

Late onset brachial artery thrombosis and total temporary peripheral neuropathy in a child with humerus supracondylar fracture: a case report

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ABSTRACT

Pediatric supracondylar fractures of the humerus are generally associated with neurovascular complications due to the deformity and sharp nature of bone fragments. When treated inadequately, these injuries may result in catastrophic complications, such as Volkmann's contracture and amputation. To our knowledge, late onset brachial arterial thrombosis and total temporary peripheral neuropathy after surgery of pediatric supracondylar fracture in the setting of normal preoperative vascular examination has not been reported yet. In this study, a 2-year and 6-month-old girl, who had delayed brachial arterial thrombosis after a displaced humerus supracondylar fracture surgery treated with embolectomy, was reported. Total lesion of median, ulnar and radial nerves completely resolved four months after surgery. Close neurovascular monitoring on the postoperative phase especially in severely displaced supracondylar fractures is strongly emphasized even in the setting of well-perfused hand.

Key words: Brachial artery; supracondylar fracture; thrombosis.

INTRODUCTION

Vascular complications associated with pediatric humerus supracondylar fractures are still common problems despite much has been written about the incidence and their management.^[1] When managed inappropriately, these injuries may result in catastrophic complications, such as Volkmann's contracture and amputations which are extremely rare situations an orthopedic surgeon may face once in his entire career. Vascular pathologies such as thrombosis, intimal tear, and rupture associated with bone fractures generally give symptoms in the acute phase of the fracture. However, late on-

set arterial occlusion after bony fractures are extremely rare situations.^[2-4] To our knowledge, there is no reported data in the literature consisting late onset vascular occlusion after surgical treatment of supracondylar fracture with preoperative and postoperative good palpable radial pulse.

CASE REPORT

A 2-year and 6-month old girl was admitted to our emergency department from another tertiary trauma center with severely displaced Gartland type-III supracondylar fracture. The fracture was due to a fall from 1.5 meter-high kitchen bench. The patient was consulted by an orthopedic surgeon at 3' o'clock in the morning and her parents stated that she ate just before being admitted to our emergency department (ED). On physical exam, there was large ecchymosis on the ante-cubital fossa with brachialis sign. Radial pulse was palpable and on motor neurologic exam, median nerve was intact; however, ulnar, radial, and anterior interosseous nerves were not evaluated optimally. On sensory exam, ulnar radial and median nerve distributions were intact. With palpation,

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forearm structures were soft and there was no pain with passive stretch of forearm muscles. On direct radiographs, postero-laterally displaced Gartlant type-III humerus supracondylar fracture was detected and proximal fragment ruptured the brachialis muscle (Fig. 1a, b). After consulting with the anesthesiology and reanimation department, patient was planned for surgery as first case in the morning. In the ED, gentle traction to the forearm was performed and a long arm splint in slight extension was made. Radial pulse was closely monitored even with palpation and pulse-oxymetry. At 7 o'clock, the patient was planned for surgery. Under general anesthesia, closed reduction and pin fixation were attempted first. However, the fracture was not reduced and surgical decision shifted to open reduction. Without a tourniquet, open reduction was performed through posterior incision exposing ulnar nerve. With direct visualization, fracture was reduced anatomically and fixed with 3 K-wires (Fig. 1c, d). After closing the wound, radial pulse was palpable at all el-

bow range of motions. After surgery, a long arm splint with 30-degree flexion was made and motor neurologic examination could not be performed after anesthesia. On sensory examination, the patient responded to all painful stimulus for median ulnar and radial nerves. At the twenty-fourth h after surgery, the patient could not still be evaluated for motor neurologic exam; however, sensory examination was normal. All bandages were opened and forearm compartments were checked; all compartments were soft and there was no pain with passive stretching. At the thirtieth hour after surgery, patient's hand became cold and radial pulse was not palpable; however, capillary refill was normal. All forearm compartments were soft and there was no pain with passive stretching and palpation. A Doppler USG was performed and revealed no flow distal to the brachial artery. Thereafter, emergent brachial arterial exploration was performed by the Cardiovascular Surgery Department. Radial pulse immediately returned following the removal of fresh embolus

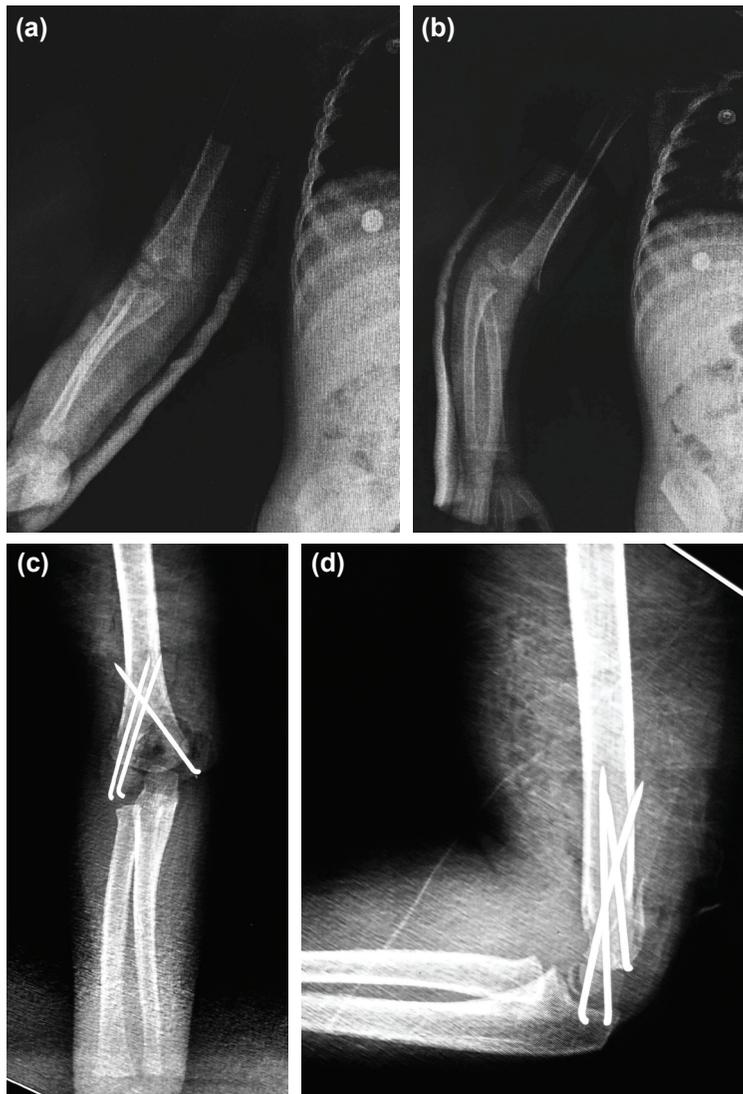


Figure 1. (a, b) Preoperative plain radiographs of the patient. Note that proximal fragment ruptured brachialis muscle. (c, d) Plain radiographs after open reduction of the fracture.

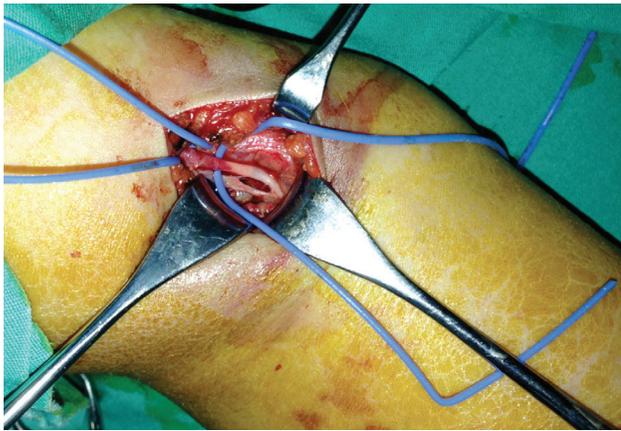


Figure 2. Intraoperative view of the thrombosed brachial artery.

with Fogarty catheter from the thrombosed segment (Fig. 2). After surgery, there was slight edema on the forearm region but compartments were soft. EMG study, performed on the third week after surgery, revealed total axonal degeneration on median, radial and ulnar nerves. Afterwards, aggressive physical therapy was started. On the thirtieth day after surgery, there was slight contracture on flexor muscles of second, third, fourth, and fifth digits. Static splint for the fingers and wrist was ordered. At the fifth week after the initial trauma, median and ulnar nerves began to heal. EMG study in the third month showed complete healing of radial nerve and partial axonal degeneration of medial and ulnar nerves. In the fourth month after surgery, all nerves healed without sensory deficit and motor loss and all of the contractures resolved completely. Patient revealed slight elbow flexion deficit in last 20 degrees.

DISCUSSION

Careful neurovascular examination must be the first step in the evaluation of a child with supracondylar fracture. However, frightened child may cause a delay in detecting vascular pathologies in preoperative and even in postoperative phases. Vascular pathologies associated with supracondylar fractures may be classified into three types including first type, acute vascular occlusion; second type, sub-acute occlusion; and third type, Volkmann's ischemia.^[5] Fortunately, acute ischemic events are extremely low as the collateral circulation around elbow joint is good enough even in the case of brachial artery rupture. Most authors indicate that non-palpable radial pulse with warm hand is not an indication for routine arterial exploration as most of the cases return to normal after reduction and pinning of the fracture.^[5,6] Absence of the pulse with palpation and by Doppler USG is important, meaning an arterial injury is probably present. At this situation, routine brachial arterial exploration must be considered.^[7]

The other but rare cause of ischemia after supracondylar fracture is compartment syndrome which may result in Volkmann's contracture and amputation when treated inad-

equately. Historically, it was generally due to casting after closed reduction and flexion of the elbow greater than 90 degrees. However, the incidence of compartment syndrome was diminished by routine closed reduction and percutaneous pinning of the displaced supracondylar fractures. It generally has 5P (pain, pallor, pulselessness, paresthesias, and paralysis) signs but the most obvious sign is pain out of proportion and pain with passive stretching of the wrist and fingers. However, pain can be masked in patients with median nerve lesion, which called silent compartment syndrome. Thus, in this group of patients, compartment pressures must be measured as soon as possible and early fasciotomies must be considered.

When the literature is reviewed, late onset vascular lesions associated with bony fractures are extremely rare and consist several cases.^[2-4] The main reason in these cases is intimal tear after trauma of the artery by fractured bone fragments.^[2,3] Copley et al.^[1] have reported two cases with late onset brachial arterial occlusion after surgical treatment of displaced supracondylar fracture in their study. However, in these cases, radial pulses were diminished or absent before surgery. It is to our belief that our case was unique as the patient had good pulses before and after the reduction of the fracture. Thrombosis at the fracture site was thought to be due to intimal tear caused by sharp proximal fracture fragment. Therefore, thrombosis at the intimal tear site aroused gradually at postoperative phase. In our case, compartment syndrome, which may mask acute arterial occlusion, was not considered since all forearm compartments were checked with physical exam throughout the hospital stay. Furthermore, patient's sensation over median nerve distributions were normal as we ruled out silent compartment syndrome. Again, immediate return of pulses after embolectomy ruled out probable compartment syndrome. Additionally, our case had total neurologic deficit involving median, ulnar and radial nerves, which was extremely rare in this type of fracture, and completely resolved in the fourth month after surgery. It was probably due to traction injury resulting from severe displacement of the fractured bone fragments after the high fall and transient ischemic situation due to brachial arterial occlusion.

We strongly emphasize that patients with severely displaced supracondylar fractures should be carefully monitored for neurovascular status even at the postoperative phase. One should consider that acute compartment syndrome might interfere with acute arterial conclusion, and therefore, in case of suspicion, compartment pressures must be checked even with physical exam or invasive techniques. Good capillary refill does not exclude arterial occlusion or compartment syndrome. A routine follow-up for at least forty-eight hours after surgery must be preferred with severely displaced supracondylar fractures.

Conflict of interest: None declared.

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OLGU SUNUMU - ÖZET

Pediyatrik humerus suprakondiler kırıklı bir hastada ge gelişen brakiyal arter trombozu ve total geici sinir paralizisi: Olgu sunumu

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ocukluk ađı humerus suprakondiler kırıkları hem kırıktan kaynaklanan deformite hem de kırık ularının keskin yapısı nedeni sıklıkla nörovaskler komplikasyonlara yol amaktadır. Yeterli tedavi edilmedikleri takdirde Volkmann iskemik kontraktr veya amputasyon gibi katastrofik komplikasyonlara yol aabilmektedirler. Bilgimize gre literatrde ameliyat ncesi dnemde normal nörovaskler muayenesi olup ameliyat sonrası ge dnemde brakiyal arter trombozu ile birlikte total sinir paralizisi henz bildirilmemiştir. alıřmamızda iki yař altı aylık olan ocuk hastada kaymıř humerus suprakondiler kırığının ameliyat sonrası dneminde gelişen brakiyal arter trombozunun embolektomi yapılarak tedavisini sunmayı amaladık. Total median, ulnar ve radial sinir lezyonları, ameliyat sonrası drdnc ayda tamamen dzeldi. Sonu olarak, zellikle ciddi olarak kaymıř pediyatrik humerus suprakondiler kırıklarında ameliyat ncesi dnemde elin dolařımı iyi olsa dahi, ameliyat sonrası dnemde nörovaskler inceleme yakinen yapılmalıdır.

Anahtar szckler: Brakiyal arter trombozu; ge dnem; humerus; kırık; suprakondiler; total paralizi.

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