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Case report

Compartment syndrome in a neonate

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ABSTRACT A neonate born with severe asphyxia and considerable risk of infection was treated with intravenous sodium bicarbonate and intravenous antibiotics. At the age of five days he developed edema of the right forearm, tense on palpation, painful, hyperemic, and the hand was pallor, hypesthetic with maceration of the fifth finger. The diagnosis of compartment syndrome was established but it was delayed at least for about 48 hours. Decompression by fasciotomy using the volar Henry approach was performed; after which the pathologic changes rapidly subsided. The cause of this syndrome was probably due to intravenous sodium bicarbonate and repeated intravenous antibiotics. From this experience it is strongly emphasized that we have to be aware and able to diagnose compartment syndrome at the earliest possible time in severely ill neonates and children who have multiple intravenous injection. Daily inspection and careful evaluation of the condition at the site of the intravenous line will be very crucial to detect the syndrome. **[Paediatr Indones 2001; 41:253-255]**

***Keywords**: Compartment syndrome, management*

COMPARTMENT SYNDROME IS A SIGNIFICANT CLINICAL problem causing major functional losses after a wide variety of traumatic, vascular, hematologic, neurologic, surgical, pharmacologic, renal, or iatrogenic conditions. Compartment syndrome is defined as a symptom complex caused by elevated pressure of tissue fluid in a closed osteofacial compartment of the limb that interferes with the circulation to the muscles and nerves of that compartment. The incidence of compartment syndrome in adult has been reported as 6% in open fractures of the tibia and 1.2% in closed tibial fractures, 30% with arterial injuries and 14% with significant venous injuries.[2,3] There is little documentation as to the true incidence of compartment syndromes in the pediatric age group especially in the neonate.

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Mubarak and Carrol[3] reported 55 cases of Volkmann's ischemic contracture admitted to the Hospital for Sick Children in Toronto over a 21-year period. Quellette[4] in her report of compartment syndrome of the hand between April 1985 and January 1990 had found 19 cases of which 9 cases were children between 5 months and 17 years old, the youngest being 5 months old, none in neonate. The diagnosis of compartment syndrome often is difficult to make, and a delay in diagnosis is a common theme in reviewing the literature on the subject. The purpose of this report is to remind us that compartment syndrome can occur in a neonate after a medical condition and that this condition is a surgical emergency case that should be handled as soon as possible to prevent permanent severe damage to the limb. This is the first compartment syndrome in a neonate in our hospital.

Report of the case

A baby boy was born on October 11, 1999 in Soetomo Hospital, Surabaya. He was delivered by vacuum

extraction because of prolonged 2nd stage labor, at term with a body weight of 3700 grams, with severe asphyxia (Apgar scores of 3 in the first minute and 3 in the fifth minute). The mother, 24 years old, had fever because of urinary tract infection, the membrane ruptured 10 hours before delivery, and the amniotic fluid was meconium stained. The baby cried weakly, dyspneic, with flaring of the nostrils and was cyanotic. The heart rate was 132 beats/min, regular. Respiratory rate 60 breaths/min, regular, temperature 38.5°C. On examination we found caput succedaneum, intercostal an subcostal retractions, cyanotic extremities which were cold on palpation. No other abnormalities were found. The boy was assessed as a term baby with severe asphyxia and risk of infection.

He was then treated with oxygen and fluid therapy containing dextrose 10% and sodium bicarbonate. Intravenous ampicillin and netilmicin were instituted. The IV injections were given on the back of the right hand. Following such treatment the baby recovered gradually. However, on the 5th day the baby looked weak with slight jaundice without dyspnea or cyanosis. The heart rate was 132 beats/min, respiratory rate 48 breaths/min, temperature 37°C. On the back of the right hand a black spot was noted and the right forearm was swollen until the elbow. It was assessed as infusion infiltrate with necrosis and phlebitis. Laboratory examination on the 6th day, revealed CRP 24 mg/L, Hb 10.5g /dL, leukocyte $6.2x10^9$/L, platelet $21x10^9$/L, and PCV 40%. Direct bilirubin level was 2.6 mg/dL, indirect bilirubin 1 mg/dL, total bilirubin 3.6 mg/dL, SGOT 5U/L, SGPT 7 U/L.

On the 7th day his condition remained the same but the black spot spread upward to the forearm. The right forearm was hyperemic, edematous, and firm on palpation. The hand was pallor, hypesthetic and painful when stretched, the fifth finger until the distal metacarpal area was bluish and macerated. The first until the fourth fingers were red and viable, the fingers could not move. It was difficult to palpate the arteries. Black bullae were found on the forearm and hand. He was then assessed as having the compartment syndrome with probable sepsis.

Decompression by fasciotomy was done using the Volar-Henry approach. Ceftriaxone and cloxacillin were given intravenously and whole blood was transfused. Ampicillin and netilmicin were discontinued. Saline compress was applied to the fasciotomy wound. The baby's condition gradually improved. On the 11th day, he looked well, cried louder, could take bottle feeding and the fifth finger became dark red, the other fingers remained red with positive capillary filling, the fingers could move actively. Blood culture taken on the 8th day yielded *Klebsiella sp*. Since then he recovered smoothly and on the 17th day the fasciotomy wound was closed and necrotomy was done on the fifth finger. He was discharged on the 20th day.

On discharge, there was no edema, all the fingers could move actively, looked red with positive capillary filling. On the fifth finger there was minimal necrotic part. On follow up at the age of 2 months, the boy was healthy, body weight 4.600 grams, scar of the fasciotomy was present on the right fore arm. The hand and fingers could move actively. No contracture was found.

Discussion

The diagnosis of compartment syndrome should be based on clinical signs and measurement of the intracompartmental pressure. The clinical findings in compartment syndrome are as follows: Pain with stretching of the toes or fingers is the earliest finding in compartment syndrome. Firm fullness of increased pressure is palpable in the involved muscle compartment, such as the anterior compartment of the leg or forearm. A pulse is present distally (This is in contrast with ischemia from direct arterial injury). Paresthesia presents distally as a result of ischemia of nerve. Paresis result from more advanced nerve ischemia. In many patients, pinkness persist in the tips of the extremity despite the muscle ischemia proximally, it can also be pale/pallor.[1,2,4-10]

In most cases, the diagnosis can be made from these clinical findings. If there is any doubt about the diagnosis, the intracompartmental pressure can be measured directly. The differential diagnosis of acute compartment syndrome includes arterial injury, nerve injury, osteomyelitis, tenosynovitis, cellulitis, and thrombophlebitis.[8] In retrospect our diagnosis on the 5th day of necrosis and phlebitis appeared to be wrong. The clinical presentation of compartment syndrome is often indefinite and confusing and delays in diagnosis occur even when physicians are aware of the signs and symptoms.[1,2,4-8]

In our case, the diagnosis of compartment syndrome was delayed at least for about 48 hours, partly

because at that time we did not recognize the signs and symptoms of compartment syndrome properly and daily inspection of the infusion site was not done properly. In Quellette series[4] of compartment syndrome of the hand 14 of 19 patients was iatrogenic; specifically it developed because of complication related to intravenous administration of drugs, the use of an arterial line, or an arthrodesis of the wrist. The etiology of compartment syndrome in our case was probably due to the administration of intravenous sodium bicarbonate, ampicillin, and netilmicin.

In our patient, intracompartmental pressure was not measured because we did not have the apparatus. However, the clinical findings were already conclusive for the compartment syndrome.

Once established, compartment syndrome must be treated by appropriate fasciotomy. There is no role for conservative management in established cases.[2] The quality of the functional result after fasciotomy is directly related to the promptness with which the decompression is performed. The frequency and severity of complications are inversely related to the promptness of decompression. Thus, delays in diagnosis or indecision about decompression when the diagnosis of a compartment syndrome is made may be costly.[7]

In our patients, the fasciotomy was done using the volar Henry approach and the result was dramatically good. Although at the time of diagnosis the possibility of amputation of the fifth finger was considered, the course of the disease was very promising. After three days of fasciotomy the fingers could move actively, capillary filling was present, and the fifth finger started to become red.

Factors affecting outcome after fasciotomy include underlying atherosclerotic disease, anemia, and hypovolemia. Infection is the principal complication of fasciotomy and may result in amputation and death.[8] The prognosis of our case was good except for the scars.

We suggest that the physician in charge should be careful for the occurrence of compartment syndrome when managing critically ill, obtunded patients—particularly children and neonates—for whom multiple intravenous or intra-arterial injections are necessary. Frequent inspection at the site of intravenous injection or line is very important.

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