Interdepartmental Conference

# An Infant with Minor Blunt Trauma with Shock

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**Abstract : An infant who had a history of minor blunt trauma and later developed shock was reported. He was initially presented at the hospital because his mother noticed an abrasion on his nose that wouldn't stop bleeding for more than ten hours. The day before, his 3-year-old brother, imitating a wrestler, hit him in his abdomen and accidentally scratched his face, which brought him to the trauma emergency department the following day. The baby appeared active at first but looked pale; within 2 hours he became hypotensive and drowsy. His blood test had a prolonged bleeding time with very low fibrinogen and his blood concentration dropped 5%. The computed topographic scan located a chronic and subacute subdural hematoma without intraabdominal abnormalities. There had been previous reports of isolated head injuries which caused hypotension but our patient had a coincident coagulopathy also. The management and etiology of the patient were discussed. We concluded that in a traumatized child with shock the major systems should be evaluated together because an isolated head injury can cause hypotension and to emphasize the abdomen as the leading cause would only delay the management.**

***Key words*** : Hypofibrinogenemia, Hypotension, Isolate head injury, Child

**เรื่องย่อ** : **เด็กทารกมีอาการช็อกจากลหุอุบัติเหตุ**

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รายงานผู้ป่วยเด็กชายอายุ 1 เดือน มาโรงพยาบาลเนื่องจากมีแผลถลอกบริเวณจมูกและเลือดออกไม่หยุด มารดาให้ประวัติว่าพี่ชายอายุ 3 ปี เล่นมวยปล้ำกับน้องเมื่อ 10 ชั่วโมงก่อน สองชั่วโมงต่อมาขณะอยู่ที่ห้องฉุกเฉิน เด็กมีอาการซึมลง ความดันโลหิตตก และชีด ตรวจหาจุดเลือดตกใน โดยการตรวจเอ็กซเรย์ปอด อุลตร้าซาวน์ของช่องท้อง ไม่พบเลือดออกที่เป็นสาเหตุของความดันโลหิตตก ผลการตรวจเลือดพบมีภาวะการแข็งตัวของเลือดผิดปกติ สงสัยภาวะขาดไฟบริโนเจนแต่กำเนิด เด็กได้รับการตรวจเอ็กซเรย์คอมพิวเตอร์ตลอดร่างกาย พบมีก้อนเลือดออกในสมองแบบกึ่งเฉียบพลัน และมีการตกเลือดบริเวณชายปอดทั้งสองข้าง ส่วนในช่องท้องและกระดูกไม่พบความผิดปกติ

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## INTRODUCTION

Trauma is an important condition in Thailand and worldwide, especially in the child population. It is the leading cause of death in children and young adults[1,2]. The motor vehicle is the cause of most deaths in childhood, whether the child is an occupant, a pedestrian or a cyclist. Children with multisystem injuries can deteriorate rapidly and develop serious complications. The priorities of assessment and management of the injured child are the same as in an adult. However, the unique anatomic characteristics of children require special consideration in assessment and management.

## CASE REPORT

A 1-month-old boy was brought to the trauma unit by his mother because of a non-stop bleeding abrasion wound on his nose. His mother informed us that the day before his 3-year-old brother played with him as a wrestler and hit him in the abdomen and scratched his nose. There was a shallow 3-mm. abrasion wound on his nose with persistent bleeding. The mother tried many ways to stop this bleeding, including compression and cold packing, but the bleeding did not stop for ten hours so she brought the infant to the trauma unit. At home the infant threw up twice after feeding but was still active. This infant is the second son of an 18-year-old mother, with full ANC and normal labor. His birth weight was 3,180 grams. Shortly after his birth, he developed jaundice; it was found that he had a G-6-PD deficiency. After 3 days of admission, the jaundice improved and he was discharged from the hospital with 43% hematocrit.

The first time in the hospital the infant was alert. His vital signs were stable with a respiratory rate of 58/min, heart rate of 180/min, and blood pressure 100/63 torr (normal vital signs of this age: respiratory rate of 60/min, heart rate of 160-180/min, and systolic blood pressure 60-80 torr). He was mildly pale. There was a 3-mm abrasion wound on his left nostril with blood oozing. The anterior fontanel was 2.5 x 2.5 cm and slightly tense, while the posterior fontanel was closed. The respiration was equal on both sides. The abdomen was not distended. He could move all extremities. His initial hematocrit, drawn from the heel, was 26%.

About two hours later, the infant became drowsy and markedly pale. He subsequently became hypotensive with blood pressure of 50/30 torr. The infant was intubated and an intravenous line accessed. At first, venesection failed; then he had fluid resuscitation by intraosseous cannulation.

His repeated blood test revealed a hematocrit of 17% with normal platelet. There was coagulopathy with prolonged PT and PTT (PT > 120 sec, PTT > 150 sec). Then a pediatric hematologist was consulted and the infant's fibrinogen level was measured, which was too low to be evaluated. After successful venesection and adequate fluid resuscitation and blood transfusion, the infant's blood pressure was stabilized.

The infant was re-evaluated; he had a history of minor blunt injuries with coagulopathy and shock. His conscious condition changed and hematocrit dropped more than 3%. Initially an intraabdominal solid organ injury was suspected to be the cause of the blood loss but his abdomen was palpated and soft and the bowel was active. There was no dullness on percussion and his liver was smooth on palpation. The infant's pupils were both sluggishly reactive to light, which might be due to hypotension. However, his anterior fontanel was quite tense, so an intracranial lesion could not be ruled out. A bedside ultrasonography was performed and no free fluid was identified, and the liver and spleen appeared normal. The consulting pediatric hematologist suspected congenital afibrinogenemia to be the cause of the very low fibrinogen level and prescribed 135 ml of fresh frozen plasma. After the infant was stabilized, he was moved to undergo in imaging study. Initial chest x-rays showed increased interstitial infiltration in both lungs. The whole body topographic scan revealed chronic and subacute subdural hematoma and bilateral pulmonary hemorrhage without intraabdominal abnormalities. Then a neurosurgeon was consulted and the infant was transferred to the operating theater for surgery. In conclusion, this child had chronic blunt injuries and bled in both lungs, intracranium and from his lacerated wound. All the bleeding points added up to an estimated blood loss of 80 ml which was the cause of shock.

## DISCUSSION

**The approach to pediatric trauma patients.**

Children are different from adults in many ways. They are smaller in size, their vital organs are closer to the surface, and the skeleton has less calcium and increased pliability, making them less likely to break. Forces may transmit through the skeleton and injure vital structures without breaking them. Psychological effects should be considered because it may cause lifelong physical and emotional harm.

The initial approaches to injured children are not different from those to adults. A primary survey should be done for immediate life threatening conditions. The classic ABCDEs should be evaluated, but normal vital signs of children differ by age. There are many important factors in taking care of children such as their small size, so they can be injured in several organs at the same time. They have less blood than adults, about 80 ml/kg, which should be only 400 ml blood in this 5-kg infant. Blood loss of just 100 ml can cause hypotension in this case. Children more easily develop hypothermia due to immature body-heat regulation and their large body surface. And the last thing that one must be aware of is if an injured child is the victim of possible child abuse. A history of suspected child abuse includes a discrepancy between the history and degree of physical injury, prolonged time of arrival, history of repeated trauma and parents responding inappropriately. The nature of injuries themselves are also clues for a battered child such as multiple subdural hematoma, retinal hemorrhage, perioral injury, trauma to the genital or perianal regions, evidence of repeated injuries, fractures of long bones in children less than 3 years old and bizarre injuries such as bites and cigarette burns or rope marks. Suspected cases should be admitted and involved authorities should be contacted.

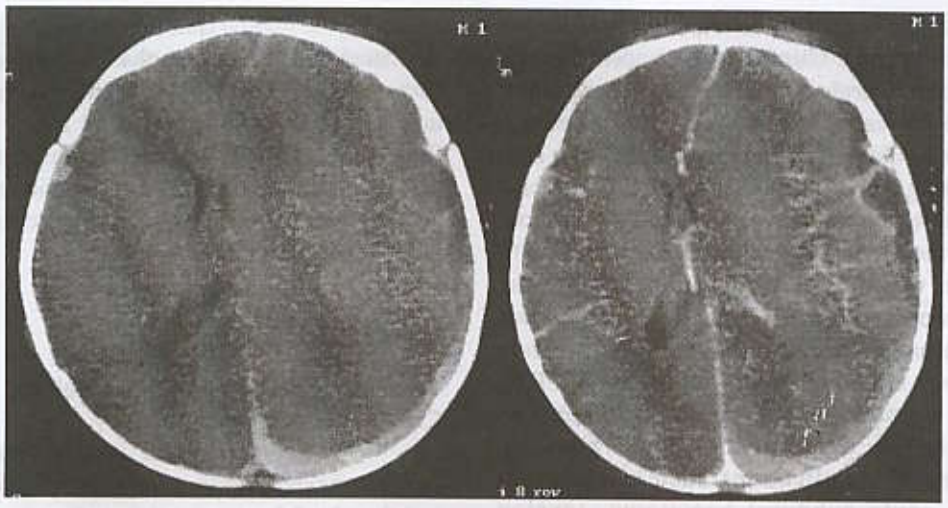
Normally in a hypotensive child with acute anemia hemorrhage should be the suspected cause. Bleeding from a blunt injury is commonly found in the thorax, abdomen and long bone fracture. We investigated the thorax by a physical examination and chest x-ray but found nothing abnormal (although subsequent CT showed bilateral lung hemorrhage). The intraabdominal organ was examined; ultrasonography and CT of the abdomen were performed but showed no bleeding. But in small children there have been reports that an isolated head injury could be the cause of hypotension. Even though this patient did not have much blood in the cranium, this may suggest that hypotension is not always an indicator of blood loss from traumatic injures in children, especially in very small children.

**Approach to a child with bleeding and prolongation of both prothrombin time (PT) and activated partial thormboplastin time (aPTT).**

Prolongation of both the PT and aPTT in the newborn period can be both congenital and acquired etiologies. This indicates the deficiency or inhibitor of one or more clotting factors. Deficiency of a single factor in the common pathway (factor V, X, prothrombin and fibrinogen) or an abnormal function of fibrinogen or dysfibrinogenemia can prolong the coagulation time. However, these conditions are rare, so deficiency of multiple factors from both the intrinsic and extrinsic pathways may have a single etiology and also prolong both the PT and aPTT.

Vitamin K deficiency is the most common cause of these findings. The absence of vitamin K results in the defective production of the vitamin K-dependent factors (factor II, VII, IX, X). Bleeding due to vitamin K deficiency can be severe, with a significant incidence of gastrointestinal, deep tissue and intracranial bleeding. Vitamin K deficiency can be classified into 3 categories: early hemorrhagic disease of the newborn (HDN), classic HDN and late HDN or acquired prothrombin complex deficiency (APCD)[1]. The bleeding onset in our patient occurred at 1 month of age, so he should be classified as late HDN or APCD. APCD typically occurs after the first week of life and extends into the first few months of life. This form is associated with a number of processes that interfere with vitamin K stores, including inadequate intake in breast feeding and inadequate absorption in infants with chronic diarrhea and other gastrointestinal abnormalities. All forms of vitamin K deficiency are best prevented by the use of prophylactic vitamin K oral or intramuscular injections from birth.[2-3]

Another cause of abnormal vitamin K metabolism is the ingestion of warfarin. Wafarin is a vitamin K antagonist. Accidental or intentional ingestion of wafarin may result in a picture identical to that seen in vitamin K deficiency. Our patient was delivered at Siriraj Hospital and received vitamin K prophylaxis since birth with no history of ingestion of any drugs, so these conditions could be ruled out in our patient. However, vitamin K administration in



critical bleeding patients is not harmful, so he received vitamin K initially. Although vitamin K bleeding responds rapidly to the administration of vitamin K, this process will take 4-6 hours to normalize coagulation time. When there is significant bleeding, fresh frozen plasma (FFP) may be required.

Disseminated intravascular coagulation (DIC) is another condition that should be considered in our patient. DIC is a consumptive coagulopthy secondary to a variety of disorders including sepsis, trauma and malignancy.[4] Although the diagnosis of DIC is based on clinical data, laboratory testing can be used to support the diagnosis of DIC. The typical laboratory abnormalities include decreased fibrinogen concentration, decreased factor V and VIII activity and reduced level of antithrombin III. Fibrinogen degradation production and D-dimers are usually elevated.

Laboratory investigation in our patient showed a prolongation of PT, aPTT and thrombin time (TT). A very low level of fibrinogen, elevated D-dimers and coagulation time after the mixing test returned to normal; this indicates that the prolongation of the coagulation time was due to the deficiency of coagulation factors and not from coagulation inhibitors. From clinical and laboratory data, the differential diagnosis in our patient included afibrinogenemia or dysfibrinogenemia and DIC.

This child was treated with normal saline, packed red cell 10 ml/Kg and FFP 30 ml/kg as fluid replacements. FFP 30 ml/kg can raise coagulation activity around 30% and the hemostatic level of coagulation activity is 30-40% but the coagulation time was still prolonged after replacement therapy because FFP is not a good source of fibrinogen and our patient had a very low level of fibrinogen initially. Cryoprecipitate (CPP) is rich in fibrinogen[5] (0.2 unit of CPP can raise the level of fibrinogen 80-100 mg/dl) and the hemostatic level of fibrinogen is 150-200 mg/dl. Therefore, after transfusing 0.4 unit/kg of CPP to our patient, the PT and aPTT returned to normal and the patient could be moved to surgery.

After surgery, we followed up the PT, aPTT and fibrinogen levels closely. The half life of fibrinogen is 3-5 days. If our patient was afibrinogenemia, the level of fibrinogen would decrease over time due to its half life, but his fibrinogen level was normal until 13 days after the last plasma transfusion. Therefore, coagulopathy in this child was most likely due to DIC from massive bleeding and shock.

**Neurological evaluation**

The initial CT scan showed there were multiple stages of blood at the left convexity consistent with chronic subdural hematoma acute on top. It caused pressure to the left hemisphere and displaced the midline structure to the right side. This condition is uncommon in children. Some factors may precipitate this such as coagulopathy, underlying structural pathology or infection. The other possibility was child abuse.

But this condition should be ruled out. The patient's condition showed signs of increase intracranial pressure including the finding from the

CT scan. So the goal of treatment was to reduce the intracranial pressure. Usually the prognosis depends on the degree of cerebral damage. The finding in this case could not cause hypovolumic shock because the amount of blood was minimal. Then a burr hole with drainage was performed on the patient. Intraoperative finding showed motor oil blood about 10 ml, and the subdural hematoma was irrigated until clear. After that the neurological condition of the patient was observed in ICU until his condition was stable. During that time the hypofibrinogenemia condition was corrected with cryoprecipitate FFP by a pediatric hematologist until the drain was removed and the infant was monitored for possible postoperative complications such as recurrent bleeding, acute subdural hematoma, tension pneumocephalus and infection.

## CONCLUSION

We present a 1-month-old case with a history of minor blunt trauma and a continuous bleeding wound, suspecting a congenital coagulation defect with anemia and subsequent hypotension. The possible site of massive bleeding was investigated but showed no significant hemorrhage. Coagulogram was performed and there was coagulopathy so the fibrinogen and D-dimer level were tested. The coagulopathy of this child was corrected by a single dose of cryoprecipitate which proved that the etiology of coagulopathy in this child might be from DIC and not from a congenital disease. CT scan showed a chronic subdural hematoma acute on top. Child abuse was also suspected and a careful history was taken .After several follow ups, the infant's parents fully cooperated and took care of the infant very well so we concluded that anemia and chronic subdural hematoma in this child may have been due to unintentional and repeated minor trauma from his elder brother. In approaching the injured child, all the major systems were evaluated together, not emphasizing any one system, because in very small children either multiple organs involved or isolated severe head injury may manifests different physiological responses and presentations. Hypotension should not be viewed only as a potential marker of blood loss but also as a possible indicator of head injury.

## COMMENT

**Clinical Professor Chana Sathornkich**
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The complexity of pediatric trauma is not rare. Most cases require a multidisciplinary approach and the physician must act fast; for some incidence there is limited time. The reported case is one with a history of minor injury and an uncommon presentation of prolonged bleeding from a small abrasion wound . So the clue to solving this case was to find the cause of abnormal bleeding and shock. We were taught to rule out an intraabdominal solid organ injury as the first cause of traumatic hypovolemic shock but with a thorough history taken and physical examination we were able to investigate other causes on time.There are reports of isolated head injury that manifest hypotension in children but the mechanism is not clear since the amount of blood loss is minimal. However, in small children the amount of blood loss which may appear to be minor to us might not be as minor as we thought for children can tolerate a very low volume of blood loss. The cause of abnormal bleeding in this case was actually the consequence of hypovolemic shock; therefore, the appropriate treatment and investigation are the keys to managing such incidence. In order to achieve that goal, one should always be aware that abnormal bleeding has multiple causes from different pathways and can be either congenital or acquired.

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