

See discussions, stats, and author profiles for this publication at: <https://www.researchgate.net/publication/228692470>

Analysis of causes that led to baby Huda Sharif's intracranial and retinal bleeding and fractures of the left humerus and the 7th rib

Article · January 2009

CITATIONS

0

READS

229

1 author:



Mohammed Al-Bayati

Toxi-Health International

51 PUBLICATIONS 614 CITATIONS

SEE PROFILE

Analysis of causes that led to baby Huda Sharif's intracranial and retinal bleeding and fractures of the left humerus and the 7th rib

Mohammed Ali Al-Bayati, PhD, DABT, DABVT

Toxicologist & Pathologist

Toxi-Health International

150 Bloom Drive, Dixon, CA 95620

Phone: +1 707 678 4484 Fax: +1 707 678 8505

Email: maalbayati@toxi-health.com

Abstract

Huda was 10-weeks-old when her parents took her to the hospital on July 13, 2008. She was diagnosed with subdural hygroma, an acute left mid shaft humerus fracture, healed left 7th rib fracture, and minor retinal bleeding. Her blood coagulation and genetic osteogenesis imperfecta tests were normal. Her father was accused of causing the bone fractures and arrested. Huda and her 15-month-old sister were placed with a foster family.

My investigation reveals that the likely source of Huda's subdural hygroma is subdural bleeding. Huda was born at 40 weeks gestation by vacuum assisted Caesarian-section and her head circumference increased by 2.1 cm within 5 days. It is likely that the vacuum instrument caused subdural bleeding. Increased intracranial pressure led to retinal bleeding.

Vitamin K, vitamin D 25-hydroxy, and protein deficiency contributed to Huda's rib and humerus fractures. She had lost 167 g between July 7th and 24th. The synergistic actions among the following factors led to her humerus fracture: a) Vitamin and protein deficiency led to bone weakness; b) vaccines received on July 7th caused inflammation, edema, and the release of vitamin D, 1, 25-dihydroxy. Huda's serum level of vitamin D, 1, 25-dihydroxy was 166 pg/mL (369% of normal). It stimulated osteoclasts and increased bone resorption. Her length decreased by 1.9 cm within 6 days after vaccination; c) Edema led to her arm muscles to stretch and force the humerus to fracture at week points. Her weight gain rate after vaccination was 309% of her expected rate.

© Copyright 2009, Medical Veritas International Inc. All rights reserved.

Keywords: birth trauma, humerus fracture, rib fracture, retinal bleeding, subdural bleeding, subdural hygroma, vacuum assisted delivery, vitamin D, 1, 25-dihydroxy, vitamin K deficiency, vitamin D deficiency, and protein deficiency.

1. Summary of the case and findings

Huda was 10 weeks old when she was diagnosed with having subdural hygroma, an acute left mid shaft humerus fracture, healed left 7th rib fracture, and minor retinal bleeding. Huda's parents noticed that she was not moving her left arm on July 13, 2008 and took her to the Methodist hospital in Sacramento. An examination revealed that Huda's left arm was tender and had some swelling over the humerus area. No evidence of injury caused by trauma was noted on Huda's body.

An X-ray exam of the left arm showed that Huda had a displaced mid shaft humerus fracture. The nerve and the vascular structures were intact. Huda was given morphine and Tylenol and transferred to UC Davis Medical Center for further evaluation and treatment.

An X-ray exam was performed on July 14th showed that Huda had a left humeral acute transverse fracture. The fracture was reduced without surgical intervention and Huda's arm was placed in a sling and swathe to keep it immobilized. In addition, a CT scan taken on July 16th revealed that Huda had an acute displaced fracture of the left humerus and left healed 7th rib fracture. The X-ray and the CT exams also showed that Huda had anterior multiple bulbous rib ends.

Head CT scan and MRI exams performed on July 14th showed Huda had subdural fluid collections. Her X-ray exam revealed sutural diastasis and enlarged fontanelles.

Huda's blood coagulation and genetic osteogenesis imperfecta tests were normal. Huda was given Tylenol and pediatric multi-vitamin oral drops 0.5 ml by mouth each morning.

Huda was discharged from the hospital on July 22nd and given to a foster parent. Her treating physician, Dr. Kevin Coulter alleged that Huda's bone fractures were caused by inflicted trauma. Huda's father was accused of causing Huda's bone fractures and arrested. In addition, Huda's 15-month-old sister was also taken from the parents and given to a foster family.

The parents requested that I review the medical evidence in Huda's case and provide an opinion concerning the likely causes that led to Huda's injuries. I am a toxicologist and pathologist with over 20 years experience in these fields and have published over 50 articles in medical and scientific journals.

I have evaluated the medical files of many cases of children who died suddenly from unexplained causes and cases of children and adults who suffered from acute and/or chronic illnesses. I was able to explain the causes of illnesses and death in these cases using differential diagnosis. I have served as an expert witness in many medical-legal cases involving children and adults.

I evaluated Huda's medical records, the court transcript of Dr. Coulter's testimony, and the documents and articles cited in this report using differential diagnosis. Approximately 220 hours were required to evaluate the medical evidence, perform an analysis, and write this report. My investigation in this case reveals the following:

1) Huda was born at 40 weeks gestation by vacuum assisted Caesarian-section. Her head circumference (HC) following birth was 34.9 cm (50 percentile) and increased to 37 cm (95% percentile) within 5 days. The expected increase in her HC within 5 days is about 0.5 cm. However, no head CT scan, MRI

or ultrasound exam was performed to determine the cause(s) of the abnormal increase in Huda's HC.

The likely cause of the abnormal increase in Huda's HC is subdural bleeding caused by the vacuum assisted delivery. Clark *et al.* stated that the use of a vacuum device as a routine procedure at the time of repeat cesarean delivery has been associated with major fetal intracranial hemorrhage (Section 2).

For example, Alexander *et al.* conducted a prospective cohort study that included a total of 37,110 cesarean deliveries to describe the incidence and type of fetal injury identified in women undergoing cesarean delivery in the USA. They identified 418 (1.1%) had fetal injuries. Cephalohematoma was identified in 88 cases. Other injuries included clavicular fracture ($n = 11$), brachial plexus ($n = 9$), skull fracture ($n = 6$), and facial nerve palsy ($n = 11$), and skin laceration ($n = 272$, 0.7%). Among primary cesarean deliveries, deliveries with a failed forceps or vacuum attempt had the highest rate of injuries (6.9%).

2) Huda was taken to Dr. Barbara Ringwald's office on May 14th and June 24th and to the emergency room at Methodest hospital on June 7th because of health problems. Huda's head circumference was not measured and no head CT scan, MRI, or ultrasound exam was performed on these visits.

Furthermore, on June 24, 2008, Dr. Ringwald found 7 round bruises on Huda's midback. However, no clinical tests were performed to check for blood clotting problems and/or vitamin K deficiency. The likely cause of Huda's bruising is vitamin K deficiency. Huda's weight on June 7th and June 24th was 4400 g and 4233 g, respectively. The expected weight for Huda on June 24th is 5102 g (Section 3).

Significant reduction of food intake occurred in serious illnesses has known to lead to vitamin K deficiency and bleeding in children. Vitamin K controls the formation of coagulation factors II (prothrombin), VII (proconvertin), IX (Christmas factor), and X (Stuart factor) in the liver. In addition, two bone matrix proteins necessary for normal bone metabolism are also vitamin K-dependent, and vitamin K deficiency in infants can lead to bone fractures.

For example, Pooni *et al.* evaluated 42 infants who developed intracranial hemorrhage (ICH) and bleeding in other sites as a result of vitamin K deficiency. The majority of these infants (76%) were in the age group of 1-3 months. They found that 71% of these infants were presented with ICH. Visible external bleeding was noted in 1/3rd of the infants. Three infants died (Section 3).

3) The likely cause of Huda's rib fracture is vitamin K and D and protein deficiency. It is estimated that the age of the fracture is about 4 weeks old. It is likely that Huda's rib fracture occurred on or about June 24th (Section 9).

4) The primary source of Huda's subdural hygroma is subdural bleeding caused by the vacuum assisted delivery. The clinical data also indicate that Huda suffered from vitamin K deficiency that led to subdural bleeding (Section 7).

5) The likely cause of Huda's minor retinal hemorrhage is the increased intracranial pressure (ICP). For example, Medele *et*

al. performed prospective ophthalmological examination in 22 consecutive individuals with subarachnoid hemorrhage (SAH) or severe brain injury and elevated ICP. Thirteen individuals were admitted to the hospital for SAH and nine for severe brain injury. Monitoring of ICP was performed at the time of admission via a ventricular catheter. Initial ICP exceeded 20 mm Hg in all individuals.

Indirect ophthalmoscopy without induced mydriasis was performed within the 1st week after the acute event. Retinal or vitreous hemorrhage was seen in six (46%) of 13 individuals with SAH and in four (44%) of nine individuals with severe brain injury. Ocular bleeding was found bilaterally in three individuals with SAH and in one individual with severe brain injury (18%) (Section 8).

6) The humerus is a relatively strong bone in 10-week-old infant and it is not medically possible to break this bone without the use of a significant external force that causes external and internal injuries and bleeding in the arm. Huda had no external injury, injury to blood vessels, and bleeding in the tissues surrounding the bone.

The clinical data and the pertinent medical studies reveals that the synergistic actions of the following biological factors had led to Huda's left humerus fracture:

a) Huda suffered from vitamin K, vitamin D, and protein deficiency on or about June 23 and deficiency of these elements causes bone weakness and fractures. In addition, a blood analysis performed on July 14th showed that Huda had low levels of 25-hydroxy vitamin D, albumin, and total protein and mild anemia (Section 5).

b) Huda received 7 vaccines on July 7, 2008 that caused inflammation and systemic edema and the release of vitamin D, 1, 25-dihydroxy by inflammatory cells. Huda's serum level of vitamin D, 1, 25-dihydroxy on July 14th was 166 pg/mL (369% of average normal). Vitamin D, 1, 25-dihydroxy has known to stimulate osteoclasts and increases bone resorption.

For example, McSheehy and Chambers found that 1,25-dihydroxyvitamin D stimulated osteoclastic bone resorption in vivo and in organ culture. The osteoblastic cells induced a two-to fourfold stimulation of osteoclastic bone resorption in the presence of 1,25-dihydroxyvitamin D. Stimulation was observed at concentrations of 10(-10) M and above (Section 10).

Huda's length on July 7th and 14th were 55.9 cm (50%) and 54.0 cm (25%), respectively. Her length decreased by 1.9 cm within 6 days. In addition, Huda's length did not increase between July 14th and July 24th. It is expected that her length to increase by 1.25 cm in 10 days based on her length increase rate of 3.75 cm/month occurred between July 22nd and August 7th. These data indicate that Huda's skeletal growth was stopped and had bone resorption between July 7th and July 22nd.

c) Huda suffered from edema that led to her arm muscles to stretch and force the humerus to fracture at week points. Huda's weight on July 7th and July 13th was 4777 and 5220 g, respectively. She had gained 477 g (10% of her body

weight) in 6 days. She gained weight at the rate of 79.5 g/day which is 309% of her weight gain rate of 25.7 g/day. This occurred during the 62 days prior to receiving vaccines on July 7th.

Huda was treated with morphine (0.6 mg IV), phenobarbital, and Tylenol at 0010-1330 on July 14th and her weight reduced to 5000 g at 1600 on July 14th. She had lost 220 g (4.2% of her body weight) within 16 hours. These data indicate that Huda's body retained fluid following vaccination in response to health problems and pain (Section 5).

Primary Congenital Hypothyroidism	Negative
Congenital Adrenal Hyperplasia (due to 21-Hydroxylase Deficiency)	Negative
MS/MS Acylcarnitine Panel	Negative
MS/MS Amino Acid Panel (including PKU)	Negative
Hemoglobinopathies	Usual hemoglobin pattern

2.2 Huda's exam at 5 days of age and the abnormal increase in her head circumference

Dr. Barbara Ringwald examined Huda at 5 days of age and her head circumference (HC) was noted to be 37 cm (95%). Her HC at birth was 34.9 cm and it increased by 2.1 cm within 5 days (Table 2). The expected increase in her HC within 5 days is about 0.5 cm.

For example, Brandt evaluated head circumference growth rate in cm/month from the prenatal period until the age of 18 months. Measurements were made in 60 appropriate for gestational age (AGA) preterm infants of very low fetal age and 68 full term infants. His study revealed a mean growth velocity of 3.0 cm in the first, of 3.4 cm in the second and of 2.5 cm in the third month [6]. The estimated HC growth rate occurred in Huda's case was 12.6 cm/month, which is 420% of the normal rate.

However, no head CT scan, MRI, or ultrasound exam was performed on May 7, 2008 to determine the cause(s) of the abnormal increase in Huda's HC. Head CT and MRI exams performed on July 14, 2008 revealed that Huda had bilateral subdural fluid collections that indicate Huda suffered from subdural bleeding several weeks prior to July 14th [7].

Simonson *et al.* reviewed a cohort of 1,123 attempted vacuum extractions of singletons to evaluate the usefulness of skull X-ray and transfontanelar ultrasonography in detecting head injury in babies after vacuum extraction. Among 913 successful vacuum-assisted, full-term deliveries, 25.7% were admitted to the neonatal intensive care unit. Scalp edema, cephalohematoma, and skull fracture were assessed by cranial radiography and were present in 18.7%, 10.8%, and 5.0% of cases, respectively. Intracranial hemorrhage occurred in eight cases (0.87%) [8].

In addition, Castillo and Fordham *et al.* presented the MRI findings in three neurologically symptomatic newborns after vacuum extraction delivery. The lesions included subdural hematomas, one tentorial hematoma, and one intracerebellar hemorrhage. One patient had hydrocephalus that required shunting [9].

7) Dr. Coulter overlooked the factual causes of Huda's injuries and the clinical data that explain the mechanisms of her injuries. The clinical data and the medical studies described in this report do not support his allegations that Huda's humerus and rib fractures were caused by inflicted trauma.

2. Huda's health condition at birth and the likely causes of the abnormal increase in her head circumference occurred within five days following birth

Huda was born at 40 weeks gestation by vacuum assisted Caesarian-section. Vacuum was applied to Huda's head to assist in the delivery. Her head circumference (HC) following birth was 34.9 cm (50 percentile) and increased to 37 cm (95% percentile) within 5 days following birth.

The expected increase in her HC within 5 days is about 0.5 cm. However, no head CT scan, MRI or ultrasound exam was performed to determine the cause(s) of the abnormal increase in Huda's HC. The clinical data and medical studies described below and in this report indicate that Huda suffered from subdural bleeding caused by the vacuum assisted delivery [1-5].

2.1 Huda's condition at birth and treatments given

Huda was born at 0802 on May 2, 2008 at Methodist hospital in Sacramento. She was born at 40 weeks gestation by vacuum assisted C-section. Vacuum was applied to Huda's head to assist in the delivery. Her Apgar score was 8 and 9 at 1 and 5 minutes, respectively.

Huda's weight was 3.22 kg (50 percentile). Her length and head circumference were 47 (24 to 50 percentile) and 34.9 cm (50 percentile), respectively. Huda's mother stated that her pregnancy was unremarkable and she received prenatal care.

Huda was given 1 mg vitamin K (IM) following birth and hepatitis B vaccine on May 3rd. She developed mild facial jaundice on May 3th. Her serum total bilirubin level was 5.9 mg/dL (reference range: 6.0-8.0 mg/dL).

Huda's newborn screening tests were performed on May 3rd and revealed normal results (Table 1). Huda was discharged from the hospital on May 5th. She was fed formula and breast milk [1-5].

Table 1. Huda's Newborn Screening Tests

Test Type	Results
Cystic Fibrosis (CF)	Negative
Biotinidase Deficiency	Negative
Galactosemia	Negative

Furthermore, Whitby *et al.* conducted a prospective study in babies who were born in a major hospital in England to establish the frequency of subdural haemorrhages in asymptomatic term neonates. 111 babies underwent MRI in this study and 9 babies had subdural haemorrhages [10].

Table 2. Huda's weight, length, and head circumference measured at birth and within 5 days following birth

Date	Age (days)	Weight (g)	Length (cm)	HC (cm)
05/02/08	Birth	3221 (50%) ¹	47 (24-50%) ¹	34.9 (50)
05/03/08	1	3140	- ²	-
05/04/08	2	3105	-	-
05/05/08	3	3125	-	-
05/07/08	5	3182	48.3 (50%)	37 (95%)

¹ Percentile

² Not measured

2.3 The likely causes of the abnormal increase in Huda's head circumference

Huda's head circumference (HC) increased by 2.1 cm within 5 days. The expected HC increase in 5 days is 0.5 cm. The estimated increase rate in Huda's HC during her first 5 days following birth is 420% of normal as indicated by Brandt's study [6].

The likely cause of the abnormal increase in Huda's HC is subdural bleeding caused by the vacuum assisted delivery. Clark *et al.* stated that the use of a vacuum device as a routine procedure at the time of repeat cesarean delivery has been associated with major fetal intracranial hemorrhage [11].

For example, Alexander *et al.* conducted a prospective cohort study of all cesarean deliveries conducted at 13 university centers between January 1, 1999, and December 31, 2000 to describe the incidence and type of fetal injury identified in women undergoing Cesarean delivery in the USA. A total of 37,110 cesarean deliveries were included in the registry, and 418 (1.1%) had an identified fetal injury.

Cephalohematoma was identified in 88 cases. Other injuries included clavicular fracture (n = 11), brachial plexus (n = 9), skull fracture (n = 6), and facial nerve palsy (n = 11), and skin laceration (n = 272, 0.7%). Among primary cesarean deliveries, deliveries with a failed forceps or vacuum attempt had the highest rate of injuries (6.9%) [12].

In addition, Baume *et al.* conducted a retrospective study of two years activity involving 4524 deliveries of which 845 (18.7%) were vacuum extractor assisted. They precisely defined neonatal complications to compare their rates in spontaneous vaginal delivery and vacuum extractor groups. They found 114 neonatal complications. The difference was significant for cephalohematomas (OR=10; p<0.001) and scalp abrasions (OR=53; p<0.001) [13].

Furthermore, Lahat *et al.* reported a case of a term newborn who was delivered by vacuum extraction and subsequently developed signs of intracranial hemorrhage and in whom subdural hemorrhage was rapidly diagnosed and successfully removed [14].

3. Huda's health problems developed between May 12-June 24, 2008 and the likely causes

Huda was taken to Dr. Barbara Ringwald's office on May 14th and June 24th and to the emergency room at Methodist hospital on June 7th because of health problems. Huda's head circumference was not measured and no head CT scan, MRI, or ultrasound exam was performed on these visits. The clinical data and medical studies described in this report indicate that Huda suffered from subdural bleeding caused by the vacuum assisted delivery (Section 2).

Furthermore, on June 24, 2008, Ringwald found 7 round bruises on Huda's midback. However, no clinical tests were performed to check for blood clotting problems and/or vitamin K deficiency. The clinical and medical studies described below indicate that Huda's bruising was caused by vitamin K deficiency. Huda's weight on June 7th and June 24th was 4400 g and 4233 g, respectively. The expected weight for Huda on June 24th is 5102 g.

Significant reduction of food intake that occurred in serious illness has known to lead to vitamin K deficiency and bleeding in children. Vitamin K controls the formation of coagulation factors II (prothrombin), VII (proconvertin), IX (Christmas factor), and X (Stuart factor) in the liver. In addition, two bone matrix proteins necessary for normal bone metabolism are also vitamin K-dependent and vitamin K deficiency in infants can lead to bone fractures.

3.1 Huda's symptoms observed at 12 days of age

Huda was sick on May 14th and her parents took her to Dr. Barbara Ringwald's office. She was vomiting for 3 days. Huda's temperature was 98.4°F. Huda was fed Similac formula and it was changed to a soy-based formula.

Huda's weight was 3409 g and she had gained 188 g (15.7 g/day) since birth. Her head circumference was not measured at this visit to evaluate the change in size that had occurred since May 7th. In addition, no head CT scan, MRI, or ultrasound exam was performed at this visit.

3.2 Huda's symptoms reported at 36 days of age

Huda was seen at Methodist Hospital at 0020 on June 7, 2008 because of an episode of a pause in her breathing that occurred during a crying episode. She apparently stiffened up during the episode, her face appeared gray, and her parents took her to the hospital.

Huda's exam revealed that her oxygen saturation was 97%. She had a heart rate of 140 beats/minutes, a blood pressure of 118/47, and respiratory rate of 48. Her temperature was 36.8°C. She was diagnosed with having colic and was given gas relief medication.

Huda's weight was 4400 g and she had gained 991g (41.3 g/day) since May 14th. Her length and head circumference were not measured at this visit.

3.3 Huda's bruises observed at 54 days of age and the likely causes

Huda was taken to Dr. Ringwald's office on June 24, 2008. Her parents noticed bruising on her back on June 23th. Ringwald examined Huda and found 7 round appearing bruises on her midback. The parents stated that they had no knowledge of how the bruising occurred.

No clinical tests were performed to check for blood clotting problems and/or vitamin K deficiency. Huda's weight on June 7th and June 24th was 4400 g and 4233 g, respectively. She had lost 167 g in 17 days (-9.8 g/day). The expected weight for Huda on June 24th is 5102 g [1, 2].

The likely cause of Huda's bruising observed on June 23rd is vitamin K deficiency. In humans, the body does not synthesize the 1, 4 naphthoquinone nucleus of vitamin K and gets it from food. In addition, the bacteria in the intestinal tract synthesize vitamin K and can supply part of the vitamin K requirement. Significant reduction of food intake that occurred in serious illness has known to lead to vitamin K deficiency and bleeding in children.

Vitamin K controls the formation of coagulation factors II (prothrombin), VII (proconvertin), IX (Christmas factor), and X (Stuart factor) in the liver. Other coagulation factors that depend on vitamin K are proteins C, S, and Z. Vitamin K-dependent proteins contain the amino acid γ -carboxyglutamic acid and the carboxyl groups of the glutamic acid residues that provide the vitamin-K-dependent proteins with characteristic calcium and phospholipid binding properties. Vitamin K deficiency has led to the production of abnormal vitamin K-dependent factors, which lack gamma-carboxy glutamic acid residues in the NH₂-terminal part of their molecules [15-21].

Pooni *et al.* evaluated 42 infants who developed intracranial hemorrhage (ICH) and bleeding in other sites as a result of vitamin K deficiency. The majority of these infants (76%) were in the age group of 1-3 months. They found that 71% of these infants presented with intracranial hemorrhage. The most common sites were intracerebral and multiple ICH. Visible external bleeding was noted in 1/3rd of the infants. Three infants died [22].

In addition, Nishio *et al.* examined 84 cases of children with intracranial hemorrhage caused by vitamin K deficiency published in literatures. Hemorrhage sites were identified by CT scans in these children. Subarachnoidal hemorrhage was in 72 cases (85.7%), subdural hemorrhage was in 41 cases (48.8%), intracerebral hematomas was in 36 cases (42.9%) and intraventricular hemorrhage was in 9 cases (10.7%) [18].

Furthermore, Choo *et al.* conducted a retrospective study of 42 newborns admitted to the hospital for spontaneous bleeding. Subdural hemorrhage was the commonest form of intracranial haemorrhage, followed by subarachnoid haemorrhage. None of the infants had bleeding due to inherited coagulopathy or disseminated intravascular coagulation [23].

Doneray *et al.* also described the clinical and demographic features of 16 cases with vitamin K deficiency bleeding. Ages of infants were between 30 and 130 days. Intracranial haemorrhage

was the most common bleeding site (37.5%), and two children (12.5%) died because of it [24].

Two bone matrix proteins necessary for normal bone metabolism are also vitamin K-dependent and vitamin K deficiency in infants can lead to bone fractures. Huda's chest CT scan taken on July 14th showed healing fracture of the 7th rib that is about 2-4 weeks old [7]. It is likely that this rib fracture occurred at the time of the bleeding due to vitamin K deficiency. No chest X-ray exam was performed in Huda's case on June 24th.

4. Huda's vaccinations on July 7, 2008 and health problems reported on July 13th

Huda was vaccinated with DTaP; HIB; PNUcon; IPV (oral) and Rotaviru on July 7th. An X-ray exam of the left arm performed on July 13th revealed that Huda had a displaced mid-shaft humeral fracture. The nerve and the vascular structures were intact and no sign of injury caused by trauma was noted on her body.

During the 6 days following vaccination, Huda gained 447 g, her head circumference increased by 1 cm, and her length decreased by 1.9 cm. Huda was treated with morphine, Tylenol, and phenobarbital and had lost 220 g within 16 hours following receiving these medications.

4.1 Vaccines given to Huda on July 7th

Huda was crying a lot on July 7th and vomited on certain occasions. Her head appeared relatively large and she did not keep her head straight. Her parents took her to Dr. Ringwald's office.

Ringwald examined Huda and felt that Huda was suffering from colic. Huda's weight, length, and head circumference were 4773 g, 55.9 cm, and 41.5 cm, respectively. She was vaccinated with DTaP; HIB; PNUcon; IPV (oral) and Rotaviru at sent home.

4.2 Huda's health condition in the evening of July 13th

Huda was crying in the evening of July 13th and her father picked her up. He gave her to his wife and they noticed that Huda was not moving her left arm. Her arm was tender when it was touched.

Huda's 15-month-old sister was standing near her in the living room when she started to cry. The father was in the bathroom and the mother was in the bedroom when they heard Huda crying. They lived in a single bedroom apartment. The parents took Huda to the emergency room at Methodist Hospital for examination.

4.3 Clinical tests performed at Methodist, findings, and treatments given

Huda brought to the ER at Methods hospital on July 13th. A physician examined her at 2122. She had a pulse of 164/minute,

respiratory rate of 28/minute, and temperature of 37 °C. No sign of injury caused by trauma was noted on her body.

Huda's left arm was tender and had some swelling over the humerus area. An X-ray exam of the left arm was performed at 2230 and revealed that Huda had a displaced mid shaft humeral fracture, angulated at about 45 degrees. The nerve and the vascular structures were intact.

Huda's weight was 5.220 kg. Her length and head circumference (HC) were 54 and 42.5 cm, respectively. Huda was given 80 mg of Tylenol (oral); 0.1 mg morphine sulfate (IV); and saline (IV). She was transferred to UC Davis medical Center at 0030 on July 14th for further examination and treatment.

4.4 Changes in Huda's growth parameters occurred between July 7th and 13th

Huda received 7 vaccines on July 7th. Her weight, length, and HC measured on July 7th and at 6 days following vaccination is presented in Table 3. She had gained 477 g (10% of her body weight) in 6 days. Huda's weight gain rates during the 62 days prior to receiving vaccines on July 7th and the 6 days following vaccinations were 25.7 g/day and 79.5 g/day, respectively.

Huda's weight gain rate following vaccination was 309% of her average weight gain rate occurred during the 62 days prior to receiving vaccines on July 7th. Huda was treated with morphine (0.6 mg IV), phenobarbital, and Tylenol at 0010-1330 on July 14th and her weight reduced to 5000 g at 1600 on July 14th. She had lost 220 g (4.2% of her body weight) within 16 hours. These data indicate that Huda's body retained fluid following vaccination in response to health problems and pain.

In addition, Huda's HC increased by 1 cm within the 6 days (5 cm/month) following vaccination. However, her HC increased rate during the period between May 7th and July 7th (prior vaccination) was 2.18 cm/month. Huda's HC growth rate during the 6 days following vaccination is equal to 229% of her HC growth rate occurred during the 62 days prior vaccination.

Huda's length increase rate between May 7th and July 7th was 3.68 cm/month. However, Huda's length decreased by 1.9 cm during the 6 days following receiving vaccines on July 7th. These data indicate that vaccines had a significant negative impact on Huda's skeletal growth.

Table 3. Huda's weight, length, and head circumference measured at 5 days of age and on July 7th and 13th

Date	Age (days)	Weight (g)	Length (cm)	HC (cm)
05/07/08	5	3182 (50%) ¹	48.3 (50%)	37.0 (95%)
07/07/08	67	4773 (50%)	55.9 (49%)	41.5 (95%)
07/13/08	73	5220 (75%)	54.0 (25%)	42.5 (95%)

¹Percentile

5. Huda's hospitalization at UCD Medical Center on July 14-22, 2008: clinical tests, health problems, and treatments

Huda was transferred to UC Davis Medical Center (UCDMC) on July 14th for further examination and treatments. She was admitted at 0030. Huda had a pulse of 146/minute, a blood pressure of 98/42 mm Hg, and a respiratory rate of 21/minute. Examination revealed that she had no external injury that indicates trauma.

Huda's weight was 5235 g. Her length and head circumference were 54 and 42.5 cm, respectively. She was treated with morphine (0.5 mg, IV), Tylenol, and phenobarbital (10 mg IV), and given IV fluid at 0345-1000 on July 14th. Huda's weight decreased to 5000 g within 15 hours following her treatment with these medications.

An X-ray exam was performed at 1117 on July 14th showed that Huda had left humeral acute transverse fracture (Table 4). The surrounding soft tissue was swollen. The nerve and the blood vessel structures were intact. The fracture was reduced without surgical intervention and Huda's arm was placed in a sling and swathe to keep it immobilized.

Head CT scan and MRI exams performed on July 14th showed Huda had subdural fluid collections. Her X-ray exam revealed sutural diastasis and enlarged fontanels (Table 5). The likely source of Huda's subdural fluid collections is an old subdural bleeding.

A serum analysis performed at 0630 on July 14th showed that Huda's levels of 25-hydroxy vitamin D, albumin, and total protein were below the normal range. A serum analysis performed at 1645 revealed that Huda had a very high level of vitamin D, 1, 25-dihydroxy. Her level was 369% of average normal (Table 6).

A blood analysis performed at 0630 on July 14th showed Huda was slightly anemic. Her hematocrit value and MCH level were slightly below the normal range and her RDW% value was higher than normal. Huda's white blood cell and differential counts were within the normal range and her urine analysis was normal (Tables 7, 8).

Huda's coagulation tests on July 14th were within the normal range (Table 6). Huda was given daily multivitamin supplements (poly-vi-sol) orally. An eye exam performed at 1300 on July 15th revealed that Huda had minor retinal hemorrhage in both eyes that did not require intervention.

A CT scan taken on July 16th revealed that Huda had an acute displaced fracture of the left humerus and left healed 7th rib fracture. The X-ray and the CT exams also showed that Huda had anterior multiple bulbous rib ends (Table 4). Huda's genetic osteogenesis imperfecta test was normal. Huda continued to appear having pain associated with her humerus fracture when she was held. She was given Tylenol.

Huda fed a Similac formula during her hospitalization. Her weight on July 22nd was 20 g higher than her weight at admission. Her head circumference reduced by 0.5 cm between July 14th and July 22nd and her length did not change (Table 9).

Huda was discharged from the hospital on July 22nd. Her discharge medications included pediatric multivitamin oral drops 0.5 ml by mouth each morning and Tylenol for pain. Below are the clinical data collected during her 8 days hospitalization.

5.1 An acute left humerus fracture and healed rib fracture

An X-ray exam was performed at 1117 on July 14th showed that Huda had left humeral acute transverse fracture. The nerve and blood vessels structures were intact. The surrounding soft tissue was swollen. The fracture was reduced without surgical intervention and Huda's arm was placed in a sling and swathe to keep it immobilized.

A follow up X-ray exam was performed on July 14th showed a mild surrounding tissue swelling. A CT scan taken on July 16th revealed that Huda had an acute displaced fracture of the left humerus and left healed 7th rib fracture. The X-ray and the CT exams also showed anterior multiple bulbous rib ends (Table 4).

Table 4. Huda's X-ray and CT exams of the left arm and the chest performed on July 14-21, 2008

Date & Time	Exam Type	Findings
07/14/08 (1117)*	X-ray	<ul style="list-style-type: none"> • Transverse mid shaft left humeral fracture, with one-half-width displacement of the distal fragment. • The surrounding soft tissue was swollen. • Anterior rib ends at multiple levels appeared somewhat bulbous.
07/14/08 (1408)	X-ray	<ul style="list-style-type: none"> • Post-reduction film of the left humeral mid shaft fracture showing satisfactory alignment with mild lateral angulation. • Mild surrounding soft tissue swelling remained.
07/16/08 (1040)	CT Scan	<ul style="list-style-type: none"> • Left humeral acute displaced fracture. • Left healed posterior 7th rib fracture. • Anterior multiple bulbous rib ends.
07/20/08 (1126)	X-ray	<ul style="list-style-type: none"> • Displaced transverse fracture through the mid shaft of the left humerus with apex angulation laterally.
07/21/08 (1444)	X-ray	<ul style="list-style-type: none"> • Displaced transverse fracture through the mid shaft of the left humerus with apex angulation laterally.

5.2 Head CT scan, X-ray, and MRI exams

Head CT scan and MRI exams performed on July 14th showed Huda had subdural fluid collections. Her X-ray exam revealed sutural diastasis and enlarged fontanels (Table 5). The likely source of Huda's subdural fluid collections is an old subdural bleeding.

No evidence of an acute injury caused by trauma observed on Huda's head. Huda's head circumference increased from 34.9 cm to 37 cm within 5 days following birth. The clinical and medical studies described in Section 2 of this report indicate that Huda suffered from subdural bleeding caused by the vacuum assisted delivery.

Table 5. Huda's head CT scan, X-ray, and MRI exams performed on July 14, 2008

Time	Exam Type	Findings
0845	CT scan	<ul style="list-style-type: none"> • Prominent extra-axial fluid that has slightly higher density than that in the lateral ventricles and appeared to be predominantly subdural in position. • Mild flattening of the brain. • Sinus and mastoid congestion or inflammatory change.
1117	X-ray	<ul style="list-style-type: none"> • Sutural diastasis and enlarged fontanels
1315	MRI	<ul style="list-style-type: none"> • Bilateral subdural fluid collections, unchanged compared to previous CT scan study. • Mild ventriculomegaly.

5.3 Blood tests and abnormal values

A serum analysis performed at 0630 on July 14th showed that Huda had low levels of 25-hydroxy vitamin D, albumin, and total protein. Her serum analysis performed at 1645 revealed a very high level of vitamin D, 1, 25-Dihydroxy that is equal 369% of average normal (Table 6).

A blood analysis performed at 0630 on July 14th showed that Huda was slightly anemic. Her hematocrit value and MCH level were slightly below the normal range and her RDW% value was higher than normal. Her white blood cell and differential counts were within the normal range (Table 7).

Table 6. Huda's serum analysis performed on July 14, 2008

Measurements	Values	Reference Range
25-hydroxy vitamin D (ng/mL)	26.2	32.0
Vitamin D, 1, 25-dihydroxy (pg/mL)	166	15-75
Albumin (g/dL)	3.0	3.8-5.4
Protein (g/dL)	5.2	5.4-7.5
Glucose	70	60-105 mg/dL
Sodium	139	133-142 mEq/L
Potassium	5.1	4.0-6.02 mEq/L
Chloride	108	95-110 mEq/L
Calcium	9.7	7.3-12 mg/dL
Phosphorus	6.9	5.0-7.0 mg/dL
Magnesium	2.1	1.2-2.6 mg/dL
BUN	13	5-14 mg/dL
Creatinine	0.25	0.10-0.50 mg/dL
ALP	217	50-260 U/L
AST	37	5-35 U/L
ALT	24	5-54
Bilirubin direct	<0.1	0.0-0.2 mg/dL
Total Bilirubin	0.5	0.2-0.9 mg/dL
Factor XIII Screen	>2	>2%
Ristocetin cofactor	131	50-150%
INR	0.96	0.75-1.19
Vonwillebrand's antigen	132	50-150% NHP

Table 7. Huda's hematology values measured on July 14, 2008

Measurements	Values	Reference Range
Red blood cell x 10 ⁶ /μL	3.67	3.1-4.3
Hemoglobin (g/dL)	9.1	9.0-14.0
Hematocrit%	27.4	28-42%
MCV fL	75.2	77-115
MCH pg	24.9	27-33
MCHC (g/dL)	33.1	32-36
RDW%	22.0	12.5-14.7
MPV (fL)	7.4	6.8-10.0
Platelet x 10 ³ /μL	361	130-400
White blood cell count x 10 ³ /μL	10.5	5.0-19.3
Neutrophils	3.30	1.0-9.0
Lymphocytes	6.1	2.5-16.5
Monocytes	0.8	0.1-0.8
Basophils	0.3	0.0-0.5

5.4 Urine test performed on July 14th

Huda's urine analysis performed at 2300 on July 14th revealed normal results (Table 8).

Table 8. Huda's urine analysis performed on July 14, 2008

Measurements	Values	Reference Range
Color	Yellow	None/Yellow
Clarity	Clear	Clear-Slight Turbid
Specific gravity (g/mL)	1.010	1.002-1030
PH	7.5	4.8-7.8
Occult blood	Negative	Negative
Bilirubin (mg/dL)	Negative	Negative
Ketones (mg/dL)	Negative	Negative
Glucose (mg/dL)	Negative	Negative
Clinitest	Negative	Negative
Protein (mg/dL)	Negative	Negative/Trace
Urobilinogen (mg/dL)	0.2	0-0.2
Nitite	Negative	Negative
Leuk. Esterase	Small	Negative

5.5 Eye exam

An eye exam performed at 1300 on July 15th revealed that Huda had a minor retinal hemorrhage in both eyes that did not require intervention. The likely cause of this bleeding is the increased intracranial pressure (Section 8).

5.6 Changes in Huda's weight, length, and head circumference occurred at the hospital

Huda's weight reduced by 235 g (4.5% of body weight) within 15 hours following her treatment with morphine, phenobarbital, and Tylenol. It indicates that her body was retaining fluid in response to pain. Huda's weight increased by 255 g between 1600 on July 14th and 22nd (31.9 g/day).

Huda's head circumference increased by 0.5 cm between July 14th and 15th and then reduced by 1 cm. Her length did not change between July 14th and July 22nd (Table 9). It is expected that Huda's length to increase by 1 cm between July 14th and 22nd based on her previous growth rate of 3.68 cm/month occurred between May 7th and July 7th.

Table 9. Huda's weight, length and head circumference measured on July 14-22, 2008

Date	Time	Weight (g)	Length (cm)	HC (cm)
07/14/08	0030	5235 (75%) ¹	54 (5%)	42.5 (95%)
07/14/08	0330	5200	54	42.5
07/14/08	1330	5040	54	42.5
07/14/08	1600	5000 (43%)	54 (5%)	42.5
07/15/08	2000	5015	54	43
07/16/08	2000	5065	-	42
07/22/08	0100	5255	54	42

¹ Percentile

6. Huda's health condition during the period between July 22nd and September 5th, 2008

Huda was discharged from the UC Davis Medical Center on July 22nd to a foster family because of an allegation of child abuse. Her discharge medications included pediatric multivitamin oral drops 0.5 mL by mouth each morning and Tylenol for pain. Huda was examined 4 times at the hospital and her pediatrician between August 7th and 28th because of health problems. In addition, a surgical procedure was performed on September 5th to place subdural to peritoneum shunt to drain Huda's subdural fluid.

1) Huda was examined on August 7th. She had a pulse of 122/minutes, a respiratory rate of 38/minute, and a temperature of 96.7°F. Her weight was 5,805 g. Her length and head circumference (HC) were 56 and 44.5 cm, respectively. Huda's weight, length, and HC had increased at the rate of 34.4 g/day, 3.75 cm/month, and 4.69 cm/month, respectively since July 22nd.

2) An X-ray exam was performed on August 14th and revealed that Huda's left humerus mid shaft fracture was healing very well.

3) Examination on August 21st revealed that Huda had a pulse of 144/minutes, a respiratory rate of 38/minute, and a temperature of 97.1°F. A head CT scan exam revealed a slight enlargement of the third and lateral ventricles and slight increase in the total volume of the extraaxial fluid as compared with Huda's CT and MRI exams of July 14th. It also revealed the presence of left mastoid and middle ear space fluid.

Huda's HC on July 22nd and August 19th was 42 cm, and 45.5 cm, respectively. Her HC increased by 3.5 cm in 30 days. Huda's weight was 6190 g and she had gained 385 g (27.5 g/day) since August 7th.

4) Huda's examination on August 28th revealed that she had a pulse of 146/minutes, a respiratory rate of 50/minute, and a temperature of 36.4°C. Her blood analysis showed that her anemia had improved since July 22nd. Her red blood cell count, hemoglobin level, and hematocrit value increased by 18% (Table 10). Her urine analysis was normal.

Huda's weight was 6,448 g. Her length and head circumference were 61.6 and 45.5 cm, respectively. She had gained 258 g (28.7 g/day) since August 19th. Her length had increased by 5.6 cm (8 cm/month) since August 7th.

5) Huda had an operation on September 5th and a right parieto-occipital subdural to peritoneum shunt was placed to drain the fluid. Her weight and length were 6400 g and 60 cm, respectively. She had lost 48 g and her length reduced by 1.6 cm since August 28th.

Table 10. Huda's hematology values measured on July 14th and August 28th

Measurements	Values		Reference Range
	July 14 th	Aug. 28 th	
Red blood cell x 10 ⁶ /μL	3.67	4.37	3.1-4.3
Hemoglobin (g/dL)	9.1	10.7	9.0-14.0
Hematocrit%	27.4	32.6	28-42%
MCV fL	75.2	74.7	77-115
MCH pg	24.9	24.5	27-33
MCHC (g/dL)	33.1	32.7	32-36
RDW%	22.0	17.3	12.5-14.7
MPV (fL)	7.4	7.6	6.8-10.0
Platelet x 10 ³ /μL	361	426	130-400
White blood cell count x10 ³ /μL	10.5	11.0	5.0-19.3
Neutrophils	3.30	3.60	1.0-9.0
Lymphocytes	6.1	5.9	2.5-16.5
Monocytes	0.8	1.0	0.1-0.8
Basophils	0.3	0.0	0.0-0.5
Glucose (mg/L)	70	85	60-105
Sodium (mEq/L)	139	136	133-142
Potassium (mEq/L)	5.1	4.3	4.0-6.02
Chloride (mEq/L)	108	105	95-110
Calcium mg/dL	9.7	10.4	7.3-12
BUN (mg/dL)	13	11	5-14

7. Progress of Huda's subdural hygroma and the likely causes

Head CT scan and MRI exams performed on July 14, 2008 revealed the presence of abnormal fluid collection in Huda's subdural space that has slightly higher density than that in the lateral ventricles. A head CT scan was performed on August 19th and compared with Huda's CT and MR exams of July 14th. It showed a slight increase in the total volume of the extraaxial fluid.

Huda had an operation on September 5th and a right parieto-occipital subdural to peritoneum shunt was placed to drain the fluid. The clinical data described below and in this report indicate that the primary source of Huda's subdural hygroma is subdural bleeding caused by the vacuum assisted delivery. The clinical data also indicate that the Huda suffered from vitamin K deficiency that led to subdural bleeding.

1) Huda's head circumference at birth was 34.9 cm and increased to 37 cm within 5 days. It increased by 2.1 cm and the expected increase in her HC within 5 days is about 0.5 cm. (Table 11). Huda was born by vacuum assisted Caesarian-section. Subdural bleeding has been reported in some infants in association with the use of vacuum instrument to assist delivery (Section 2).

Brandt evaluated head circumference growth rate in cm/month from the prenatal period until the age of 18 months. Measurements were made in 60 appropriate for gestational age (AGA) preterm infants of very low fetal age and 68 full term infants. His study revealed a mean growth velocity of 3.0 cm in the first, of 3.4 cm in the second and of 2.5 cm in the third

month [6]. The estimated HC growth rate occurred in Huda's case was 12.6 cm/month, which is 420% of the normal rate.

2) Dr. Barbara Ringwald examined Huda on June 24th and found 7 round appearing bruises on her midback. Huda's weight on June 7th and June 24th was 4400 g and 4233 g, respectively. She had lost 167 g in 17 days (-9.8 g/day). The expected weight for Huda on June 24th is 5102 g.

Signification reduction of food intake occurred in serious illness has known to lead to vitamin K deficiency and bleeding in children. Vitamin K controls the formation of coagulation factors II (prothrombin), VII (proconvertin), IX (Christmas factor), and X (Stuart factor) in the liver. Vitamin K deficiency has known to cause subdural bleeding in infants (Section 3).

For example, Nishio *et al.* examined 84 cases of children with intracranial hemorrhage caused by vitamin K deficiency published in literatures. Hemorrhage sites were identified by CT scans in these children. Subarachnoidal hemorrhage was in 72 cases (85.7%), subdural hemorrhage was in 41 cases (48.8%), intracerebral hematomas was in 36 cases (42.9%) and intraventricular hemorrhage was in 9 cases (10.7%) [18].

3) Huda's head circumference (HC) increased at a high rate of 4.69 cm/month between July 22nd and August 7th. Her HC also increased at the rate of 5.0 cm/month between August 13th and 19th (Table 11). Huda's head CT scan of August 19th showed a slight increase in the total volume of the extraaxial fluid and compared with Huda's CT and MR exams of July 14th. These data indicate that Huda had subdural bleeding following her release from the hospital on July 22nd.

Table 11. Huda's head circumference measurements

Date	Age (days)	HC (cm)
05/02/08	Birth	34.9 (50%)*
05/07/08	5	37 (95%)
07/07/08	67	41.5
07/13/08	73	42.5 (95%)
07/15/08	75	43.0
07/22/08	82	42.0
08/07/08	98	44.5
08/13/08	104	44.5
08/19/08	110	45.5 (100%)
08/28/08	119	45.5

*: Percentile

8. The likely cause of Huda's retinal bleeding

An eye exam performed at 1300 on July 15th revealed that Huda had a minor retinal hemorrhage in both eyes that did not require intervention. The likely cause of her retinal bleeding is the increased intracranial pressure (ICP). The arteries that perfuse and the vein that drains the orbit are exposed to the ambient ICP while coursing through the cerebrospinal fluid or optic nerve [25].

Querfurth *et al.* measured the venous outflow pressure (VOP) of the central retinal vein using occlusion in six intensive care patients treated for acute hydrocephalus or brain hemorrhage and in whom transducers of intracranial pressure used

to provide standardized continuous output. Simultaneously, the central retinal (CRA) and ophthalmic (OA) arterial flow velocities were recorded using color Doppler imaging technique. Linear regression and correlation testing were performed to evaluate these variables for ICP predictability [25].

They found that the VOP increased linearly with ICP ($r=0.87$). The arterial pulsatility indices for both OA and CRA decreased inversely with ICP ($r=0.66$). An empiric index combining both venous and arterial parameters (VOP/Gosling Pulsatility Index [GPI]) was significantly more correlated with absolute ICP than either parameter alone ($r=0.95$, $p<0.005$ [25].

Head CT scan and MRI exams of July 14th showed that Huda had subdural hygroma (Section 5 and 7). Her head circumference (HC) on July 7th and July 15th were 41.5 and 43 cm, respectively. It increased by 1.5 cm within 8 days (5.63 cm/month) (Section 5 and 7). A head X-ray exam performed on July 14th revealed that Huda had sutural diastasis and enlarged fontanels. A sudden rise in the ICP has caused intraocular bleeding in some individuals.

For example, Medele *et al.* performed prospective ophthalmological examination in 22 consecutive individuals with subarachnoid hemorrhage (SAH) or severe brain injury and elevated ICP. Thirteen individuals were admitted to the hospital for SAH and nine for severe brain injury. Monitoring of ICP was performed at the time of admission via a ventricular catheter. Initial ICP exceeded 20 mm Hg in all individuals.

Indirect ophthalmoscopy without induced mydriasis was performed within the 1st week after the acute event. Retinal or vitreous hemorrhage was seen in six (46%) of 13 individuals with SAH and in four (44%) of nine individuals with severe brain injury. Ocular bleeding was found bilaterally in three individuals with SAH and in one individual with severe brain injury (18%)[26].

Furthermore, Stiebel-Kalish *et al.* evaluated the medical records of 70 individuals with subarachnoid hemorrhage resulted from ruptured cerebral aneurysms. They found that 30 eyes of 19 individuals had intraocular hemorrhages; 14 eyes had a vitreous hemorrhage; 12 eyes had subhyaloid blood without a vitreous hemorrhage; and 4 eyes had retinal hemorrhages alone [27].

9. The likely causes of Huda's 7th rib fracture

A chest CT scan performed on July 16, 2008, revealed that Huda had a left posterior healed 7th rib fracture and anterior multiple bulbous rib ends. The likely causes of Huda's rib fractures are vitamin K and D and protein deficiency. It is estimated that the age of the fracture is about 4 weeks old. It is likely that Huda's rib fracture occurred on or about June 24th.

Huda's weight on June 7th and June 24th was 4400 g and 4233 g, respectively. The expected weight for Huda on June 24th is 5102 g. Significant reduction of food intake that occurred in serious illness has known to lead to vitamin K deficiency. Matrix proteins necessary for normal bone metabolism are also vitamin K-dependent and vitamin K deficiency in infants can lead to bone fractures (Section 3). No chest X-ray exam was performed in Huda's case on June 24th to check for rib fractures.

In addition, vitamin K deficiency has also caused bleeding in infants. Vitamin K controls the formation of coagulation factors

II (prothrombin), VII (proconvertin), IX (Christmas factor), and X (Stuart factor) in the liver. Dr. Barbara Ringwald found 7 round bruises on Huda's midback on June 24th. It is likely that these bruises were resulted from vitamin K deficiency (Section 3). Ringwald did not perform clinical tests to check for blood clotting problems and/or vitamin K deficiency.

I evaluated the medical records of infants who had subdural bleeding and bone fractures (skull fractures and/or rib fractures) and differential diagnosis identified vitamin K and protein deficiencies are the primary causes [17, 28, 29].

Vitamin K is essential for bone development and health. Some of the bone matrix proteins necessary for normal bone metabolism are vitamin K-dependent. Vitamin K is a coenzyme for glutamate carboxylase that mediates the conversion of glutamate to gamma-carboxyglutamate (Gla). There are at least three Gla proteins associated with bone tissue. Osteocalcin is the most abundant Gla and it is the major non-collagenous protein incorporated in bone matrix during bone formation. Gla residues attract Ca²⁺ and incorporate these ions into the hydroxyapatite crystals [15, 30–34].

Bugel found that vitamin K deficiency in people results in an increase in undercarboxylated osteocalcin, a protein with low biological activity. Several studies have demonstrated that low dietary vitamin K intake is associated with low bone mineral density or increased fractures. Additionally, vitamin K supplementation has been shown to reduce undercarboxylated osteocalcin and improve the bone turnover profile. Some studies have indicated that high levels of undercarboxylated osteocalcin are associated with low bone mineral density and increased hip fracture [31].

Shiraki *et al.* investigated the effectiveness of vitamin K2 (menatetrenone) treatment in preventing incidence of new fractures in osteoporotic individuals. A total of 241 osteoporotic individuals were enrolled in a 24-month randomized open label study. The control group (without treatment; n = 121) and the vitamin K2-treated group (n = 120), which received 45 mg/day orally vitamin K2.

These individuals were followed for lumbar bone mineral density (LBMD; measured by dual-energy X-ray absorptiometry [DXA]) and occurrence of new clinical fractures. Serum level of Glu-osteocalcin (Glu-OC) and menaquinone-4 levels were also measured at the end of the follow-up period. They found that the incidence of clinical fractures during the 2 years of treatment in the control was higher than the vitamin K2-treated group ($\chi^2 = 10.935$; $p = 0.0273$) [33].

Furthermore, Booth *et al.* conducted a study to determine the associations between vitamin K intake and hip fracture in a population-based cohort of elderly men and women. They found that low vitamin K intakes were associated with an increased incidence of hip fractures in this cohort of elderly men and women. They assessed the dietary vitamin K intake and the incidence of hip fractures in 335 men and 553 women. They found that individuals in the highest quartile of vitamin K intake (median: 254 μg per day) had a significantly lower fully adjusted relative risk (0.35; 95% CI: 0.13, 0.94) of hip fracture than did those in the lowest quartile of intake (median: 56 $\mu\text{g}/\text{day}$) [34].

Huda had lost weight between June 7th and 24th and her body was not getting enough protein because of health problems.

Proteins are important for bone development and health. Rizzoli *et al.* reported that protein deficiency contributes to the occurrence of osteoporotic fractures not only by decreasing bone mass but also by altering muscle function [35]. Tanaka *et al.* also stated that protein malnutrition increases the fracture risk due to decreased bone mineral density and muscle weakness [30].

10. The likely causes of Huda's left humerus fracture

Huda was admitted to the Methodest hospital at 2122 on July 13, 2008. Her left arm was tender and had some swelling over the humerus area. An X-ray exam of the left arm was performed at 2230 and revealed that Huda had a displaced mid shaft humeral fracture, angulated at about 45 degrees. The nerve and the vascular structures were intact. No evidence of injury caused by trauma was noted.

Huda was transferred to UC Medical Center and a second X-ray exam of her left arm was performed at 1117 on July 14th. It showed that Huda had transverse mid shaft left humeral fracture, with one-half-width displacement of the distal fragment. The surrounding soft tissue was swollen. The fracture was reduced without surgical intervention and Huda's arm was placed in a sling and swathe to keep it immobilized.

A post-reduction X-ray exam of Huda's left humeral mid shaft fracture was performed at 1408 on July 14th. It showed satisfactory alignment with mild lateral angulation and mild surrounding soft tissue swelling.

It was alleged that Huda's left humeral fracture was caused by trauma. However, the clinical data in this case did not show evidence of external injury caused by trauma or injury to blood vessels and bleeding in the tissues surrounding the bone. The humerus is a relatively strong bone in 10-week-old infants and it is not medically possible to break this bone without the use of a significant external force that causes external and internal injuries and bleeding in the arm.

My review of the clinical data and the pertinent medical studies reveals that the synergistic actions of the following biological factors had led to Huda's left humerus fracture:

1) Huda suffered from vitamin K, vitamin D, and protein deficiency on or about June 23th and deficiency of these elements causes bone weakness and fractures. Huda's weight on June 7th and June 24th, 2008 was 4400 g and 4233 g, respectively. The expected weight for Huda on June 24th is 5102 g (Section 9).

Huda developed bruises on June 23rd and had a rib fracture which are signs of vitamin K deficiency. A blood analysis performed on July 14th showed that Huda had low levels of 25-hydroxy vitamin D, albumin, and total protein and mild anemia (Section 5).

2) Huda received 7 vaccines on July 7, 2008 that caused inflammation and systemic edema and the release of vitamin D, 1, 25-dihydroxy by inflammatory cells. Huda's serum level of vitamin D, 1, 25-dihydroxy on July 14th was 166 pg/mL (369% of average normal). Vitamin D, 1, 25-dihydroxy has known to stimulate osteoclasts and increases bone resorption (Section 10).

Huda's length on July 7th and 14th were 55.9 cm (50%) and 54.0 cm (25%), respectively. Her length decreased by 1.9 cm within 6 days. In addition, Huda's length did not increase between July 14th and July 24th. It is expected that her length to increase by 1.25 cm in 10 days based on her length increase rate of 3.75 cm/month occurred between July 22nd and August 7th (Section 6). These data indicate that Huda's skeletal growth was stopped and had bone resorption between July 7th and July 22nd.

3) Huda suffered from edema that led to her arm muscles to stretch and force the humerus to fracture at week points. Huda's weight on July 7th and July 13th was 4777 and 5220 g, respectively. She had gained 477 g (10% of her body weight) in 6 days. She gained weight at the rate of 79.5 g/day which is 309% of her weight gain rate of 25.7 g/day occurred during the 62 days prior to receiving vaccines on July 7th.

Huda was treated with morphine (0.6 mg IV), phenobarbital, and Tylenol at 0010-1330 on July 14th and her weight reduced to 5000 g at 1600 on July 14th. She had lost 220 g (4.2% of her body weight) within 16 hours. These data indicate that Huda's body retained fluid following vaccination in response to health problems and pain (Section 5).

10.1 Vitamin D, 1, 25-dihydroxy and bone resorption

Vitamin D3, 1,25-dihydroxy (calcitriol) is usually produced in the kidneys via 25-hydroxyvitamin D3 1- α -Hydroxylase by conversion from 25-hydroxycholecalciferol. It is essential in regulating a wide variety of biologic processes, such as calcium homeostasis, immune modulation, and cell proliferation and differentiation [36, 37].

Inflammatory cells also produce vitamin D3, 1,25-dihydroxy in certain pathological conditions to regulate the immune system and control inflammation. For example, Kruse *et al.* evaluated two infants with subcutaneous fat necrosis and had elevated blood level of 1,25-dihydroxyvitamin D and hypercalcemia. The granulomatous cells of fat necrosis produced unregulated level of 1,25-dihydroxyvitamin D that led to hypercalcemia that was normalized with glucocorticoid treatment [38].

In addition, Saggese *et al.* evaluated three cases of children with tuberculosis and hypercalcemia. Their serum levels of 1,25-dihydroxyvitamin D were elevated and returned to a normal range after three months of antituberculosis therapy. An ectopic and unregulated synthesis of 1,25-dihydroxyvitamin D by macrophages of granulomatous tissue was suggested [39].

Furthermore, Glass *et al.* evaluated 2 cases of men who had sarcoidosis, hypercalcemia, and elevated serum levels of 1,25-dihydroxy-vitamin. These men were treated with ketoconazole (600-800 mg/day) for four to six days and their serum 1,25-dihydroxyvitamin D level was reduced by approximately 40% [40].

Huda's serum level of vitamin D, 1, 25-dihydroxy on July 14th was 166 pg/mL, which is equal to 369% of average normal (Table 6). Vitamin D, 1, 25-dihydroxy has known to stimulate osteoclasts and increases bone resorption. For example, McSheehy and Chambers found that 1,25-dihydroxyvitamin D stimulated osteoclastic bone resorption in vivo and in organ culture. The osteoblastic cells induced a two- to four-fold stimulation of osteoclastic bone resorption in the presence of 1,25-

dihydroxyvitamin D. Stimulation was observed at concentrations of 10(-10) M and above [41].

In addition, Staal *et al.* examined the effects of 1,25-dihydroxyvitamin D in a mouse long bone culture model with respect to bone resorption. Bone resorption analyses showed that 1,25(OH)₂D stimulated bone resorption in dose-dependent manner [42].

Furthermore, Key *et al.* found that 1,25-dihydroxyvitamin D (calcitriol) stimulated a human osteosarcoma cell line, U2-OS, to produce a factor(s), which stimulated bone degradation in human monocyte cultures and osteoclastic bone resorption in fetal rat long bone cultures. The factor(s) was elicited by as little as 10(-10) M calcitriol. The factor is effective in stimulating peripheral blood monocytes to degrade bone, suggesting a direct effect on cellular bone breakdown [43].

Bowden *et al.* obtained data on serum 25-hydroxyvitamin D, 1,25 dihydroxyvitamin D, parathyroid hormone, and other bone markers, as well as bone mineral density for 85 children with primary osteoporosis and secondary osteopenia or osteoporosis caused by various underlying chronic illnesses. Pearson's correlation was used to assess the relationship between vitamin D levels and different bone parameters.

Vitamin D insufficiency (defined as serum 25-hydroxyvitamin D <30 ng/mL) was observed in 80.0% of the children. There was a significant inverse correlation between 25-hydroxyvitamin D and parathyroid hormone levels. They also found a positive correlation between 1,25 dihydroxyvitamin D and parathyroid hormone, alkaline phosphatase, and urine markers for bone turnover [44].

10.2 Reported adverse reactions to vaccines given to Huda

Huda was vaccinated at the age of 67 days with DTaP; HIB; PNUcon; IPV (oral) and Rotaviru. She developed systemic edema, skeletal growth retardation, bone resorption, and acute humeral mid shaft fracture within 6 days following vaccination. Huda was suffering from subdural hygromas when she was vaccinated.

Vaccines should not be given to sick children. It has been reported that sick children have failed to respond adequately to vaccines as compared to healthy children. For example, Krober *et al.* examined 47 infants with colds and 51 well infants at the age of 15 to 18 months, who received the standard measles-mumps-rubella (MMR) vaccine, for their response to develop the measles antibody [45].

Pre-vaccination serum samples were obtained prior to vaccine administration and post-vaccination serum samples were obtained 6 to 8 weeks later. Measles antibody was measured in these serum samples by an indirect fluorescein-tagged antibody test. Ten (21%) of 47 infants with colds failed to develop the measles antibody, while only one (2%) of 51 well infants failed to develop an antibody [45].

Vaccines given to Huda contain various antigens, heavy metals, antibiotics, and preservatives [46-50]. Additive and synergistic actions among these components in causing serious health problems can occur even in healthy children and adults. I have evaluated cases of infants and a toddler who died as a result of adverse reactions to vaccines [48; 49; 51; 52].

I have also evaluated cases of children and adult who developed serious health problems from vaccines [17; 53-56]. One of these cases was a two months old infant who received 7 vaccines while he was ill and suffered from developmental delay, anemia, subdural bleeding, and femoral abnormalities. His head circumference (HC) was 38.7 cm on the day of vaccination and it reduced to 37.3 cm at 32 days post vaccination. The baby's HC growth rate during the 2 months prior to vaccination was 2.8 cm/month [54].

In addition, I evaluated a case of triplets who were vaccinated with DTaP, IPV, Hib, and PCV vaccines at the age of 2-2.5 months and developed serious illnesses at two weeks following vaccination. The first baby (female) suffered from apnea, metabolic acidosis, seizure, infection, intracranial and retinal bleeding, and skull fracture. The second baby (male) developed respiratory tract and eye infection, severe anemia, bleeding, and skull fracture. The third baby (male) had severe anemia and skull fracture [17].

Serious illnesses and death have been reported in children who received fewer vaccines than Huda. For example, reports sent to VAERS, concerning infant immunization against pertussis between January 1, 1995 and June 30, 1998 were analyzed. During the study period, there were 285 reports involving death, 971 non-fatal serious reports (defined as events involving initial hospitalization, prolongation of hospitalization, life-threatening illness, or permanent disability), and 4,514 less serious reports after immunization with any pertussis-containing vaccine [57].

In addition, Wise *et al.* evaluated 4154 reports of events occurring after vaccination with 7-valent pneumococcal conjugate vaccine (PCV) in the United States during the first two years after licensure of PCV (February 2000 through February 2002). Reports studied were for children younger than 18 years and vaccinated with PCV. These reports were obtained from the Vaccine Adverse Event Reporting System (VAERS) database [58].

The most frequently reported symptoms and signs included fever, injection site reactions, fussiness, rashes, and urticaria. Serious events were described in 14.6% of reports. There were 117 deaths, 23 reports of positive rechallenges, and 34 cases of invasive pneumococcal infections possibly representing vaccine failure. Immune-mediated events occurred in 31.3% of reports. Thrombocytopenia developed in 14 children, serum sickness in 6 children, and 14 children suffered from anaphylactic or anaphylactoid reactions. Neurologic symptoms occurred in 38% of reports. Seizures described in 393 reports included 94 febrile seizures [58].

Furthermore, Niu *et al.* evaluated reports of neonatal deaths (aged 0-28 days) after hepatitis B (Hep B) immunization reported to the National Vaccine Adverse Event Reporting System (VAERS) January 1, 1991, through October 5, 1998. They identified 18 deaths (8 boys, 9 girls, 1 unclassified). The mean birth weight of the neonates ($n = 15$) was 3034 g (range, 1828-4678 g). The mean age of the infants at vaccination was 12 days. The median time from vaccination to onset of symptoms was 2 days and the median time from symptoms to death was 0 days (range, 0-15 days). The causes of death for the 15 autopsied cases were sudden infant death syndrome for 12 and infection for 3 [59].

11. Conclusions

The clinical data and medical studies presented in this report indicate the following:

1) The likely source of Huda's subdural hygroma is subdural bleeding, which occurred following birth. Huda was born at 40 weeks gestation by vacuum assisted Caesarian-section. Her head circumference (HC) following birth was 34.9 cm (50 percentile) and increased to 37 cm (95% percentile) within 5 days. The expected increase in her HC within 5 days is about 0.5 cm. The likely cause of the abnormal increase in Huda's HC is subdural bleeding caused by the vacuum assisted delivery.

In addition, Huda showed signs of vitamin K deficiency on June 23rd and vitamin K deficiency has caused subdural bleeding in infants. Huda's health problems were not discovered because no head CT scan, MRI or ultrasound exam was performed between May 7th and July 13th, 2008 to determine the cause(s) of the abnormal increase in Huda's HC.

2) The likely causes of Huda's bruises observed on her back June 23rd and 24th, 2008 and her rib fracture is vitamin K deficiency. In addition, vitamin D and protein deficiency also contributed to her rib fractures. Huda had lost 167 g between June 7th and June 24th and it is expected that she gained 702 g during that period. No clinical tests were performed to check for blood clotting problems and/or vitamin K deficiency.

3) The likely cause of Huda's minor retinal hemorrhage is the increased intracranial pressure.

4) Huda's mid shaft humerus fracture was caused by the synergistic actions of the biological factors:

a) Huda suffered from vitamin K, vitamin D, and protein deficiency on or about June 23th and deficiency of these elements causes bone weakness and fractures. In addition, a blood analysis performed on July 14th showed that Huda had low levels of 25-hydroxy vitamin D, albumin, and total protein and mild anemia.

b) Huda received 7 vaccines on July 7, 2008 that caused inflammation and systemic edema and the release of vitamin D, 1, 25-dihydroxy by inflammatory cells. Huda's serum level of vitamin D, 1, 25-dihydroxy on July 14th was 166 pg/mL (369% of average normal). Vitamin D, 1, 25-dihydroxy has known to stimulate osteoclasts and increases bone resorption.

Huda's length decreased by 1.9 cm between July 7th and 13th. In addition, her length did not increase between July 14th and July 24th. It is expected that her length to increase by 1.25 cm in 10 days. These data indicate that Huda's skeletal growth was stopped and had bone resorption between July 7th and July 22nd.

c) Huda suffered from edema that led to her arm muscles to stretch and force the humerus to fracture at weak points. Huda's weight on July 7th and July 13th was 4777 and 5220

g, respectively. She had gained 477 g (10% of her body weight) in 6 days. She gained weight at the rate of 79.5 g/day which is 309% of her weight gain rate of 25.7 g/day that occurred during the 62 days prior to receiving vaccines on July 7th.

Huda was treated with morphine, phenobarbital, and Tylenol on July 14th and had lost 220 g (4.2% of her body weight) within 16 hours. These data indicate that Huda's body retained fluid following vaccination in response to health problems and pain.

4) The factual causes of Huda's injuries and the clinical data that explain the mechanisms of her injuries were overlooked by the treating physicians.

References

- [1] Huda Sharif's medical records from Methodist Hospital (May 2nd -July 13th, 2008). 7500 Hospital Way. Sacramento, CA 95823.
- [2] Huda Sharif's medical records from Ringwald Medical Corporation, Inc. for the period of May 14th -September 22nd, 2008. 77 Cadillac Dr., Ste. 200. Sacramento, CA 95825.
- [3] Coulter, K. Report on Huda Sharif's case, November 10, 2008. Department of Pediatrics, UC Davis Medical Center. 2521 Stockton blvd, Sacramento 95817.
- [4] Rosas, A. Report on the case of Huda Sharif. November 10, 2008. Medical Group of Sacramento, INC. Sacramento, CA.
- [5] Court transcripts in the case of baby Sharif (December 15 and 17, 2008: pages 7-155). Superior Court of California in County of Sacramento, Sacramento, CA 95826.
- [6] Brandt I. Distance and velocity standards for head circumference growth before and after term until 18 months--results from a longitudinal study. *Monatsschr Kinderheilkd.* 1976 Apr;124(4):141–50.
- [7] Huda Sharif's medical records from UC Davis Medical Center (July 14th -September 22nd, 2008) 2521 Stockton blvd, Sacramento 95817.
- [8] Simonson C, Barlow P, Dehennin N, Sphel M, Toppet V, Murillo D, Rozenberg S. Neonatal complications of vacuum-assisted delivery. *Obstet Gynecol.* 2007 Mar;109(3):626–33.
- [9] Castillo M, Fordham LA. MR of neurologically symptomatic newborns after vacuum extraction delivery. *AJNR Am J Neuroradiol.* 1995 Apr;16(4 Suppl):816–8.
- [10] Whitby EH, Griffiths PD, Rutter S, Smith MF, Sprigg A, Ohadike P, Davies NP, Rigby AS, Paley MN. Frequency and natural history of subdural haemorrhages in babies and relation to obstetric factors. *Lancet.* 2004 Mar 13;363(9412):846–51.
- [11] Clark SL, Vines VL, Belfort MA. Fetal injury associated with routine vacuum use during cesarean delivery. *Am J Obstet Gynecol.* 2008 Apr;198(4):e4. Epub 2008 Mar 4.
- [12] Alexander JM, Leveno KJ, Hauth J, Landon MB, Thom E, Spong CY, Varner MW, Moawad AH, Caritis SN, Harper M, Wapner RJ, Sorokin Y, Miodovnik M, O'Sullivan MJ, Sibai BM, Langer O, Gabbe SG; National Institute of Child Health and Human Development Maternal-Fetal Medicine Units Network. Fetal injury associated with cesarean delivery. *Obstet Gynecol.* 2006 Oct;108(4):885–90.
- [13] Baume S, Cheret A, Creveuil C, Vardon D, Herlicoviez M, Dreyfus M. Complications of vacuum extractor deliveries. *J Gynecol Obstet Biol Reprod (Paris).* 2004 Jun;33(4):304–11.
- [14] Lahat E, Schiffer J, Heyman E, Dolphin Z, Starinski R. Acute subdural hemorrhage: uncommon complication of vacuum extraction delivery. *Eur J Obstet Gynecol Reprod Biol.* 1987 Jul;25(3):255–8.
- [15] The Merck Manual of Diagnosis and Therapy. Editors Beets MH and Berkow R, Seventeenth edition, 1999. Published by Merck Research Laboratories, Whitehouse Station, N.J.
- [16] Al-Bayati MA. Analysis of causes that led to bleeding, cardiac arrest, and death in the case of baby Nadine. *Medical Veritas.* 206 Nov;3(2):997–1012.
- [17] Al-Bayati MA. Analysis of causes that led to rib and skull fractures and intracranial bleeding in the case of the premature triplets Parneet, Sukhsaihaj, and Imaan. *Medical Veritas.* 2008 Apr;5(1):1589–609.
- [18] Nishio T, Nohara R, Aoki S, Sai HS, Izumi H, Miyoshi K, Morikawa Y, Mizuta R. Intracranial hemorrhage in infancy due to vitamin K deficiency: report of a case with multiple intracerebral hematomas with ring-like high density figures. *No To Shinkei.* 1987 Jan;39(1):65–70.
- [19] Bhat RV, Deshmukh CT. A study of Vitamin K status in children on prolonged antibiotic therapy. *Indian Pediatr.* 2003 Jan;40(1):36–40.
- [20] Sunakawa K, Akita H, Iwata S, Sato Y. Clinical superinfection and its attendant symptomatic changes in pediatrics. *Infection.* 1985;13(Suppl 1):S103–11.
- [21] de Montalembert M, Lenoir G, Saint-Raymond A, Rey J, Lefrere JJ. Increased PIVKA-II concentrations in patients with cystic fibrosis. *J Clin Pathol.* 1992 Feb;45(2):180–1.
- [22] Pooni PA, Singh D, Singh H, Jain BK. Intracranial hemorrhage in late hemorrhagic disease of the newborn. *Indian Pediatr.* 2003 Mar;40(3):243–8.
- [23] Choo KE, Tan KK, Chuah SP, Ariffin WA, Gururaj A. Haemorrhagic disease in newborn and older infants: a study in hospitalized children in Kelantan, Malaysia. *Ann Trop Paediatr.* 1994;14(3): 231–7.
- [24] Doneray H, Tan H, Buyukavci M, Karakelleoglu C. Late vitamin K deficiency bleeding: 16 cases reviewed. *Blood Coagul Fibrinolysis.* 2007 Sep;18(6):529–30.
- [25] Querfurth HW, Arms SW, Lichy CM, Irwin WT, Steiner T. Prediction of intracranial pressure from noninvasive transocular venous and arterial hemodynamic measurements: a pilot study. *Neurocrit Care.* 2004;1(2):183–94.
- [26] Medele RJ, Stummer W, Mueller AJ, Steiger HJ, Reulen HJ. Terson's syndrome in subarachnoid hemorrhage and severe brain injury accompanied by acutely raised intracranial pressure. *J Neurosurg.* 1998 May;88(5):851–4.
- [27] Stiebel-Kalish H, Turtel LS, Kupersmith MJ. The natural history of non-traumatic subarachnoid hemorrhage-related intraocular hemorrhages. *Retina.* 2004 Feb;24(1):36–40.
- [28] Al-Bayati MA. Analysis of causes that led to subdural bleeding and rib fractures in the case of baby Patrick Gorman. *Medical Veritas.* 2006 Nov;3(2): 1019–40.
- [29] Al-Bayati MA. Analysis of causes that led to subdural bleeding, skull and rib fractures, and death in the case of baby Aerial Buie. *Medical Veritas.* 2007 Nov;4(2):1452–69.
- [30] Tanaka K, Nakanishi Y, Kido S. Role of nutrition in the treatment of osteoporosis. *Clin Calcium.* 2005 Apr; 15(4):666–72.
- [31] Bugel S. Vitamin K and bone health. *Proc Nutr Soc.* 2003 Nov;62(4):839–43.
- [32] Iwamoto J, Takeda T, Sato Y. Effects of vitamin K2 on osteoporosis. *Curr Pharm Des.* 2004; 10(21):2557–76.
- [33] Shiraki M, Shiraki Y, Aoki C, Miura M. Vitamin K2 (menatetrenone) effectively prevents fractures and sustains lumbar bone mineral density in osteoporosis. *J Bone Miner Res.* 2000 Mar; 15(3):515–21.
- [34] Booth SL, Tucker KL, Chen H, Hannan MT, Gagnon DR, Cupples LA, Wilson PW, Ordovas J, Schaefer EJ, Dawson-Hughes B, Kiel DP. Dietary vitamin K intakes are associated with hip fracture but not with bone mineral density in elderly men and women. *Am J Clin Nutr.* 2000 May; 71(5):1201–8.
- [35] Rizzoli R, Ammann P, Chevalley T, Bonjour JP. Protein intake and bone disorders in the elderly. *Joint Bone Spine.* 2001 Oct;68(5):383–92.
- [36] Tan X, Li Y, Liu Y. Therapeutic role and potential mechanisms of active Vitamin D in renal interstitial fibrosis. *J Steroid Biochem Mol Biol.* 2007 Mar;103(3-5):491–6. Epub 2007 Jan 5.
- [37] Müller K, Bendtzen K. 1,25-Dihydroxyvitamin D3 as a natural regulator of human immune functions. *J Investig Dermatol Symp Proc.* 1996 Apr;1(1):68–71.
- [38] Kruse K, Irle U, Uhlig R. Elevated 1,25-dihydroxyvitamin D serum concentrations in infants with subcutaneous fat necrosis. *J Pediatr.* 1993 Mar;122(3):460–3.
- [39] Saggese G, Bertelloni S, Baroncelli GI, Fusaro C, Gualtieri M. Abnormal synthesis of 1,25-dihydroxyvitamin D and hypercalcemia in children with Tuberculosis. *Pediatr Med Chir.* 1989 Sep-Oct;11(5):529–32.
- [40] Glass AR, Cerletty JM, Elliott W, Lemann J Jr, Gray RW, Eil C. Ketoconazole reduces elevated serum levels of 1,25-dihydroxyvitamin D in hypercalcemic sarcoidosis. *J Endocrinol Invest.* 1990 May;13(5):407–13.

- [41] McSheehy PM, Chambers TJ. 1,25-Dihydroxyvitamin D3 stimulates rat osteoblastic cells to release a soluble factor that increases osteoclastic bone resorption. *J Clin Invest*. 1987 Aug; 80(2):425–9.
- [42] Staal A, Geertsma-Kleinekoort WM, Van Den Bemd GJ, Buurman CJ, Birkenhäger JC, Pols HA, Van Leeuwen JP. Regulation of osteocalcin production and bone resorption by 1,25-dihydroxyvitamin D3 in mouse long bones: interaction with the bone-derived growth factors TGF-beta and IGF-I. *J Bone Miner Res*. 1998 Jan;13(1):36–43.
- [43] Key LL Jr, Weichselbaum RR, Carnes DL Jr. A link between calcitriol and bone resorption. *Bone Miner*. 1988 Jan;3(3):201–9.
- [44] Bowden SA, Robinson RF, Carr R, Mahan JD. Prevalence of vitamin D deficiency and insufficiency in children with osteopenia or osteoporosis referred to a pediatric metabolic bone clinic. *Pediatrics*. 2008 Jun;121(6):e1585–90.
- [45] Krober MS, Stracener CE, Bass JW. Decreased measles antibody response after measles-mumps-rubella vaccine in infants with colds. *JAMA*, 1991; 265(16):2095–6.
- [46] Physicians' Desk Reference, Edition 53, 1999. Medical Economics Company, Inc, Montavale, NJ, USA.
- [47] Physicians' Desk Reference, Edition 57, 2003. Thomson PDR, Montavale, NJ, USA.
- [48] Al-Bayati MA. Analysis of causes that led to Toddler Alexa Shearer's cardiac arrest and death in November 1999. *Medical Veritas*, 2004 Apr; 1(1):86–117.
- [49] Al-Bayati MA. Analysis of causes that led to baby Alan Ream Yurko's cardiac arrest and death in November of 1997. *Medical Veritas*, 2004 Apr; 1(2):201–31.
- [50] Duclos P. Safety of immunisation and adverse events following vaccination against hepatitis B. *Expert Opin Drug Saf*. 2003 May;2(3):225–31.
- [51] Al-Bayati MA. Analysis of Causes That Led to Baby Lucas Alejandro Mullenax-Mendez's Cardiac Arrest and Death in August-September of 2002. *Medical Veritas*, 2004 Apr;1(1):45–63.
- [52] Analysis of causes that led to Evyn Vaughn's respiratory arrest, intracranial and retinal bleeding, and death. *Medical Veritas*, 2009 Apr;6(1):1937–58.
- [53] Al-Bayati MA. Analysis of causes that led to baby Ryan's hemorrhagic pneumonia, cardiac arrest, intracranial bleeding, and retinal bleeding. *Medical Veritas*, 2008 Nov; 5(2), 1757–74.
- [54] Al-Bayati MA. Analysis of causes that led to baby Jackie Ray's developmental delay and intracranial bleeding. *Medical Veritas*, 2008, Nov;5(2):1836–44.
- [55] Al-Bayati MA. Analysis of causes that led to the development of vitiligo in Jeanett's case with recommendations for clinical tests and treatments. *Medical Veritas*, 2007 Apr;4(1):1251–62.
- [56] Al-Bayati MA. Severe hair loss induced by vaccines and reversed by the treatment with zinc. *Medical Veritas*, 2004 Apr;1(2):159–62.
- [57] Braun MM, Mootrey GT, Salive ME, Chen RT, and Ellenberg SS. Infant immunization with acellular pertussis vaccines in the United States: assessment of the first two years' data from the Vaccine Adverse Event Reporting System (VAERS). *Pediatrics*, 2000; 106(4):E51.
- [58] ise RP, Iskander J, Pratt RD, Campbell S, Ball R, Pless RP, Braun MM. Postlicensure safety surveillance for 7-valent pneumococcal conjugate vaccine. *JAMA*. 2004 Oct 13;292(14):1702–10.
- [59] Niu MT, Salive ME, Ellenberg SS. Neonatal deaths after hepatitis B vaccine: the vaccine adverse event reporting system, 1991–1998. *Arch Pediatr Adolesc Med*. 1999 Dec;153(12):1279–82.