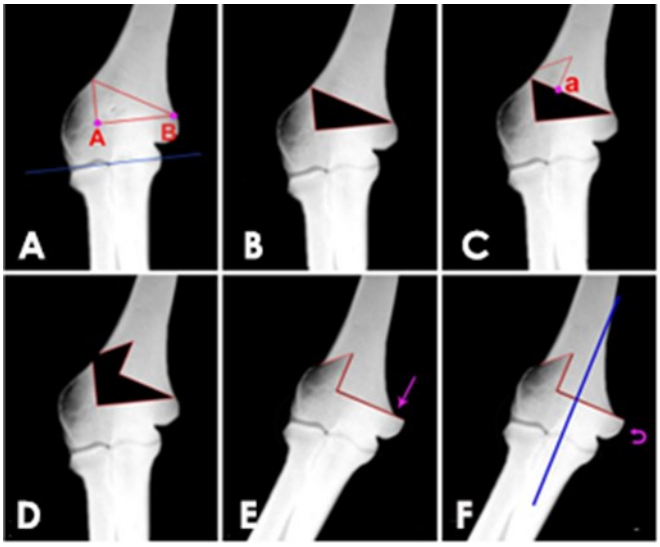
Before and after surgery, we performed clinical and radiological assessments. The HEW and LPI angles were assessed radiographically (Figures 21). On extended upper extremity radiographs with the forearm fully supine, the angle created at the intersection of the longitudinal axes of the forearm and the humerus is measured by the HEW angle. The longitudinal axis of the forearm was determined using the midpoints of the upper and lower transverse lines, bounded by the medial and lateral cortex of the ulna and the radius [63], respectively.



Figures. 21 A-B: A*. Ulnar nerve palsy is one of the complications related to cubitus varus deformity. In the figure,* ***B****. Anterior transposition of the nerve is performed before the osteotomy for correction the deformity.*

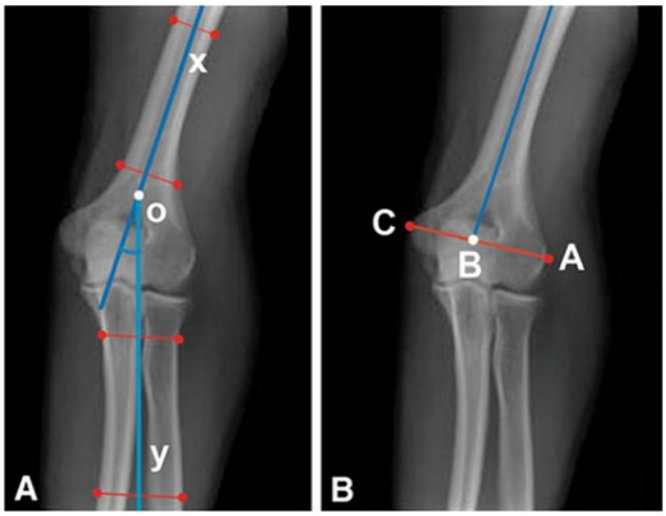
A posterior longitudinal skin incision in the posterior midline of the distal humerus was made, while the ulnar nerve was decompressed and protected. In complex cases where we needed to adjust the height of the longitudinal and axial planes, we used the olecranon osteotomy for better contact and fixation, but in children and for ring adjustment. simply, we used the near-peak method. Wedge osteotomy was performed proportional to the degree of correction by comparing the HEW angle of both upper extremities. In contrast to the technique described by DeRosa and Graziano [62], the lower margin of this right triangle (Line AB) (Figures 22) is drawn parallel to the seam 0.5 cm above the speculum (Figure 22A). Then, from the midpoint (Point B) of this first line, a second line (BC) is drawn, making an angle (Angle B) between the first and second line equal to the desired corrected HEW angle. . Next, from the lateral end of the second line (Point C), a third line perpendicular to the first line (CA) is drawn. Finally, our desired right triangle was outlined and removed (Figues. 22B, 22C, 22E).

By comparing the LPI of both upper extremities before surgery, we calculated the required amount of transverse displacement and adjusted it by distal displacement, then we cut a notch on the corresponding proximal segment. with the vertex of the distal segment and the second triangle removed. Any prominent bone remaining at the edge of the bone cut is clipped. Coronal correction of distortion and translation was achieved as needed. Any excessive internal rotation was corrected using the midline of the lower margin as a hinge and rotated the distal part as much as was measured preoperatively (Figure. 22D, 22E, 21F). Due to previous surgery, some patients lose their entire elbow extensor. We corrected any flexural deformities by sloping the lower border of the osteotomy triangle for superior esthetic results. The osteotomy is initiated with two Steinmann pins. We then examined the HEW angle, center and side highlights, and elbow ROM. To obtain a firm fixation, an inter-piece delay screw through the corresponding point and groove was used. For adults or if there is any doubt about the possibility of fixation, we apply a reconstructive plate. Actively supported ROM exercises begin 2 weeks after suture removal. A posterior brace is used for protection between periods of exercise until radiographic and clinical fusion occurs.



Figures 22 A-F: A. *The proposed triangle is shown. AB is parallel to the articular surface and 0.5 cm above the olecranon fossa, Angle B is as much as the coronal correction, and Angle A is 90.* ***B****. The triangle is removed and the lateral bone spike remains on the distal fragment.* ***C****. In the proximal fragment, according to the amount of translation required at Point a, the notch for the distal fragment spike is proposed.* ***D****. The second triangle is removed.* ***E.*** *Coronal correction of the deformity is achieved, and translation applied as needed.* ***F.*** *Excessive internal rotation deformity is corrected using the middle of the inferior margins as a hinge*

Before and after surgery, we performed clinical and radiological assessments. The HEW and LPI angles were assessed radiographically (Figure 23). On extended upper extremity radiographs with the forearm fully supine, the angle created at the intersection of the longitudinal axes of the forearm and the humerus is measured by the HEW angle.



Figures 23 A–B: *Measurements of the A. HEW angle and B. LPI are shown. A is the most prominent lateral point and C is the most prominence medial point. x is axial axis of the humerus and y is the axial axis of the forearm. HEW angle=xOy angle. LPI= (AB BC)/AC%.*

The longitudinal axis of the forearm was determined using the midpoints of the upper and lower transverse lines, bounded by the medial and lateral cortex of the ulna and the radius [63], respectively.

**** Dome technique*** [64]

The supracondylar osteotomy was performed according to the technique described by Tien et al. [64] (Figures 24, 25, 26, 27). After the patient was under general anesthesia, the distal humerus was exposed through a posterior approach by lifting a tongue-shaped flap from triceps aponeurosis and separation of the triceps. The periosteum was incised in the midline and reflected on both sides. The intersection of the midline axis and the upper margin of the olecranon pit (point O) is indicated as the center of the dome. With segment OA as the base, a second line (line OB) is drawn from point O to form an angle equal to the expected correction angle (a). The length of the OB segment forming the radius of the desired arch and arch is marked with Bovie. A 2.5 mm drill bit was used to make drill holes along the marked position of the dome. To avoid any inadvertent damage to the anterior neurovascular structures, great care was taken not to completely drill the anterior cingulate cortex. The osteotomy was completed using a quarter-inch osteotomy.

Before rotating the distal segment, segment AB was clipped and the segment edges smoothed to allow free rotation inside the arc. The distal segment is then rotated along the arc until point A in the distal segment overlaps point B on the proximal segment, thus re-aligning the elbow as planned. The bone incision was then fixed with two Kirschner wires (1.8 mm) inserted in a diagonal fashion.

Removal of the meniscus leaves two fragments of unequal width and articulates in the medial cortex, whereas closure of the osteotomy effectively shifts the distal piece to the side, thereby rendering the condylar condylar. side is more prominent and affects the aesthetic result. Some authors suggest inward displacement of the distal segment to improve the esthetic outcome.

A plaster on the elbow of the Paris plate with the elbow flexed at 90 degrees was worn after surgery for 3 weeks, and then gradually active mobility was started. The Kirschner cords were removed after 6 to 8 weeks.

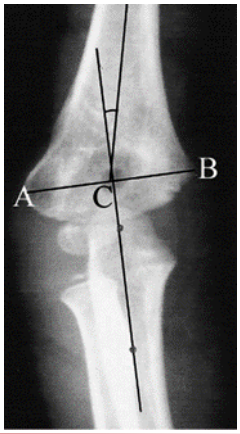
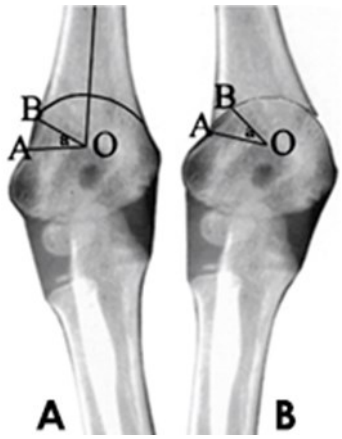
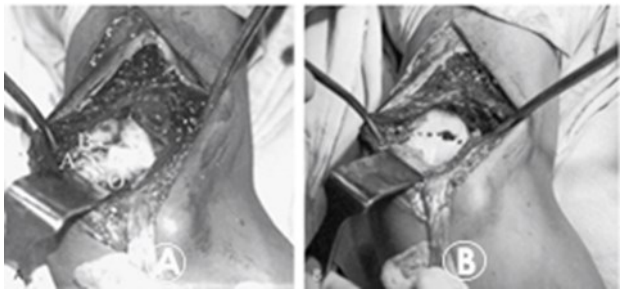


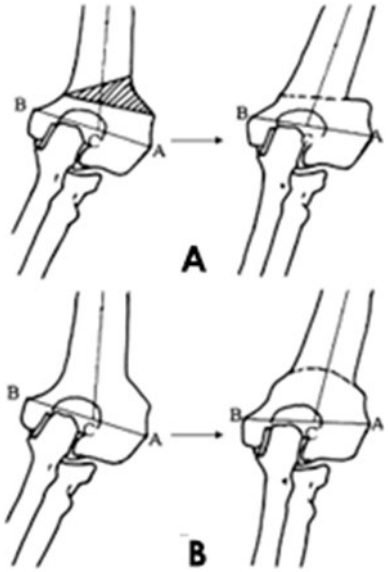
Figure 24: *The lateral prominence index (LPI) = (AC – BC) 100/AB. There is usually a slight medial prominence, making the LPI predominantly negative. Carrying angle is the angle between the midhumeral and the ulnar axes. Midpoints were determined for the humerus at the flare of the metaphysis and in the distal diaphysis. Midpoints for the ulna were determined at the level of the radial tubercle and at the most proximal ossification.*



Figures 25 A-B: *Dome supracondylar osteotomy as described by Tien et al.* ***A,*** *the intersection of the midhumeral axis and the upper border of the olecranon fossa was designated as the center of the dome (point O). Point A was marked at the junction of the periosteum and perichondrium. With segment OA as the base, a second line, OB, was drawn according to the planned angle of correction (a). Point B acted as the starting point of the osteotomy, and a dome was drawn with OB as the radius of the arc.* ***B.*** *Completed osteotomy; the distal fragment was rotated till point A reached the margin of the dome.*



Figures 26 A-B: A*. The distal humerus was exposed through the posterior approach and the osteotomy was marked with a Bovie.* ***B****, Drill holes were made along the marked site of the dome with a 2.5-mm drill bit.*



Figures 27 A-B: *A. Lateral closing-wedge osteotomy results in two fragments of unequal widths and the lateral condyle becomes prominent after closing the osteotomy. B. In contrast, no such prominence occurs after dome osteotomy.*

Conclusion

Complications can occur depending on the type of flexion and the cartilage depending on the patient's condition and the location of the flexion. Diagnosis requires accurate and timely treatment for each complication that occurs. Among the complications, there are very common complications such as compartment syndrome, vascular/nerve damage... However, there are rare complications such as hypercalcemia causing bone immobility. There are complications that occur immediately after the fracture, but there are also common complications later.

Treatment issues need to be actively managed and coordinated between clinical and other diagnostic means such as imaging, laboratory diagnosis, etc. Patients with complications after fracture need urgent surgery, but must be meticulous and cautious. During the recovery process, it is necessary to combine physical therapy to regain the function of the limb.

It is necessary to educate the patient's parents about possible complications so that the child can cooperate with the doctor in treatment.

Conflict of Interest

The authors declare no conflict of interest.

References

1. ***Compartment Syndrome***

1. Volkmann R (1881) Die ischaemischen Muskellähmungen und Kontrakturen. Centralbl Chir 8:801–803 [DOI:10.1007/BF02514894]

2. Petersen F (1888) Ueber ischämische Muskellähmung. Arch Klin Chir 37:675–677

3. Hoffmeyer P, Cox JN, Fritschy D (1987) Ultrastructural modifications of muscle in three types of compartment syndrome. Int Orthop 11:53–59. [PMID: 3557756] [DOI: 10.1007/BF00266058]

4. Due J Jr, Nordstrand K (1987) A simple technique for subcutaneous fasciotomy. Acta Chir Scand 153:521–522. [PMID: 3425100]

5. Berman SS, Schilling JD, McIntyre KE, Hunter GC, Bernhard M (1994) Shoelace technique for delayed primary closure of fasciotomies. Am J Surg 167:435–436 ;[PMID: 8179090] [DOI: 10.1016/0002-9610(94)90130-9]

2. ***Vascular Injuries***

6. Mubarak, S. J., and Hargens, A. R.: Compartment Syndromes and Volkmann's Contracture. Philadelphia, W. B. Saunders: 24. 1991 [PMID: 6346542] [DOI: 10.1016/s0039-6109(16)43030-6]

7. Claffey TJ. Common fractures of the lower limb. Aust Fam Physician 1980 Mar;9(3):185-95. [DOI: 10.1007/bf01282711]

8. Mangat KS, Martin AG, Bache CE. The 'pulseless pink' hand after supracondylar fracture of the humerus in children: the predictive value of nerve palsy. J Bone Joint Surg Br. 2009 Nov;91(11):1521-5. [PMID: 19880900] [DOI: 10.1302/0301-620X.91B11.22486]

9. Herring, J. A.: Tachdjian's Pediatric Orthopedics, 3rd ed., p. 2148. 2020 [DOI:10.1016/s1529-9430(02)00191-2]

10. Campbell CC, Waters PM, Emans JB, Kasser JR, Millis MB. Neurovascular injury and displacement in type III supracondylar humerus fractures. J Pediatr Orthop. 1995 Jan-Feb;15(1):47-52.[DOI: 10.1097/01241398-199501000-00011]

11. Blakey CM, Biant lC, Birch R. Ischaemia and the pink, pulseless hand complicating supracondylar fractures of the humerus in childhood: long-term follow-up. J Bone Joint Surg [Br]2009;91-B(11):1487-1492. [PMID: 19880895] [DOI: 10.1302/0301-620X.91B11.22170]

12. Carbonell R, Moraleda I, Valencia M, Dier J. Long-term functional results of pink pulseless supracondylar fractures in children when vascular injury is managed conservatively. Podium presentation, EPOSNA Annual Meeting2017, Barcelona, Spain. [DOI:10.1302/2058-5241.3.170049]

13. Weller A, Garg S, Larson AN, Fletcher ND, Schiller JR, Kwon M, Copley LA, Browne R, Ho CA. Management of the pediatric pulseless supracondylar humeral fracture: is vascular exploration necessary? J Bone Joint Surg Am. 2013 Nov 06;95 (21):1906-12. [PMID: 24196459] [DOI: 10.2106/JBJS.L.01580]

3*. Neurological injury*

14. Mcgraw JJ, Akbarnia BA, Hanel DP et al (1986) Neurological complications resulting from supracondylar fractures of the humerus in children. J Pediatr Orthop 6(6):647–650 [DOI:10.1097/01241398-198611000-00001]

15.Valencia M, Moraleda l, díez-sebastián J.Long-term functional results of neurological complications of pediatric humeral supracondylar fractures. J Pediatr Orthop 2015;35(6):606-610. [PMID: 25379825] [DOI: 10.1097/BPO.0000000000000337]

16. Lyons JP, ashley E, Hoffer MM.Ulnar nerve palsies after percutaneous crosspinning of supracondylar fractures in children's elbows. J Pediatr Orthop1998;18(1):43-45. [DOI: 10.1097/00004694-199811000-00025.]

17. Brown IC, Zinar DM. Traumatic and iatrogenic neurological complications after supracondylar humerus fractures in children. J Ped Orthop 1995;15:440–3. [PMID: 7560030] [DOI: 10.1097/01241398-199507000-00005]

4. Fat Embolism Syndrome

18. Gurd AR, Wilson RI. The fat embolism syndrome. *J Bone Joint Surg Br.* 1974;56B (3):408–16. [DOI:10.1016/S0140-6736(72)91669-8]

19. Rahman SA, Valliani A, Chanda A. Fat embolism syndrome. Fat Embolism Syndrome. Intensive Care 2017.https://doi.org/10.5772/intechopen.69815. InTech. [DOI:10.5772/INTECHOPEN.69815.]

20. Dorr LD, Merkel C, Mellman MF, Klein I. Fat emboli in bilateral total knee arthroplasty. Predictive factors for neurologic manifestations. Clin Orthop Relat Res. 1989 ;(248):112-8 [DOI: 10.1097/00003086-198911000-00019]

21. Fukumoto LE, Fukumoto KD. Fat Embolism Syndrome. Nurs Clin North Am. 2018 Sep;53(3):335-347. [DOI: 10.1097/00003465-198305000-00005]

5. *Spontaneous Deep Vein Thrombosis*

22. Oudega R, Moons KGM, Hoes AW. Limited value of patient history and physical examination in diagnosing deep vein thrombosis in primary care. Family Practice. 2005;22(1):86–91. [PMID: 15640292] [DOI: 10.1093/fampra/cmh718]

23. Ikard RW, Ueland K, Folse R. Lower limb venous dynamics in pregnant women. Surg Gynecol Obstet. 1971;132 (3):483–488. [DOI: 10.1097/00006254-197107000-00003]

24. Hirsh J, Raschke R. Heparin and low-molecular-weight heparin the Seventh ACCP Conference on Antithrombotic and Thrombolytic Therapy. Chest. 2004;126(3 Suppl):188S–203S. [PMID: 15383472] [DOI: 10.1378/chest.126.3_suppl.188S]

25. Lee AY, Levine MN, Baker RI, et al. Low-molecular-weight heparin versus a coumarin for the prevention of recurrent venous thromboembolism in patients with cancer. N Engl J Med. 2003;349(2):146–153. [PMID: 12853587] [DOI: 10.1056/NEJMoa025313]

26. Patterson BO, Hinchliffe R, Loftus IM, Thompson MM, Holt PJ. Indications for catheter-directed thrombolysis in the management of acute proximal deep venous thrombosis. Arterioscler Thromb Vasc Biol. 2010;30(4):669–674. [PMID: 20237328] [DOI: 10.1161/ATVBAHA.109.200766]

27. Streiff MB. Vena caval filters: a comprehensive review. Blood. 2000;95(12):3669–3677. [https://doi.org/10.1182/blood.V95.12.3669]

6. *Infections After Fracture*

28. Lu D, Wang T, Chen H, Sun L (2017) Management of pin tract infection in pediatric supracondylar humerus fractures: a comparative study of three methods. European Journal of Pediatrics 176: 615-620. [PMID: 28251295] [DOI: 10.1007/s00431-017-2884-1]

29. Oetgen M, Mirick G, Atwater L, Lovejoy J (2015) Complications and Predictors of Need for Return to the Operating Room in the Treatment of Supracondylar Humerus Fractures in Children. The Open Orthopaedics Journal 9: 139-142. [DOI: 10.2174/1874325001509010139]

7. Pin tract infection

30. Green SA. Complications of external skeletal fixation.Clin Orthop. 1983;108:109-116. [DOI:10.1097/00003086-198311000-00015]

31 Herstik I, Pelletier JP, Kanat IO. Pin track infections: incidence and management in foot surgery.J Am Pediatr Med Assoc. 1990;80: 135-144. [DOI:10.7547/87507315-80-3-135]

32. Stahl S, Schwartz O. Complications of K-wire fixation of fractures and dislocations in the hand and wrist.Arch Orthop Trauma Surg. 2001; 121:527-530 [https://doi.org/10.1007/s004020100279]

8. ***Hypercalcemia of Immobilization***

33. Dodd K, Graubarth H, Rapoport S: Hypercalcemia nephropathy and encephalopathy following immobilization. Pediatrics 6:124-130, 1952. [DOI: 10.1542/peds.6.1.124]

34. Richet G, Ardaillore R, Amiel C, et al: Acidification des urines et augmentation de l'ammoniurie apres injection intraveineuse de gloconate de calcium chez l'homme. Rev Fr Etud Clin Biol 7:355-361,1962 [PMID: 14013634]

35. Cannon PJ, Heinemann H, Albert JS, et al: Contraction alkalosis following diuresis of edematous patients with ethacrynic acid. Fed Proc 23:306, 1964. [doi: 10.7326/0003-4819-62-5-979.]

36. Suki WN, Yium JJ, von Minden M, et al: Acute treatment of hypercalcemia with furosemide. N Engl J Med 283-836-840, 1970. [DOI: 10.1056/NEJM197010152831603]

37. Zeffren JL, Heinemann HO: Reversible defect in renal concentrating mechanism in patients with hypercalcemia. Am J Med 33:54-63, 1962. [https://doi.org/10.1016/0002-9343(62)90276-0]

9. ***Osteomyelitis***

38. Gutierrez K. Bone and joint infections in children. Pediatr Clin North Am 2005; 52: 779–94. doi: http://dx.doi.org/10.1016/j.pcl.2005.02.005

39. Waldvogel FA, Vasey H. Osteomyelitis: the past decade. N Engl J Med 1980; 303: 360–70. doi: http://dx.doi.org/10.1056/NEJM198008143030703

40. Hunter D. Osteomyelitis of the clavicle after Swan-Ganz catheterization. Arch Intern Med 1983; 143: 153–4. doi: http://dx.doi.org/10.1001/archinte.1983. 00350010163030

41. Hung NN. Cortical bone fenestrations with continuous antibiotic irrigation to mediate hematogenous tibial osteomyelitis in children. Journal of Pediatric Orthopaedics B (2010); 19:497–506. [PMID: 20634722] [DOI: 10.1097/BPB.0b013e32833cb8a2]

10. ***Delayed Union***

42. Miranda MA, Moon MS. Treatment strategy for nonunions and malunions. In: Stannard JP, Schmidt AH, Kregor PJ, editors. Surgical Treatment of Orthopaedic Trauma, vol. 1. New York, NY: Thieme; 2007. p. 77–100. [DOI:10.1053/j.oto.2008.09.001]

43. Rajasekaran S, Giannoudis PV. Open injuries of the lower extremity: issues and unknown frontiers. Injury 2012;43(11):1783–4. [DOI: 10.1016/j.injury.2012.08.039]

44. Bishop JA, Palanca AA, Bellino MJ, Lowenberg DW. Assessment of compromised fracture healing. J Am Acad Orthop Surg 2012;20:273–82 [PMID: 22553099] [DOI: 10.5435/JAAOS-20-05-273] Top of Form

11. ***Non Union***

45. Kenwright J, Richardson JB, Cunningham JL, White SH, Goodship AE, Adams MA, et aL Axial movement and tibia [fractures. A controlled randomised trial of treatment. J Bone Joint Surg [Br] 1991 ;73B:654-659. [DOI: 10.1302/0301-620x.73b4.2071654]

46. Judet R, Judet J Roy-Camille R. La vascularisation des psedarthroses des os longs d'ars unatude c[inique et exp6rimentale. Rev Chir Orthop 1958;44:381.

47. Muller ME. Treatment of nonunion by compression. Ctin Orthop 1966;43:83 91.

48. Hutth A. Current concepts in fracture heatin 8. Clin Orthop 1989;249:265-84, [DOI:10.1097/00003086-198912000-00028]

49. Weber BG, Cech O. Pseudarthrosis. Bern: Hans Huber Publishers; 1976. [**DOI:** 10.12691/js-2-1-2]

12. ***Re-displacement***

50. Hung NN. Chapter 2 . Basic Knowledge of bone Grafting. Edited by Alessandro and João Bone Graging , In Tech: 11-39. 2011 . [DOI:10.5772/30442]

51. Oetgen M, Mirick G, Atwater L, Lovejoy J (2015) Complications and Predictors of Need for Return to the Operating Room in the Treatment of Supracondylar Humerus Fractures in Children. The Open Orthopaedics Journal 9: 139-142. [PMCID: PMC4484238] [DOI: 10.2174/1874325001509010139]

52. Friberg KS. Remodelling after distal forearm fractures in children. III. Correction of residual angulation in fractures of the radius.Acta Orthop Scand1979;50 (6 Pt 2):741–749. [PMID: 534550] [DOI: 10.3109/17453677908991304]

53, Bae DS. Paediatric distal radius and forearm fractures.J Hand Surg Am 2008;33:1911–1923. [PMID: 19084202] [DOI: 10.1016/j.jhsa.2008.10.013]

54. Lee BS, Esterhai JL Jr, Das M. Fracture of the distal radial epiphysis. Characteristics and surgical treatment of premature, post-traumatic epiphyseal closure.Clin Orthop Relat Res1984;185:90–96. [DOI:10.1097/00003086-198405000-00016]

55. Mani GV, Hui PW, Cheng JCY, Bone J. Translation of the radius as a predictor of outcome in distal radial fractures of children.Joint Surg Br 1993;75:808–811. [DOI: 10.1302/0301-620X.75B5.8376446]

56, Proctor MT, Moore DJ, Paterson JMH. Redisplacement after manipulation of distal radial fractures in children.J Bone Joint Surg1993;75-B:453–454. [PMID: 8496221] [DOI: 10.1302/0301-620X.75B3.8496221]

57. McLauchlan GJ, Cowan B, Annan IH, Robb JE. Management of completely displaced metaphyseal fractures of the distal radius in children: a prospective, randomized controlled trial. J Bone Joint Surg2002;84B:413–417. [PMID: 12002503] [DOI: 10.1302/0301-620x.84b3.11432]

58. Abid A, Accadbled F, Kany J, De Gauzy JS, Darodes P, Cahuzac JP. Ulnar styloid fractures in children: a retrospective study of 46 cases.J Pediatr Orthop B2008;17:15–19. [DOI:10.1097/BPB.0b013e3282f3cacb]

13. ***Cubitus varus***

59. De gheldere a, Bellan d.Outcome of Gartland type II and type III supracondylar fractures treated by Blount's technique. Indian J Orthop2010;44(1):89-94. [DOI: 10.4103/0019-5413.58612]

60. Mousavi SJ, Parnianpour M, Abedi M, Askary-Ashtiani A, Karimi A, Khorsandi A, Mehdian H. Cultural adaptation and validation of the Persian version of the Disabilities of the Arm, Shoulder and Hand (DASH) outcome measure.Clin Rehabil. 2008;22:749–757 [PMID: 18678575] [DOI: 10.1177/0269215508085821]

61. O'driscoll sW, spinner rJ, McKee Md, et al.Tardy posterolateral rotatory instability of the elbow due to cubitus varus. J Bone Joint Surg [Am]2001;83-A(9):1358-1369. [PMID: 11568199] [DOI: 10.2106/00004623-200109000-00011]

62. DeRosa GP, Graziano GP. A new osteotomy for cubitus varus. Clin Orthop Relat Res.1988;236:160–165. [DOI 10.1007/s11999-012-2756-y]

63. Kim HT, Lee JS, Yoo CI. Management of cubitus varus and valgus.J Bone Joint Surg Am.2005;87:771–780 [PMID: 15805206] [DOI: 10.2106/JBJS.D.01870]

64. Tien YC, Chih HW, Lin GT, et al. Dome corrective osteotomy for cubitus deformity.Clin Orthop. 2000;380:158–166 [PMID: 11064986] [DOI: 10.1097/00003086-200011000-00021]

Citation: Hung NN, Duc HH, Anh LT, Sang PC, Hoang NV. "Management and Complications After Paediaric Fracture" *SVOA Orthopaedics 2022,* 2:6, 139-165.