Analysis of causes that led to rib and skull fractures and intracranial bleeding in the case of the premature triplets Parneet, Sukhsaihaj, and Imaan

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Abstract

Parneet, Sukhsaihaj, and Imaan were born at 30 weeks gestation via cesarean section and their weights were 1.31, 1.38, and 1.46 Kg, respectively. They suffered from respiratory acidosis, hypoglycemia, respiratory distress syndrome, sepsis, jaundice, gastroesophageal reflex, and anemia. They were discharged from the hospital at 42 days of age. The triplets were vaccinated with DTaP, IPV, Hib, and PCV vaccines at the age of 61 days in Imaan's case and 75 days in Parneet and Sukhsaihaj's case. At 15 days following vaccination, Parneet suffered from apnea, metabolic acidosis, seizures, infections, intracranial and retinal bleeding, and a skull fracture. Sukhsaihaj developed respiratory tract and eye infections, severe anemia, bleeding, and skull fracture. Imaan had severe anemia and a skull fracture. It was alleged that the babies' injuries were caused by blunt trauma to the head. The parents were accused of causing the triplet's injuries.

The clinical data described in this report reveal that the triplet's health problems were caused by vaccines. The severity of their injuries correlates with their hemoglobin levels at the time of vaccination. The subcutaneous and the intracranial bleeding resulted from protein and vitamin K deficiencies and infections. Severe anemia, vitamin K deficiency, and systemic infections are the likely causes of Parneet's retinal bleeding. The skull fractures resulted from protein and vitamin K deficiencies. The severity of the fractures correlates with the severity of the protein and vitamin K deficiencies, the degree of the infection, and the level of the intracranial pressure.

Keywords: anemia; apnea; blunt trauma; child abuse; DTaP; gastroesophageal reflex; *Haemophilus influenzae* type B; Hib; hypoglycemia; inactivated polio vaccine; IPV; intracranial bleeding; Jaundice respiratory acidosis; rib fracture; respiratory distress syndrome; pneumococcal conjugate vaccine; PCV; sepsis; subdural bleeding; triplets.

1. Summary of the case and findings

Parneet, Sukhsaihaj, and Imaan are premature triplets from Ontario, Canada. They were born at 30 weeks gestation via cesarean section due to pregnancy complications. One day prior to delivery (June 28, 2007), one of the triplets was noted to have an absent placental and diastolic blood flow and intrauterine growth retardation.

Their mother suffered from a bacterial infection and anemia at 40 days prior to delivery and was treated with antibiotics and ferrous sulfate. She was hospitalized at 24 weeks gestation and was treated with betamethasone at 24 and 28 weeks gestation.

Parneet, Sukhsaihaj, and Imaan's birth weights were 1.46, 1.31, and 1.38 Kg, respectively. The triples had poor respiratory efforts and they were intubated and placed on a mechanical ventilator following birth. Each infant was given 1 dose of Bless pulmonary surfactant. Blood analysis performed following birth showed the triplets were suffering from respiratory acidosis and hypoglycemia. They had blood pH of 7.23-7.28 (normal range: 7.33-7.49) and blood glucose level of 1.7-2.7 mmol/L (normal range: 3.3-5.6).

The triplets stayed in the newborn intensive care unit (NICU) for 42 days following birth. They suffered from respiratory distress syndrome, sepsis, jaundice, gastroesophageal reflex (GER) and anemia. They were treated with antibiotics, phototherapy, and Ranitidine. They were also given red blood cell transfusions prior to discharge from the hospital on Aug. 10th due to severe anemia.

Imaan was vaccinated with six vaccines on Aug. 29th. He was 61 days old (39 weeks post-menstrual age). These vaccines include diphtheria-tetanus toxoids-acellular pertussis (DTaP); inactivated polio vaccine (IPV); Haemophilus influenzae type B (Hib); and pneumococcal conjugate vaccine (PCV). Parneet and Sukhsaihaj were vaccinated with these six vaccines on September 12th at 75 days of age (41 weeks post-menstrual age).

On Sept. 27, 2007, Parneet vomited the majority of her food after she was bottle-fed at approximately 1800. Approximately one hour later, she was noted to be apenic, cyanotic and not responsive. The parents called the Emergency Medical Services (EMS). The EMS transported Parneet by ambulance to the emergency room (ER) at Windsor Regional Hospital (WRH).

Upon arrival at the ER, Parneet had a heart rate of 80/minute and her temperature was 34^{o}C. She appeared pale and had shallow respiration on oxygen but her blood was well saturated. Her respiratory rate was 40-50/minute. The paramedics and the physician examined Parneet at the ER and observed no evidence of injuries caused by trauma.

Parneet was admitted to the Neonate Intensive Care Unit (NICU). Blood analysis showed that she was suffering from metabolic acidosis, hyperglycemia, and severe anemia. She had blood pH of 7.29, glucose level of 14.3 mmol/L, and hemoglobin level of 6.8 g/dL.

She was treated with fluids and sodium bicarbonate and given a red blood cell transfusion.

In addition, she had an elevated white blood cell count (WBC) of 22.1 x 10^3/µL and a lymphocyte count of 13.9 x 10^3/µL. She was treated with fluconazole IV and her WBC and lymphocyte count went down to a normal level following this treatment.

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Parneet developed twitching in her right arm and leg. Her CT scan and an MRI exam of the head showed subcutaneous hematoma and intracranial bleeding. An X-ray exam of the head revealed a comminuted fracture of the left posterior parietal and occipital region.

An eye exam showed retinal bleeding in the right eye. She continued to have seizures until Oct. 7th. She was treated with Dilantin, phenobarbitol, and chloral hydrate.

Sukhsaihaj and Imaan were admitted at WRH on Sept. 28, 2007 because of concerns about child abuse that resulted from Parneet's hospitalization on Sept. 27th. The MRI exam of Sukhsaihaj's head revealed the presence of a subcutaneous hematoma over the left parieto-occipital region and a small contusion in the brain. An X-ray exam of his head showed evidence of a left parietal fracture.

A CT scan of Imaan's head revealed the presence of a skull fracture involving the right parietal bone and a brain contusion with hemorrhage involving the right parietal lobe. The nurse called the Children's Aid Society (CAS) and the CAS came with police and took custody of the babies. The babies were discharged from the hospital on Oct. 9th and the parents had contact with them only under supervision.

Dr. D.W. Warren examined the triplets at the Children's Hospital of Western Ontario on Oct. 11, 2007. He also reviewed their medical records from Windsor Regional Hospital. Warren alleged that the bleeding, brain lesions, and the skull fractures observed in the case of the triplets were caused by blunt trauma to the head.

He stated that the force used in the triplet's case is consistent with a fall from a changing table on to a wood or tile floor in Imaan's case; a fall from a greater height, possibly from the top of a stairs in Sukhsaihaj's case; and a fall from a height of 3 to 5 floors, on to a hard surface in Parneet's case.

The triplet's parents were accused of abusing the triplets and causing their injuries. The triplet's mother contacted me and requested that I evaluate the medical evidence in the triplet's case to find the likely causes that led to their serious illnesses. I am a toxicologist and pathologist with over 20 years experience in these fields. I have published over 45 articles in medical and scientific journals.

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

I evaluated the following medical records and the articles cited in this report using differential diagnosis: (1) The triplet's prenatal medical records; (2) the triplet's medical records from birth to the time of their release from the hospital on Oct. 9, 2007; (3) published medical articles on the adverse reactions to vaccines given to the triplets; (4) medical articles pertinent to the triplet's illnesses; (5) Warren's report on the triplet's case issued on Oct. 25, 2007.

Approximately 300 hours were required to evaluate the medical evidence, perform an analysis, and write this report. My findings in this case include:

1) The triplet's health problems were caused by the 6 vaccines given to each infant (Section 5). Parneet's spitting and vomiting of milk became more severe after her vaccination on Sept. 12th. She also developed loose stools, appeared sleepy and irritable, and experienced episodes of vomiting during the week prior to her hospitalization on Sept. 27th.

Parneet was suffering from severe anemia and systemic infection at the time of admission to the hospital on Sept. 27th. She lost an average of 3 g/day during the first three days following admission. Her weight gain rate for the period after vaccination (21.3 g/day) was 26.3% less than her weight gain rate for the period prior to vaccination (28.8 g/day).

Sukhsaihaj had nasal congestion during the 11 days prior to his admission to the hospital on Sept. 28th. He had nasal congestion and his face was swollen at the time of admission. His right eye had a yellow green discharge, bleeding under the cornea, conjunctival hemorrhage, and retinal bleeding. He was treated with polysporin eye drops for 9 days. He also suffered from severe anemia. He did not gain weight during the first five days after admission. His weight gain rate for the period after vaccination was 20.5% less than his weight gain rate for the time prior to vaccination.

Imaan was also suffering from severe anemia at the time of admission. He had a hemoglobin level of 8.5 g/dL and hematocrit value of 24.5%. He lost an average of 8 g per day during the first three days after admission.

2) The severity of the injuries and the infections observed in the triplet's case correlates with their hemoglobin levels at the time of vaccination. Imaan was vaccinated on Aug. 29th and Sukhsaihaj and Parneet were vaccinated on Sept. 12th. Imaan did not suffer from infection following vaccination because he had a significantly higher blood hemoglobin level than his siblings.

The expected blood hemoglobin levels at the time of vaccination in Parneet, Sukhsaihaj, and Imaan's case were 8.5 g/dL, 10.2 g/dL, and 11.4 g/dL, respectively. The hemoglobin levels of these babies were 12.9-14.4 g/dL on Aug. 4th because they received red blood cell transfusions. However, their hemoglobin levels were reduced at the rate of 0.1 g/dL per day for the time between Aug. 4th and Sept. 27th.

3) The subcutaneous and intracranial bleeding observed in the triplet's case resulted from protein and vitamin K deficiencies and infection. The severity of the bleeding correlates with the severity of the protein and vitamin K deficiencies and the degree of the infection.

The severity of bleeding in Parneet's case was greater than in Sukhsaihaj and Imaan's case. Her weight gain rate during the first 91 days following birth was 26.8 g/day, which was less than those of her siblings (30.6-39.6 g/day). She also suffered from systemic infections. The severity of the bleeding in Sukhsaihaj's case is greater than Imaan's case because Sukhsaihaj suffered from infections.

Babies suffering from vitamin K deficiency have also developed intracranial bleeding and bleeding in other locations. For example, Chaou *et al.* reported late-onset intracranial hemorrhage related to vitamin K deficiency in 32 infants (1/2 to 6 months of age). Computerized tomography showed mild to severe intracranial hemorrhage. Most (90.6%) had subarachnoid

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hemorrhage, either alone or in combination with subdural hemorrhage, parenchymal hemorrhage, or intraventricular hemorrhage (Section 9).

4) Severe anemia, vitamin K deficiency, and systemic infections are the likely causes of Parneet's retinal bleeding. At the time of admission, she had hemoglobin levels of 6.8 g/dL and hematocrit of 19.4%, respectively. Asien *et al.* evaluated the occurrence of clinically apparent retinal changes in 35 anemic individuals. Seven (20%) of the anemic individuals exhibited extra vascular lesions (flame-shaped hemorrhages, hard exudates, and cotton-wool spots). The mean hemtocrit reading for these individuals was 24.7% (see Section 10).

5) The bone fractures observed in the triplet's case resulted from protein and vitamin K deficiencies. The severity of their skull fractures correlates with the severity of their protein and vitamin K deficiencies, and the degree of the infections, and the levels of the intracranial pressure.

The intracranial bleeding and the edema in Parneet's case were more severe than those observed in Imman and Sukhsaihaj's case. Her head circumference on Oct. 1st was 38.5 cm, which is 2 cm more than the expected. She was treated with mannitol and her head circumference decreased to 37 cm on Oct. 4th (see Section 11).

6) Warren's allegations that the triplet's injuries were caused by blunt trauma to the head and child abuse are not medically valid based on the following observations:

a) Warren alleged that the forces used to cause the skull fracture s in these infants are equivalent to a fall from a changing table on to a wood or tile floor in Imaan's case, the top of stairs on to a hard surface in Sukhsaihaj's case, and a height of 3 to 5 floors on to a hard surface in Parneet's case. However, the significant forces applied in these cases did not cause neck injuries or even a tiny scratch on the skin of the head in any infant. The triplets were examined in the hospital for 11 days and the treating physicians and nurses observed no evidence of external injury caused by trauma.

b) Warren reviewed Parneet, Sukhsaihaj, and Imaan's chest X-ray exams performed on Sept. 27-29th at Windsor Regional Hospital (WRH). He reported the presence of 9, 3, and 2 healed rib fractures in Parneet, Sukhsaihaj, and Imaan's X-rays, respectively. However, the radiologist(s) at WRH did not report the finding of any rib fractures in any of the infants. Warren's allegations require that radiologist(s) at WRH failed to identify 14 fractured ribs on the triplet's chest X-rays.

Furthermore, Warren reviewed Parneet, Sukhsaihaj, Imaan's chest X-rays performed on Oct. 11th at Children's Hospital. He reported the finding of additional healed rib fractures involving left 4th in Parneet's case and left and right 10th in Sukhsaihaj's case. Warren stated that the rib fractures in the infants resulted from excessive squeezing of the chest.

Warren also stated that the examination of Parneet's legs "revealed increased periosteal thickening of the right and left femurs. There is also evidence of a left tibial chip fracture. These are consistent with excessive force in grabbing the legs of this young infant." Parneet's X-rays taken at WRH in Sept. 27-29th did not show bone abnormalities in the femurs and the tibia (Section 12).

The triplets were taken into custody by the Children's Aid Society (CAS) on Sept. 28th and stayed in the hospital. The parents had contact with the babies only under supervision. If Warren's allegations are true then his observations raise the following issues:

1) Someone other than the parents allegedly squeezed the chests of these infants between Sept. 28th and Oct. 11th to cause the additional rib fractures identified by Warren on Oct. 11th .

2) Someone other than the parents allegedly squeezed Parneet's legs to cause the periosteal thickening of the femurs and the tibial chip fracture.

I believe that the following recommendations will help in monitoring the triplet's injuries and the healing process. They may also help in preventing the occurrence of similar vaccine related injuries in the future:

1) The clinical data show that the triplets were suffering from severe anemia and thrombocytosis due to bone marrow depression and hyperplasia. Blood analysis should be performed periodically to monitor for these illnesses.

2) Vaccine(s) should not be given to the infants while they are anemic and sick. Blood tests should be performed prior to giving vaccines to prematurely born children to check for anemia, liver problem, clotting problem, and immune depression. In addition, benefit and risk analysis should be performed prior to vaccination. Vaccines like other medicines, are capable of causing serious health problems in children. I believe that giving six vaccines in one single visit to a premature and sick infant is not a medically justified action.

2. Conditions of the triplets at birth and treatments given to the mother and infants

The triplets (Parneet, Sukhsaihaj, and Imaan) were born on June 29, 2007 at 30.2 weeks gestation via cesarean section. The mother is a 26-year-old G1ToPoA0Lo, B Rh+ woman. She was rubella immune, hep B negative, and Venereal Disease Research Laboratory (VDRL) test negative.

She suffered from a bacterial infection at 40 days prior to delivery and was treated with antibiotics for seven days. She was treated with ampicillin (500 mg po qid) and erythromycin (250 mg po qid). In addition, she suffered from anemia and was treated with ferrous sulfate (300 mg/day po). She was hospitalized at 24 weeks gestation and she was treated with betamethasone at 24 and 28 weeks gestation [1-4].

One day prior to delivery, one of the triplets was noted to have an absent placental and diastolic blood flow and intrauterine growth retardation. A caesarean section was performed under spinal anesthesia to deliver the triplets. Imaan was born with the umbilical cord tied around his neck x 1 [1-4]. The triplet's weights, lengths, head circumferences, and Apgar scores are presented in Table 1.

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Table 1. Triplet's birth weights, lengths, head circumferences and Apgar scores

Measurements	Parneet (Female)	Sukhsaihaj (Male)	Imaan (Male)
Weight (g) and percentile	1460 g (25^{th}-50^{th})	1310 g (25^{th}-50^{th})	1380 g (25^{th}-50^{th})
Length (cm) and percentile	43 cm (75^{th}-90^{th})	40 cm (25^{th}-50^{th})	40 cm (25^{th}-50^{th})
Head circumference and percentile	28 cm (50^{th})	29 cm (75^{th})	29 cm (75^{th})
Apgar score at 1 minute	9	9	6
Apgar score at 5 minutes	9	9	9

The triples had a poor respiratory effort and were intubated and placed on a mechanical ventilator following birth. Blood analysis performed following birth showed that the triplets were suffering from respiratory acidosis. They also had low glucose blood levels at birth. Their initial blood glucose levels were 1.7-2.7 mmol/L. They were given an intravenous glucose infusion that raised their glucose level to 4.97 mmol/L (Table 2).

Each infant was given 1 dose of Bless pulmonary surfactant. They were treated with erthyromycin opthamic ointment within two hours of birth. Five mg of erthyromycin ointment was instilled into each conjunctival sac. Furthermore, each infant was treated with phytonadione (vitamin K1) 1 mg IM within 6 hours after birth.

The triplets stayed in the newborn intensive care unit (NICU) for 42 days following birth. They suffered from respiratory distress syndrome, sepsis, jaundice, and anemia. They were treated with antibiotics and given red blood cell transfusions. They were discharged from the hospital on Aug. 10, 2007 [1-4].

Table 2. Triplet's blood gases and glucose levels following birth

Measurements	Parneet (Female)	Sukhsaihaj (Male)	Imaan (Male)	Reference range
PH	7.27	7.28	7.23	7.33-7.49
PCO_2 (mm Hg)	50	46	54	26-41
PO_2 (mm Hg)	150	212	167	65-76
HCO_3 (mmol/L)	31	20	20	16-25
Base excess (mmol/L)	-3.6	-4.8	-4.3	-2.0-2.0
Blood glucose (mmol/L)	1.7	2.7	1.7	3.3-5.6

3. Summary of the triplet's forty-two days hospitalization, clinical tests, illnesses, and treatments given

Parneet, Sukhsaihaj, and Imaan stayed in the newborn intensive care unit (NICU) for 42 days following birth. They were discharged from the hospital on Aug. 10, 2007. They suffered from respiratory distress syndrome, sepsis, and anemia. They were treated with antibiotics and given red blood cell transfusions. Below are the results of the clinical testes performed and their significance, treatments given, and the triplet's growth rates.

3.1 Respiratory distress syndrome

The triplets suffered from respiratory distress syndrome (RSD) following birth. They were intubated and each infant was given 1 dose of Bless pulmonary surfactant.

Parneet was intubated at 4 minutes of age and she was extubated to room air roughly at 21 hours of age. She received low flow at 25% at 0.5 liter per minute for 2 days. She was then weaned to room air by day 4 [2-5].

Sukhsaihaj was intubated at 4 minutes of age and he was extubated to room air by 10 hours of age. Imaan was intubated at 3 minutes of age and exubated to room air by 21 hours of age. He later required nasal continuous positive airway pressure (CPAP) up until day 2 with 30% oxygen alone. Blood analyses performed during the six days following birth showed the infants suffered from respiratory acidosis (Tables 3-5).

Table 3. Parneet's blood gases measured during the six days following birth

Date	PH	PCO_2 (mm Hg)	PO_2 (mm Hg)	HCO_3 mmol/L	Base Excess mmol/L
June 29 (1525)	7.27	50	150	31	-3.6
June 29 (1840)	7.36	38	122	21	-3.8
June 30	7.33	42	85	21	-3.6
July 1	7.26	49	36	20	-4.6
July 2	7.27	47	39	20	-4.7
July 3	7.25	49	44	19	-5.2
July 4	7.27	42	46	18	-7.1
Ref. range	7.33-7.49	26-41	65-76	16-25	-2.0-2.0

Table 4. Sukhsaihaj's blood gases measured during the three days following birth

Date	Time	PH	PCO_2 (mm Hg)	PO_2 (mm Hg)	HCO_3 (mmol/L)	Base Excess (mmol/L)
June 29	1531	7.28	46	212	20	-4.8
June 29	1843	7.29	49	90	21	-3.1
June 29	2350	7.35	41	102	22	-2.8
June 30	0700	7.33	41	79	21	-4.5
June 30	1400	7.31	40	64	20	-5.4
July 1	0715	7.26	53	41	20	-3.3
Ref. range		7.33-7.49	26-41	65-76	16-25	-2.0-2.0

Table 5. Imaan's blood gases measured during the six days following birth

Date	Time	PH	PCO_2 (mm Hg)	PO_2 (mm Hg)	HCO_3 (mmol/L)	Base Excess (mmol/L)
June 29	1521	7.23	54	167	20	-4.3
June 29	1839	7.29	48	114	21	-3.0
June 30	0009	7.30	46	84	21	-3.8
June 30	0700	7.36	34	63	20	-6.1
June 30	1400	7.32	39	57	20	-5.3
July 1	0715	7.23	59	38	20	-2.6
July 1	1335	7.22	56	40	19	-4.6
July 1	1938	7.22	57	41	19	-4.4
July 2	0730	7.22	54	41	19	-4.9
July 3	0731	7.25	48	42	18	-6.1
July 4	0820	7.25	44	43	18	-7.4
Ref. range		7.33-7.49	26-41	65-76	16-25	-2.0-2.0

3.2 Treatment with antibiotics

The infants started on ampicillin (130 mg IV q 24) and gentamicin (5.2 mg IV q24) following birth and antibiotics were discontinued at five days of age [2-4]. The treatment of infant

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with antibiotics for significant period of time increases the risk for vitamin K deficiency [6-9].

3.3 Jaundice

The triplets developed jaundice at 2 days following birth. Their total blood bilirubin reached significant levels of 191-228 µmol/L at 4 days after birth (Table 6). The triplet's mother is group B positive. Parnnet and Sukhsaihaj are also group B positive and Imaan is group AB positive. The three infants were Coombs negative. The infants were treated with phototherapy for six days [2-4].

Table 6. The triplet's blood bilirubin levels measured during the forty days following birth

	Parneet		Sukhsaihaj		Imaan	
Date	Total µmol/L	Conj. µmol/L	Total µmol/L	Conj. µmol/L	Total µmol/L	Conj. µmol/L
June 30	88	4	94	5	83	3
July 1	124	4	135	5	136	4
July 2	176	5	162	4	181	5
July 3	191	3	195	4	228	6
July 4	137	4	115	4	148	6
July 5	94	3	58	5	98	4
July 6	78	3	-	-	-	-
July 11	126	6	164	7	164	7
July 18	80	7	127	7	127	7
July 25	61	5	113	6	113	6
Aug. 1	74	5	101	5	101	5
Aug. 8	75	4	98	4	98	4
Reference range	0-120	0-10	0-120	0-10	0-120	0-10

* (-): Not measured

3.4 Anemia

The triplets had normal red blood cell counts, hemoglobin levels, and platelet counts at birth. However, their red blood cell counts and hemoglobin levels went down to low levels at 33 days of age. Their hemoglobin levels were reduced by 31-34% of their levels following birth. In addition, their platelet counts were increased to 2.6-3.40 times their initial values at birth as a result of bone marrow hyperplasia (Tables 7-9). These data indicate that the babies suffered from bone marrow depression. The infants were treated with ferrous sulfate orally.

The infants were also given packed red blood cell transfusions at 33-35 days of age. The hemoglobin levels were increased from 9.2 to 13.6 g/dL, 8.8 to 14.4 g/dL, 10.4-13.7 g/dL in Parneet, Sukhsaihaj, and Imaan's case, respectively. However, their hemoglobin levels were reduced by 5% in 4 days after being given red blood cell transfusions in Parneet and Imaan's case and 11% in Sukhsaihaj's case.

Table 7. Parneet's hematology values measured during the forty days after birth

Date	RBC x 10^6/µL	HGB (g/dL)	HCT %	Platelet x 10^3/µL
June 29	4.11	14.0	40.6	215
July 4	3.46	14.1	32.6	5.21
July 11	3.80	12.1	34.9	480
July 18	3.82	11.8	34.3	700
July 25	3.62	11.1	32.5	739
August 1	2.99	9.2	26.6	541
August 4	4.57	13.6	39.7	461
August 8	4.32	12.9	37.7	395
Reference range	3.5-5.0	11.5-15	35-54	140-400

Table 8. Sukhsaihaj's hematology values measured during the forty days after birth

Date	RBC x 10^6/µL	HGB (g/dL)	HCT %	Platelet x 10^3/µL
June 29	3.36	12.7	35.1	241
July 4	3.79	13.3	37.9	384
July 11	3.42	11.5	33.4	601
July 18	3.22	10.8	30.4	305
July 25	3.46	11.3	32.6	831
August 1	2.72	8.8	25.4	637
August 4	4.64	14.4	42.6	492
August 8	4.27	12.8	38.2	354
Reference range	3.5-5.0	11.5-15	35-54	140-400

Table 9. Imaan's hematology values measured during the forty days following birth

Date	RBC x 10^6/µL	HGB (g/dL)	HCT %	Platelet x 10^3/µL
June 29	4.60	15.8	45.0	151
July 4	5.30	17.1	50.4	251
July 11	5.33	16.9	49.3	422
July 18	4.47	13.9	40.4	472
July 25	4.07	12.5	36.4	503
August 1	3.46	10.4	30.4	407
August 4	4.63	13.7	40.3	308
August 8	4.51	13.1	39.2	277
Reference range	3.5-5.0	11.5-15	35-54	140-400

3.5 White blood cell and differential counts

The triplet's white blood cell and differential counts measured during the forty days following birth are presented in Tables 10-12. At the time of discharge from the hospital, Sukhsaihaj's neutrophil count was 40% below the low normal value. Parneet and Imaan had noutrophil counts slightly higher than the low normal value.

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Table 10. Parneet's white blood cell and differential counts measured during the forty days after birth

Date	WBC x10^3/µL	Neutro x10^3/µL	Lympho x10^3/µL	Mono x10^3/µL	Esino x10^3/µL	Baso x10^3/µL
June 29	8.1	2.7	4.3	0.7	0.4	0.0
July 4	19.2	8.8	6.1	2.5	1.7	-
July 11	13.8	5.8	3.4	2.4	0.4	-
July 18	23.3	9.0	8.3	4.2	1.6	0.3
July 25	18.6	3.1	10.1	1.7	2.8	1.0
Aug. 1	12.3	2.1	7.1	1.6	1.3	0.2
Aug. 4	11.3	2.2	6.4	1.3	1.3	0.1
Aug. 8	9.9	1.2	6.1	1.0	1.4	0.2
Reference range	5.0-20.0	1.0-9.5	2.0-17.0	0.2-2.4	0.1-1.0	0.0-0.2

Table 11. Sukhsaihaj's white blood cell and differential counts measured during the forty days after birth

Date	WBC x10^3/µL	Neutro x10^3/µL	Lympho x10^3/µL	Mono x10^3/µL	Esino x10^3/µL	Baso x10^3/µL
June 29	11.1	2.8	6.9	0.8	0.7	-
July 4	11.9	5.4	4.8	1.2	0.4	-
July 11	16.1	5.2	7.6	2.1	0.3	-
July 18	13.9	4.3	7.6	0.6	1.0	0.3
July 25	12.8	2.4	7.8	1.4	1.1	0.1
Aug. 1	10.5	2.1	6.7	1.0	0.6	0.2
Aug. 4	10.7	1.6	6.6	1.2	1.0	0.2
Aug. 8	9.1	0.6	7.0	0.8	0.4	-
Reference range	5.0-20.0	1.0-9.5	2.0-17.0	0.2-2.4	0.1-1.0	0.0-0.2

Table 12. Imaan's white blood cell and differential counts measured during the forty days after birth

Date	WBC x10^3/µL	Neutro x10^3/µL	Lympho x10^3/µL	Mono x10^3/µL	Esino x10^3/µL	Baso x10^3/µL
June 29	10.3	2.3	6.3	1.0	0.5	0.2
July 4	7.9	3.2	3.6	0.8	0.4	-
July 11	11.6	4.4	5.7	1.0	0.0	0.5
July 18	10.9	2.2	6.4	1.5	0.6	0.2
July 25	11.0	3.3	5.2	1.1	1.2	-
Aug. 1	9.6	1.6	5.9	0.8	1.1	0.2
Aug. 4	8.8	1.4	4.9	0.6	1.8	0.1
Aug. 8	8.2	1.6	4.3	0.9	1.2	-
Reference. range	5.0-20.0	1.0-9.5	2.0-17.0	0.2-2.4	0.1-1.0	0.0-0.2

3.6 Blood glucose levels

The triplet's blood glucose levels were low at birth and they were given intravenous glucose infusion. Parneet had the lowest blood glucose level after birth as compared with Sukhsaihaj and Imaan. Her blood glucose level was also lower than Sukhsaihaj and Imaan at forty days of age (Table 13)

Table 13. Triplet's blood glucose levels measured during the forty days after birth

Date	Parneet Glucose (mmol/L)	Sukhsaihaj Glucose (mmol/L)	Imaan Glucose (mmol/L)
June 29	1.7	2.7	2.7
June 30	7.4	7.5	7.3
July 1	4.3	5.9	4.5
July 2	4.2	7.3	4.9
July 3	4.7	5.6	4.6
July 4	3.2	5.8	5.0
July 5	3.7	4.0	4.5
July 11	3.9	4.2	4.0
July 18	3.1	4.8	3.4
July 25	3.5	3.7	3.1
August 1	3.7	4.7	3.1
August 8	3.4	4.6	4.0
Reference range	2.2-5.0	2.2-5.0	2.2-5.0

3.7 Blood creatinine and urea levels

The triplet's blood creatinine and urea levels decreased steadily during the forty days of hospitalization. However, their creatinine and urea levels stayed within the normal range (Table 14).

Table 14. Triplet's blood levels of creatinine and urea measured during the forty days after birth

Date	Parneet		Sukhsaihaj		Imaan	
	Creatinine[1]	Urea[2]	Creatinine[1]	Urea[2]	Creatinine[1]	Urea[2]
June 30	91	6.1	84	3.7	65	4.7
July 1	65	6.4	62	3.8	61	4.9
July 2	60	6.2	55	3.9	55	4.3
July 3	43	6.1	45	3.4	48	4.2
July 4	49	6.3	49	4.1	48	5.1
July 5	40	5.9	51	4.3	45	5.2
July 11	44	2.6	47	2.5	41	2.1
July 18	33	2.3	29	2.0	34	2.5
July 25	26	2.9	27	2.1	30	2.5
Aug. 1	24	2.3	25	1.3	25	2.2
Aug. 8	19	3.4	15	1.7	17	3.2
Reference range	10-90	0.7-6.7	10-90	0.7-6.7	10-90	0.7-6.7

[1] µmol/L
[2] mmol/L

3.8 Blood electrolytes levels

The levels of electrolytes in the serum of the triplets measured during the forty days after birth are presented in Tables 15-18. The levels of chloride, calcium, and phosphorous stayed within the normal rage. Sukhsaihaj developed hyponatremia and was given sodium chloride orally.

In addition, the serum potassium level exceeded the normal upper limit on July 4th, 18th, and 25th in Parneet's case and she suffered from hyperkalemia (Table 15). The level of potassium in the blood usually increases in cases of acidosis [10, 11].

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Table 15. Parneet's blood electrolyte levels measured during the forty days after birth

Date	Sodium (mmol/L)	Potassium (mmol/L)	Chloride (mmol/L)	Calcium (mmol/L)	Phosphate (mmol/L)
Jun. 30	137	4.2	107	2.13	-
July 1	141	4.7	110	2.33	-
July 2	139	5.6	106	2.55	-
July 3	134	5.4	104	2.48	-
July 4	133	6.2	103	2.52	-
July 5	132	6.8	100	-	-
July 11	134	5.2	102	2.41	-
July 18	136	6.5	102	2.55	2.30
July 25	134	6.9	101	2.9	2.50
Aug. 1	135	5.1	108	2.40	-
Aug. 8	140	4.8	108	2.48	2.12
Ref. range	134-142	3.5-6.0	96-110	1.87-2.50	1.29-2.58

Table 16. Sukhsaihaj's blood electrolyte levels measured during the forty days after birth

Date	Sodium (mmol/L)	Potassium ((mmol/L)	Chloride (mmol/L)	Calcium (mmol/L)	Phosphate (mmol/L)
Jun. 30	137	3.8	107	2.19	-
July 1	139	3.9	108	2.40	-
July 2	137	4.8	108	2.36	-
July 3	134	5.1	105	2.37	-
July 4	132	4.7	102	2.36	-
July 5	131	5.1	100	-	-
Jul. 11	128	5.2	97	2.49	-
Jul. 18	131	5.3	95	2.53	2.24
Jul. 25	129	5.8	94	2.65	-
Aug. 1	134	4.3	105	2.39	-
Aug. 8	136	5.3	104	2.45	2.03
Ref. range	134-142	3.5-6.0	96-110	1.87-2.50	1.29-2.58

Table 17. Imaan's blood electrolyte levels measured during the forty days after birth

Date	Sodium (mmol/L)	Potassium (mmol/L)	Chloride (mmol/L)	Calcium (mmol/L)	Phosphate (mmol/L)
June 30	135	4.7	106	2.16	-
July 1	141	4.8	109	2.45	-
July 2	140	5.2	109	2.49	-
July 3	138	5.2	107	2.54	-
July 4	135	5.7	105	2.53	-
July 5	132	5.9	101	-	-
July 11	133	5.2	101	2.44	-
July 18	132	5.5	98	2.47	2.12
July 25	136	5.3	100	2.54	-
Aug. 1	133	5.9	103	2.37	-
Aug. 8	138	5.3	105	2.48	2.18
Ref. range	134-142	3.5-6.0	96-110	1.87-2.50	1.29-2.58

3.9 Cranial ultrasound exams

The results of the triplet's cranial ultrasound exams performed during the forty-one days after birth are presented in Tables 18-20. Sukhsaihaj's ultrsound exam performed on July 3rd suggests the presence of small old hemorrhage in the brain (Table 19). Furthermore, Imaan's ultrasound exams of July 17th and Aug. 9th are also suggest the presence of old bleeding in the brain (Table 20).

Table 18. The results of Parneet's ultrasound exams performed during the 41 days after birth

Date	Findings
July 3	• No hemorrhage was identified. However, the left lateral ventricle appeared slightly more prominent.
July 17	• The ventricles were slightly asymmetrical with the left being more prominent than the right. This is likely an anatomical variation. No hemorrhage was demonstrated in the germinal matrix or the ventricles.
Aug. 9	• The sizes of the ventricles were again slightly asymmetrical. There was a slight increased echogenicity in the left germinal matrix area. This was slightly more prominent than the previous examination of July 17, 2007.

Table 19. The results of Sukhsaihaj's ultrasound exams performed during the 41 days after birth

Date	Findings
July 3	• There was a tiny 0.2 cm round anechoic focus on the left near the caudothalamic notch. This may represent a tiny old germinal matrix hemorrhage. • The sizes of the ventricles were normal.
July 6	• No evidence of intraventricular hemorrhage was observed.
July 17	• There was no intracranial hemorrhage. The previously demonstrated cystic area in the left germinal matrix was not seen. • The sizes of the ventricles were normal.
Aug. 9	• There was no intracranial hemorrhage and no cystic lesion was seen. • The sizes of the ventricles were normal.

Table 20. The results of Imaan's ultrasound exams performed during the 41 days after birth

Date	Findings
July 6	• No evidence of intraventricular hemorrhage was seen.
July 17	• There was a small echogenic area of 0.5 cm in the left germinal matrix. This may represent a Grade I hemorrhage. This was not clearly visualized in the previous examination. • The sizes of ventricles were normal.
Aug. 9	• The ventricles were normal in size. • There was again a small increased echogenicity in the left germinal matrix. This had not changed from July 17, 2007. It may represent hemorrhage or prominent choroids plexus.

3.10 Chest X-ray exams

The results of the triplet's chest X-ray exams are presented in Tables 21-23. These indicate that the infants were suffering from respiratory distress syndrome (RDS). Sukhsaihaj and Imaan's exam of Aug. 6th indicate that the infants had RDS at the time of discharge from the hospital when they were 42 days of age.

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Table 21. Parneet's chest X-rays

Date	Findings
June 29	• The cardiothymic silhouette was normal. • The lungs were clear.
July 3	• There were mild ground glass opacities involving both lungs.

Table 22. Sukhsaihaj's chest X-rays

Date	Findings
June 29	• The cardiothymic silhouette was within normal limits • Lungs appeared clear .
July 1	• The cardiac silhouette was within normal limits • The lungs had a reticular appearance.
July 2	• The heart was normal in size and lungs were clear.
Aug. 6	• Mild perihilar ground glass opacity with slight prominence of the reticular interstitial markings was noted in the perihilar region of the lungs. The appearance is suggestive of RDS.

Table 23. Imaan's chest X-rays

Date	Findings
June 29	• The cardiothymic silhouette was within normal limits. • The appearance of the lungs was hazy.
July 1	• The cardiac silhouette was within normal limits. • The lungs had a reticular appearance to the lungs.
Aug. 6	• There was mild perihilar ground opacity with slight prominence of the reticular interstitial markings noted in the perihilar region of the lungs. The appearance is suggestive of RDS.

3.11 Eye and hearing exams

The triplets had eye and hearing exams prior to their discharge from the hospital on Aug. 10th and their exams were normal.

3.12 The triplet's feeding and growth rates during the forty-two days after birth

The triplets were fed via parental and oral routes during the 12-13 days following birth. Sukhsaihaj got up to full oral feeds at 12 days of age and the parental feeding was discontinued. Parneet and Imaan reached their full feeds at 13 days of age at which point the parental feeding was stopped.

In the hospital, babies were fe breast milk and formula (Enfamil). They developed gastroesophageal reflux problems and they were treated with Reglan. They were discharged from the hospital on Aug. 10th. Their weight gain rates and growth rates data during the forty-two days after birth are presented in Table 24. Parnnet had slightly less weight gain rate and growth than those of her brothers.

Table 24. The triplet's weight gain rates and growth rates during the forty-two days after birth

Measurements	Parneet (Female)	Sukhsaihaj (Male)	Imaan (Male)
Weight on June 29th	1460 g	1310 g	1380 g
Weight on Aug. 10th	2608 g	2500 g	2580 g
Weight gained in 42 days	1148 g	1190 g	1200 g
Weight gain rate (g/day)	27.3	28.3	28.6
Length on June 29th	43 cm	40 cm	40 cm
Length on Aug. 10th	47.5 cm	45.5 cm	46 cm
Length gained in 42 days	4.5 cm	5.5 cm	6 cm
Length gain rate (cm/month)	3.21	3.93	4.29
Head circumference (cm) on June 29th	28	29	29
Head circumference (cm) on August 10th	32.5	34	34
Head circumf. growth (cm) in 42 days	4.5	5	5
Head circumf. growth rate (cm/month)	3.21	3.57	3.57

4. The triplet's growth performance between August 10th and September 28th and vaccination

The babies were discharged from the hospital on Aug. 10th at 42 days of age, 36.1 weeks post-menstrual age. They stayed at home for 48 days and were hospitalized again on Sept. 27-28th for developing health problems. Parneet was hospitalized on Sept. 27th for developing apnea. She also had bacterial and/or viral infections, subdural bleeding, and a skull fracture. Sukhsaihaj and Imaan were examined and hospitalized on Sept. 28th. Sukhsaihaj had an eye infection and bleeding and Imaan had a skull fracture.

The triplet's cranial ultrasound exams performed on Aug. 30th did not show intracranial bleeding, significant abnormality, or changes from the exam of Aug. 9th. However, in Pareent's case, it showed the lateral ventricle was slightly asymmetrical and both occipital horns were somewhat prominent in size.

Imaan was given with six vaccines on Aug. 29th. He was 61 days old (39 weeks post-menstrual age). These vaccines included diphtheria-tetanus toxoids-acellular pertussis (DTaP); inactivated polio vaccine (IPV); Haemophilus influenzae type B (Hib); and pneumococcal conjugate vaccine (PCV). Parneet and Sukhsaihaj were given these same six vaccines on Sept. 12th at 75 days of age (41 weeks post-menstrual age). They were treated with Tylenol prior to receiving vaccines.

Blood tests were not performed prior to vaccinating the infants to check for anemia, liver problems, clotting problems, or immune depression. These infants were born at 30 weeks gestation and suffered from respiratory distress syndrome, jaundice, anemia, and gastroesophageal reflux (GER). Parneet also developed a tremor after birth.

The mother was treated with betamethasone at 24 and 28 weeks gestation and corticosteroids which are immunosuppressant. The infants were treated with antibiotics for five days after birth. The treatment of infants with antibiotics for a significant period of time can lead to vitamin K deficiency and increases the risk for bleeding [7-9].

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The infants were fed breast milk and Enfamil formula from Aug. 10th until their hospitalization on Sept. 27-28th. The infants suffered from gastroesophageal reflux (GER) and were treated with Reglan. Parneet and Imaan spitted and vomited after feedings more often than Sukhsaihaj.

In Parneet's case, the spitting and vomiting became more severe after her vaccinations on Sept. 12th. Furthermore, Parneet had loose stools, appeared sleepy and irritable, and experienced episodes of vomiting during the week prior to her hospitalization on Sept. 27th. The triplet's weight, length, and head circumference measurements taken between Aug. 10th and Sept. 28th are presented in Table 25. The infant's weight measurements reveal the following:

1) Parneet's weight gain rate (21.3 g/day) for the period after vaccination (September 12-28th) was 26.3% less than her weight gain rate (28.8 g/day) for the period prior to vaccination (Aug. 10th – Sept. 12th).

2) Sukhsaihaj's weight gain rate (41.9 g/day) for the period after vaccination (Sept. 12-28th) was 20.5% less than his weight gain rate (52.7 g/day) for the period prior to vaccination (Aug. 10th – Sept. 12th).

3) Imaan's weight gain rate (37.6 g/day) for the period after vaccination (Aug. 30th –Sept. 28th) was 51.6% more than his weight gain rate (24.8 g/day) for the period between Aug. 10th – Aug. 30th. Imaan was vaccinated on Aug. 29th and his weight was not given.

4) Sukhsaihaj's weight gain rate during the 92 days after birth was 39.1 g/day, which is 44% and 29.3% higher than those of Parneet and Imaan, respectively.

Table 25. The triplet's weights, lengths, and head circumferences measured between August 10th and September 28th

Measurements	Parneet (Female)	Sukhsaihaj (Male)	Imaan (Male)
Weight (g) on Aug. 10	2608	2500	2580
Length (cm) on Aug. 10	47.5	45.5	46
Head circumference (cm) on Aug. 10	32.5	34	34
Weight (g) on Aug. 30	3190	-	3075
Length (cm) on Aug. 30	51	-	19.2
Head circumference (cm) on August 30th	35	-	35.5
Weight (g) on Sept. 12	3560	4240	-
Length (cm) on Sept. 12	53.7	50.5	37
Head circumference (cm) on Sept. 12	36	-	-
Weight (g) on Sept. 28	3900	4910	4166
Head circumference (cm) on Sept. 28	38	-	37

(-): not measured

5. Adverse reactions to vaccines

Each infant of the triples received six vaccines on a single visit to their pediatrician. These vaccines included diphtheria-tetanus toxoids-acellular pertussis (DTaP); inactivated polio vaccine (IPV); Haemophilus influenzae type b (Hib); and pneumococcal conjugate vaccine (PCV). Imaan was vaccinated on Aug. 29th, at 61 days of age (39 weeks post-menstrual age). Parneet and Sukhsaihaj were vaccinated on Sept. 12th, at 75 days of age (41 weeks post-menstrual age).

The vaccines given to the premature triplets contain various antigens, heavy metals, antibiotics, and preservatives [11-13]. 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 [6, 11, 14]. I have also evaluated cases of children and adults who developed serious health problems from vaccines [15, 16].

Serious adverse reactions to the vaccines given to the triplets have been reported in children. These include: allergic reactions, upper and lower respiratory tract infections, ear infections, fever, encephalitis, neurological problems, deafness, pancreatitis, diabetes mellitus, poor appetite, loss of weight, and thrombocytopenia. The followings are clinical studies that describe the adverse reactions to vaccines in children.

In the USA, reports to the Vaccine Adverse Event Reporting System (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 [17].

Furthermore, Zhou *et al.* analyzed reports on the adverse events of vaccines reported to the Vaccine Adverse Event Reporting System (VAERS) from January 1, 1991, through December 31, 2001. VAERS received 128,717 reports. They found that a total of 14.2% of all reports described serious adverse events, which by regulatory definition include death, life-threatening illness, hospitalization or prolongation of hospitalization, or permanent disability [18].

Furthermore, 211 two month-old infants were vaccinated with IPV and DTaP and some of them developed systemic adverse reactions at 24 hours post-inoculation. These include: fever > 102.2 ^{o}F (0.5%); irritability (24.6%); tiredness (31.8%); anorexia (8.1%); and vomiting (2.8%) [12]. In addition, Sakaguchi *et al.* reported the cases of 8 children who developed systemic urticaria within 30 min after administration of acellular diphtheria-tetanus-pertussis (DTaP) vaccine [19].

Carrasco-Garrido *et al.* conducted a 6-month, prospective, observational, multicentre epidemiological vaccine safety study in 2002 in Spain. They wanted to detect the appearance and specify the types of adverse reactions to vaccines in children. Of a total sample of 946 children (0-14 years of age) vaccinated, 191 non-serious suspected adverse reactions were detected (19% of the children). Reactions to the diphtheria, tetanus, pertussis acellular and Haemophilus influenzae type b (DTPa + Hib) vaccine appeared in 43.4% of cases. They stated that an active search for subjects with suspected adverse reactions to vaccines led to the detection of reactions that are usually not reported [20].

Haemophilus influenzae type B (Hib) vaccine has also been known to cause acute and chronic health problems in some

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children. For example, 365 infants were inoculated with Hib, and some of them developed systemic adverse reactions. The following adverse reactions and their percentages occurred in two-month-old infants during the 48 hours following inoculation: fever > 100.8 F (0.6%); irritability (12.6%); drowsiness (4.9%); diarrhea (5.2%); and vomiting (2.7%) [12].

Also, the Institute of Medicine of the United States of America examined putative serious adverse consequences associated with administration of diphtheria and tetanus toxoids, oral polio vaccine and inactivated polio vaccine, and Haemophilus influenzae type B (Hib) vaccines. The committee spent 18 months reviewing all available scientific and medical data from individual case reports to controlled clinical trials.

The committee found that the evidence favored acceptance of a causal relation between diphtheria and tetanus toxoids and Guillain-Barre syndrome and brachial neuritis; between oral polio vaccine and Guillain-Barre syndrome; and between unconjugated Hib vaccine and susceptibility to Hib disease. The committee also found that the evidence established causality between diphtheria and tetanus toxoids and anaphylaxis; between the oral polio vaccine and poliomyelitis and death from polio vaccine-strain viral infection [21].

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. 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.

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 [22].

It has been reported that ill children have failed to respond adequately to vaccination 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.

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 [23].

Parneet, Sukhsaihaj, and Imaan were born at 30 weeks gestation and suffered from respiratory distress syndrome, jaundice, anemia, and gastroesophageal reflux (GER). In addition, Parneet developed a tremor after birth. Their mother was treated with betamethasone at 24 and 28 weeks gestation and corticosteroids which are immunosuppressant. The infants were treated with antibiotics for five days after birth. They were also treated with Reglan for GER problems at the time of vaccination.

On Sept. 27, 2007 (15 days post-vaccination), Parneet vomited the majority of her food after she was bottle-fed at approximately 1800. Parneet was placed in her crib to fall asleep. Approximately one hour later, she was noted to be apenic, cyanotic and not responsive. The parents called the Emergency Medical Services (EMS).

Parneet's mother reported that Parneet appeared sleepy and irritable, experienced episodes of vomiting of food, and had loose stools during the week prior to her apnea on Sept. 27th. Parneet's weight gain rate for the period after vaccination (Sept. 12-28$^{th)}$ was 26.3% less than her weight gain rate for the period prior to vaccination (Aug. 10th – Sept. 12th). Sukhsaihaj's weight gain rate for the period after vaccination (Sept. 12-28th) was also reduced by 20.5% as compared to his weight gain rate for the period prior to vaccination.

6. Parneet's hospitalization on September 27th, clinical tests, illness, and treatments given

Parneet was bottle-fed at approximately 1800 on Sept. 27, 2007 and she vomited the majority of her food. She was placed in her crib to fall asleep. Approximately one hour later, she was noted to be apenic, cyanotic and not responsive. The parents called The Emergency Medical Services (EMS).

The EMS transported Parneet by ambulance to the emergency room (ER) at Windsor Regional Hospital (WRH). In transit, the baby was given oxygen and intermittent positive pressure bagging. The baby had significant bradycardia and poor respiratory drive.

Parneet was admitted to the ER at about 2120 on Sept. 27th. Upon arrival to the ER, Parneet had a heart rate of 80/minute and her temperature was 34^{o}C. She appeared pale and had shallow respiration on oxygen but her blood was well saturated. Her respiratory rate was 40-50/minute. She had a brief apnea episode and no cyanosis. The paramedics and the physician who examined Parneet at the ER observed no evidence of injuries caused by trauma [24].

Parneet was admitted to the Neonate Intensive Care Unit (NICU) at 2132. Physical exam revealed that her fontanelles were flat, her chest was clear, her abdomen was soft and she had umbilical hernia. She had a rectal temperature of 35.9 ^{o}C, pulse rate of 166-176/minute, respiratory rate of 32-37/minute, and blood pressure of 100/72 mm Hg.
The physician and nurses who examined Parneet at the NICU also saw no evidence of injuries caused by trauma [24].

Blood analysis performed following admission showed Parneet was suffering from metabolic acidosis, hyperglycemia, and severe anemia (Tables 26-28).
She was given IV fluids and treated with sodium bicarbonate and her blood pH was raised from 7.29 to 7.43 within 3 hours.

Parneet was breathing quite well on Sept. 28th and she was weaned to room air. However, she was still looking pale and lethargic. She was given a red blood cell transfusion. Parneet had an elevated blood lymphocyte count on Sept. 27th and she was treated with fluconazole 48 mg IV. Her lymphocyte count was reduced by 60% on Sept. 29th (Table 29). Her serum elec-

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trolytes, creatinine, and bilirubin leves were within the normal ranges (Tables 30, 31).

Parneet developed twitching in her right arm and leg. The nurse witnessed six events consisting of right arm and leg twitching, each lasting for two to three minutes. Parneet was given an IV load of Dilantin 10 mg/Kg. She continued to have seizures until Oct. 7th. She was treated with Dilantin, phenobarbitol, and chloral hydrate [24].

The CT scan exam of Parneet's head performed on Sept. 28th showed a large swelling in the left frontal temporal parietal region. A magnetic resonance imaging (MRI) of the head taken at one hour following the CT scan showed subcutaneous hematoma and intracranial bleeding (Table 32). Parneet's eyes were examined on Sept. 29 at 1304 and she had retinal bleeding.

The CT scan of the head performed on Sept. 28th did not show a skull fracture. However, a skull fracture was observed on the X-ray exams of Parneet's head performed on Sept. 29th and 30th (Table 33). Patneet's head circumference of Sept. 29 and Oct. 1st were 38 and 38.5, respectively.

Parneet was treated with mannitol to reduce the edema. The CT scan exam of her head performed on Oct. 1st showed progressive resolution of the intracranial hemorrhage with no evidence of hydrocephalus developing (Table 32). Parneet was also treated with Ramitidin for gastroesophageal reflux (GER).

The examination of Parneet's chest, abdominal region, and limbs did not reveal rib fractures or bone abnormalities (Table 34). She was discharged from the hospital on Oct. 9th. The results of her clinical tests performed in the hospitals are presented in Section 6.1-11.

6.1 Metabolic acidosis

Blood analysis performed following admission showed Parneet was suffering from metabolic acidosis. She had a blood pH of 7.29 and bicarbonate level of 15 mmol/L. Parneet was treated with sodium bicarbonate that raised her blood pH to 7.43 within 3 hours (Table 26). Her blood gases stayed within the normal limits thereafter.

Table 26. Parneet's venous blood gases measured in the hospital

Measurements	Sept. 27 at 2125	Sept. 27 at 2347	Sept. 29 at 1245	Reference range
Blood pH	7.29	7.43	7.37	7.31-7.41
PCO_2 (mm Hg)	28	27	39	40-52
CO_2 (mmol/L)	13	-	22	17-29
Bicarbonate (mmol/L)	15	20	22	22-28
Base Excess	-12.2	-5.5	-2.6	-3.0 to 3.0
PO_2 (mm Hg)	51	50	57	30-50
O_2 Saturation %	84	90	92	60-85

6.2 Hyperglycemia

Parneet's blood glucose was highly elevated (14.3 mmol/L) at the time of admission. Her blood glucose reached a normal level of 5.3 mmol/L on Sept. 29th (Table 27). Blood glucose usually increases as a result of reduction in the level of insulin and/or increase in the level of glucagons caused by stress and/or infection.

Table 27. Parneet's blood glucose levels

Date & Time	Time	Values (mmol/L)
Sept 27	2125	14.3
Sept 27	2130	12.1
Sept 27	2347	8.9
Sept 28	0558	6.8
Sept 29	0840	5.3
Sept 29	2009	5.7
Sept 30	1246	5.6
Oct. 1	0556	5.1
Oct. 3	0600	5.2
Reference range		3.3-5.6

6.3 Severe anemia

Parneet was suffering from severe anemia at the time of admission in the hospital. Her hemoglobin level and red blood cell count were 56% and 54% of the normal average value, respectively. Her platelet count was 117% of the upper limit normal value due to bone marrow hyperplasia which resulted from chronic anemia (Table 28). A blood smear was examined microscopically on Sept. 30th and it showed a mild degree (1+) of anisocytosis. She was given a red blood cell transfusion on Sept. 28th.

Table 28. Parneet's hematology values measured on Sept. 27 through Oct. 3, 2007

Measurement	Sept 27th 2130	Sept 29th 0558	Sept 30th 1246	Oct 3rd 0600	Reference range
Red blood cells x $10^6/\mu L$	2.29	3.68	3.29	3.53	3.50-5.00
HGB (g/dL)	6.8	11.0	9.9	10.4	10-14
HCT%	19.4	31.3	28.1	30.1	30-42
MCV (fL)	85.9	85.1	85.5	85.5	73-85
MCH (pg)	29.7	30.0	30.1	29.6	24-31
MCHC (g/dL)	34.6	35.2	35.2	34.6	32-36
RDW	15.0	15.4	15.6	15.5	12-15
Platelet x $10^3/\mu L$	471	336	339	349	140-400

6.4 Clotting paremeters

On Sept. 28th, Parneet's paritial prothrombin time (PTT) and INR value were 22 seconds (normal range = 30-50) and 0.9 (normal range = 0.9-1.3), respectively. Her PTT value was 27% below the low normal limit. Parneet had an elevated platelet counts of 471 x $10^3/\mu L$ which was 17% higher than the upper normal value. The PPT test is usually influenced by high platelet count [25, 26]. In this case, the result of Parneet's PPT test is probably not valid. The prothrombin time (PT) was not measured in Parneet's case.

6.5 Evidence of infection

Parneet's white blood cell count and lymphocyte count were elevated following admission. She was treated with fluconazole

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48 mg IV on Sept. 28th. Her white blood cell and lymphocyte counts were reduced to normal levels on Sept. 29th (Table 29). These data indicate that Parneet suffered from viral and/or bacterial infections.

Table 29. Parneet's white blood cell and differential counts measured on Sept. 27 through Oct. 3, 2007

Measurement	Sept 27th 2130	Sept 29th 0558	Sept 30th 1246	Oct 3rd 0600	Reference range
White blood cells x 10^3/μL	22.1	12.8	7.1	9.1	5.0-15.0
Lymphocytes x 10^3/μL	13.9	5.2	3.7	3.6	4.0-10.5
Neutrophils x 10^3/μL	6.4	6.0	2.3	4.1	1.0-9.5
Monocytes x 10^3/μL	1.6	1.4	0.7	0.8	0.1-1.1
Eosinophils x 10^3/μL	0.3	0.0	0.4	0.6	0.1-0.7
Basosinophils x 10^3/μL	0.2	0.1	0.0	0.0	0.0-0.2

6.6 Blood electrolytes

Serum analysis performed on Sept. 27th revealed that Parneet had normal levels of sodium, potassium, chloride, and calcium (Table 30). In addition, a blood sample analyzed on Sept. 28th showed that Parneet had a normal serum phosphorous level of 2.06 mmol/L normal range:1.29-2.58) and magnesium level of 0.93 mmol/L (normal range: 0.65-1.05).

Table 30. Parneet's serum levels of electrolytes

Date	Time	Sodium (mmol/L)	Potassium (mmol/L)	Chloride (mmol/L)	Calcium (mmol/L)
Sep. 27	2130	133	5.1	104	-
Sep. 28	0840	138	5.3	105	1.95
Sep. 29	0558	139	3.9	107	2.12
Sep. 29	2009	137	4.5	106	2.22
Sep. 30	1249	138	4.1	106	2.28
Oct. 1	0556	134	5.0	103	2.25
Oct. 3	0600	133	5.7	102	2.39
Ref. range		134-142	3.5-6.0	96-110	1.87-2.50

6.7 Blood levels of urea, creatinine, and bilirubin

Parneet's serum urea, creatinine, total bilirubin, and conjugated bilirubin were within the normal limits at the time of admission and thereafter (Table 31).

Table 31. Parneet's serum levels of urea, creatinine, and bilirubin

Date	Time	Urea (mmol/L)	Creatinine (μmol/L)	Bilirubin total (μmol/L)	Bilirubin conjugated (μmol/L)
Sep. 27	2130	4.0	35	-	-
Sep. 28	0840	8.0	30	17	4
Sep. 29	0558	4.1	25	-	-
Sep. 29	2009	2.8	16	20	4
Sep. 30	1249	3.5	20	16	4
Oct. 1	0556	2.7	20	14	2
Oct. 3	0600	2.2	-	7	1
Ref. range		1.8-8.2	10-90	0-18	0-10

6.8 Subcutanous hematoma and intracranial bleeding

Parneet's cranial ultrasound and CT scan taken at about 15 and 19 hours following admission respectively did not show any bleeding in the brain. The CT scan showed a large swelling in the left frontal temporal parietal region (Table 32)

The magnetic resonance imaging (MRI) taken at 20 hours following admission showed subcutaneous hematoma and intracranial bleeding. The CT scan of the head taken on Oct. 1st showed progressive resolution of the intracranial hemorrhage with no evidence of hydrocephalus developing (Table 32).

Table 32. The results of Parneet's unltrasound, CT, and MRI exams of the head

Date (time)	Test type	Findings
Sept. 28 (1111)	Ultra-sound	• The sizes of ventricles were normal. • No intracranial hemorrhage was seen. • No change from previous examination of Sept. 14th, 2007 was seen.
Sept. 28 (1505)	CT scan	• There was soft tissue swelling in the left scalp. • No fracture of cranium was seen.
Sept. 28 (1614)	MRI	• There were subcutaneous hematomas of different ages over the left frontotemporal parietal region; • diffuse subarachnoid hemorrhages on the left side; • parenchymal hematoma involving the right parietal lobe with multiple stages of hemosiderin.
Oct. 1 (1210)	CT scan	• There was edema of the brain and the right ventricle was not well visualized; • subdural hemorrhage; • intraparencheal hemorrhage involving right parietal lobe.

6.9 Retinal bleeding

Parneet's eyes were examined on Sept. 29 at 1304 and revealed the presence of hemorrhage in the macula (severe) and optic nerve (mild) of the right eye. This exam was conducted at about 39 hours following admission at the hospital.

6.10 Examination of the skull, chest, and other region by CT scan and X-rays

Parneet's CT scan of the head taken at about 15 hours following admission did not show any skull fracture. A skull fracture was first observed on the X-ray taken at 30 hours following admission (Table 33). The examination of Parneet's chest, abdominal region, and limbs did not reveal rib fractures or bone abnormalities (Table 34).

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Table 33. The results of Parneet's CT scan and X-rays

Date (time)	Test Type	Findings
Sept. 28 (1505)	CT scan	• There was soft tissue swelling in the left scalp. No skull fracture was seen.
Sept. 29 (0623)	X-ray	• There was a comminuted fracture of the left posterior parietal and occipital region.
Sept. 30 (0957)	X-ray	• There was a comminuted fracture of the left posterior parietal and occipital region

Table 34. The results of Parneet's X-ray

Date (time)	Region	Findings
Sept. 27 (2131)	Chest	• The heart was not enlarged • The lung field appeared clear • No rib fracture was observed
Sept. 29 (0623)	View of the chest, abdomen, upper and lower limbs	• No bony injury was identified.

6.11 Assessment of Parneet's weight gain rate during her hospitalization

Parneet was admitted to the hospital on Sept. 27th and she was discharged on Oct. 9th. Parneet's weight measured at various time during her stay in the hospital is presented in Table 35. She lost an average of 3 g/day during the first three days following admission. However, she gained weight thereafter. Her weight gain rate between Oct. 1 and 4th was 47.3 g/day. Her weight gain rate was increased to 67 g/day for the period between Oct. 4th and 8th.

Table 35. Parnneet's weight measured in the hospital

Date	Age (days)	Weight (g)
September 27	90	3900
September 30	93	3891
October 4	97	4080
October 8	101	4348

7. Sukhsaihaj's hospitalization on September 28th, clinical tests, illness, and treatments given

Sukhsaihaj was admitted at Windsor Regional Hospital (WRH) at 2220 on Sept. 28, 2007. He was examined because of concerns about child abuse that resulted from Parneet's hospitalization on September 27th. His physical examination revealed that he had a temperature of 37.1°C, pulse rate of 141/minute, and respiratory rate of 28/minute. His blood saturation with oxygen was 99% [27].

Blood analysis performed on Sept. 28th revealed that Sukhsaihaj had severe anemia. His hemoglobin level and hematocrit value were 71% and 68% of the average normal value, respectively. His platelet count was 14% higher than the normal upper limit due to bone marrow hyperplasia (Table 36). His white blood cell count and serum electrolyte levels were within the normal range (Tables 37, 38).

Examination on Sept. 29th revealed that Sukhsaihaj had nasal congestion and his face was swollen. He had difficulty breathing at certain times. His fontanelles were soft. There were three circular smaller than dime-sized bruises on the left temple. He also had a purple dark red streaking like line on the upper chest and both arms with an almost scratch-like appearance. He had a large umbilical hernia.

The MRI exam of Sukhsaihaj's head revealed the presence of a subcutaneous hematoma over the left parieto-occipital region and a small contusion in the brain. An X-ray of the head showed evidence of a left parietal fracture. A skeletal survey X-ray exam did not reveal rib fractures or bone abnormality (Table 39). Sukhsaihaj was treated with chloral hydrate. The nurse called the Children's Aid Society (CAS) and the CAS came with police and took custody of the baby [27].

Examination of Sukhsaihaj's eyes on Sept. 30th revealed that he had bleeding under the cornea of the right eye at 0555. A yellow green discharge, conjunctival hemorrhage, and retinal bleeding were observed in the right eye at 0620 and 1730. The right eye was treated with polysporin eye drops for 9 days.

Sukhsaihaj stayed in the hospital for 11 days and he was discharged with his siblings on Oct. 9th. His daily weight measurements show that he did not gain weight during the first five days after admission. However, he showed weight gain during the five days prior to his discharge. His average weight gain rate was 38 g/day (Table 40).

Table 36. Sukhsaihaj's hematology values

Measurements	Sept. 28 2135	Sept. 30 0720	Reference range
Red blood cell x 10^6/μL	2.84	2.87	3.5-5.00
Hemoglobin g/dL	8.5	8.3	10-14
Hematocrit %	24.4	24.4	30-42
MCV fL	85.7	85.2	73-85
MCH pg	29.9	28.8	24-31
MCHC g/dL	34.9	33.7	32-36
RDW %	15.9	15.8	12-15
MPV fL	7.1	7.4	7.4-10.4
Platelet x 10^3/μL	457	479	140-400

Table 37. Sukhsaihaj's white blood cell and differential count

Measurements	Sept. 28 2135	Sept. 30 0720	Reference range
White blood cell x 10^3/μL	9.1	8.0	5.0-15
Neutrophil x 10^3/μL	1.9	1.2	1.5-8.5
Lymphocyte x 10^3/μL	6.0	5.8	4.0-10.5
Monocyte x 10^3/μL	0.8	0.2	0.1-1.1
Eosinophil x 10^3/μL	0.3	0.5	0.1-0.7
Basophil x 10^3/μL	0.1	0.2	0.0-0.2

Table 38. Sukhsaihaj's serum electrolyte levels

Measurements	Sept. 28 2135	Sept. 30 0720	Reference range
Sodium (mmol/L)	137	135	134-142
Potassium (mmol/L)	6.3	5.2	3.5-6.0
Chloride (mmol/L)	105	102	96-110
Carbon dioxide (mmol/L)	23	20	17-29
Glucose (mmol/L)	4.8	4.6	3.3-5.6
Urea (mmol/L)	2.1	2.1	1.8-8.2
Creatine (μmol/L)	27	22	10-90

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Table 39. The results of Sukhsaihaj's MRI and X-rays performed on September 29th

Time	Exam type	Findings
1430	MRI brain	• Signal change over the left parieto-occipital subcutaneous tissue likely represents a hematoma. • Findings in the right occipital lobe and left frontal lobe are consistent with focal parenchymal contusion.
1034	X-ray skeletal survey	• There was evidence of a left parietal fracture. • No fractured rib was noted. • No metaphyseal corner injuries were present.

Table 40. Sukhsaihaj's weight

Date	Weight (g)
September 9	4910
October 4	4910
October 9	5100

8. Imaan's hospitalization on September 28th, clinical tests, illness, and treatments given

Imaan was admitted at Windsor Regional Hospital (WRH) at about 0440 on Sept. 29, 2007. He was examined because of concerns about child abuse which resulted from Parneet's hospitalization on Sept. 27th. His physical examination revealed that he was alert and awake. His fontanelles were flat. His eyes were normal. His chest was clear. He had a large umbilical hernia [28].

Imaan had a temperature of 36.8°C, pulse rate of 47/minute, and respiratory rate of 38/minute. His blood saturation with oxygen was 99%. There was no evidence of injury caused by trauma observed in the head region or the rest of his body. He was fed Enfamil formula.

Imaan's head CT scan performed on Sept. 29 revealed the presence of a skull fracture involving the right parietal bone and a brain contusion with hemorrhage involving the right parietal lobe. The MRI exam of the head did not show evidence of intracranial hemorrhage. Skeletal survey X-ray exam showed a fracture of the right parietal bone (Table 41). Imaan was treated with chloral hydrate. Imaan was taken with his siblings by the Children's Aid Society (CAS) on suspicion of child abuse [28].

Blood analysis performed on Sept. 30th revealed that Imaan had severe anemia and his platelet count was 12% above the normal upper limit (Table 41). His white blood cell count and serum electrolyte levels were within the normal range (Tables 42, 43).

Imaan stayed in the hospital for 11 days and he was discharged with his siblings on Oct. 9th. His daily weight measurements show that he lost an average of 8 g per day during the first three days after admission. However, he showed weight gain during the eight days prior to his discharge. His average weight gain rate was 28.1 g/day (Table 45).

Table 41. The results of Imaan's MRI, CT scan, and X-rays

Date	Test	Findings
Sept 29 1340	CT scan (head)	• Punctate hyperdensity involving the right parietal lobe likely represents contusion with hemorrhage. • Findings also consistent with a fracture involving the right parietal bone.
Sept 29 1035	Skeletal survey	• There was a fracture of the right parietal bone. • No rib fracture was seen. • No metaphyseal corner injuries were present.
Sept 30 0922	MRI head	• No definite evidence of a subdural or subarchnoid signal abnormality was present.

Table 42. Imaan's hematology values

Measurements	Sept. 30 0720	Reference range
Red blood cell x 10^6/μL	3.15	3.5-5.00
Hemoglobin g/dL	8.5	10-14
Hematocrit %	24.8	30-42
MCV fL	78.7	73-85
MCH pg	27.0	24-31
MCHC (g/dL)	34.3	32-36
RDW %	12.9	12-15
MPV fL	7.8	7.4-10.4
Platelet x 10^3/μL	447	140-400

Table 43. Imaan's white blood cell and differential counts

Measurements	Sept. 30 0720	Reference range
White blood cell x 10^3/μL	8.0	5.0-15
Neutrophil x 10^3/μL	0.9	1.5-8.5
Lymphocyte x 10^3/μL	6.1	4.0-10.5
Monocyte x 10^3/μL	0.4	0.1-1.1
Eosinophil x 10^3/μL	0.5	0.1-0.7
Basophil x 10^3/μL	0.0	0.0-0.2

Table 44. Imaan's serum electrolyte levels

Measurements	Sept. 30 0720	Reference range
Sodium (mmol/L)	134	134-142
Potassium (mmol/L)	5.4	3.5-6.0
Chloride (mmol/L)	102	96-110
Carbon dioxide (mmol/L)	25	17-29
Glucose (mmol/L)	4.1	3.3-5.6
Urea (mmol/L)	2.7	1.8-8.2
Creatine (μmol/L)	31	10-90

Table 45. Imaan's weight

Date	Weight (g)
September 28	4190
October 1	4165
October 9	4390

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9. The likely causes of the intracranial bleeding, brain lesions, and the subcutaneous bleeding observed in the case of the triplets

Magnetic resonance imaging (MRI) of Parneet's head performed on Sept. 28th showed that she had a subcutaneous hematomas of different ages, diffuse subarachnoid hemorrhage, and a parenchymal hematoma (Table 32). Sukhsaihaj's MRI performed on Sept. 29th revealed that he had a subcutaneous hematoma over the left parieto-occipital region (Table 39). Imaan's head CT scan exam of Sept. 30th revealed the presence of a contusion with hemorrhage involving the right parietal lobe brain (Table 41).

Dr. D.W. Warren examined the triplets at the Children's Hospital of Western Ontario on Oct. 11, 2007. He also reviewed their medical records from Windsor Regional Hospital [29]. Warren alleged that the bleeding and the brain lesions observed in the case of triplets were caused by blunt trauma to the head.

The medical data and studies described below clearly show that (1) the subcutaneous and the intracranial bleeding was caused by infections and vitamin K deficiency; (2) brain edema was caused by anoxia, treatment with sodium bicarbonate, and irritation caused by bleeding; (3) the contusion in the brain resulted from the death of cells due to anoxia, edema, and irritation caused by bleeding.

9.1 Infection causes blood clotting problem and bleeding

Parneet's white blood cell and lymphocyte counts were elevated on Sept. 27th. She was treated with fluconazole 48 mg IV on Sept. 28th. Her white blood cell and lymphocyte counts were reduced on Sept. 29th by 42% and 63%, respectively (Table 29). These data indicate that Parneet was suffering from bacterial and/or viral infections.

Parneet was also suffering from severe anemia at the time of admission. Her hemoglobin level and red blood cell count were 56% and 54% of the normal average value, respectively (Table 28). Parneet lost an average of 3 g/day during the first three days following admission in the hospital (Table 35).

Parneet's mother reported that Parneet appeared sleepy and irritable, experienced episodes of vomiting of food, and had loose stools during the week prior to developing her apnea on Sept. 27th. Parneet's weight gain rate for the period after vaccination (Sept. 12-28th) was 26.3% less than her weight gain rate for the period prior to vaccination (Aug. 10th – Sept. 12th).

Sukhsaihaj had nasal congestion on September 29th and his face was swollen. He had difficulty breathing at certain times. He developed nasal congestion at about 11 days prior to his admission in the hospital on Sept. 28th. Examination of Sukhsaihaj's eyes on Sept. 30th revealed that he had bleeding under the cornea of the right eye at 0555. A yellow green discharge, conjunctival hemorrhage, and retinal bleeding were observed in the right eye at 0620 and 1730. The right eye was treated with polysporin eye drops for 9 days.

Sukhsaihaj was also suffering from anemia at the time of admission in the hospital on Sept. 28th (Table 36). He did not gain any weight during the first five days after admission (Table 40). In addition, Sukhsaihaj's weight gain rate for the period after vaccination (Sept. 12-28th) was 20.5% less than his weight gain rate for the time prior to vaccination.

These clinical observations indicate that Parneet and Sukhsaihaj were suffering from infections. Localized and systemic inflammatory reactions associated with the progression of infection usually induce the generation of anti-inflammatory cytokines. They activate leucocytes, endothelium, coagulation and fibrinolysis [30].

Septicemia is frequently accompanied by changes in the plasmatic as well as the cellular coagulation systems and by microclot formation. The activation of coagulation by endotoxin is mediated by synthesis of tissue factor by monocytes and endothelial cells [31].

The formation of microthrombi is caused by the precipitation of circulating soluble fibrin under the influence of localizing factors. It is observed under conditions of reduced fibrinolysis activation. These changes can lead to bleeding diathesis. Furthermore, thrombocytopenia, thrombocytopathy and endothelial cell damage caused by a direct effect of the toxic agent contribute to the bleeding diathesis [31].

Levi *et al.* reviewed articles and published peer-reviewed abstracts on the mechanism of the initiation of disseminated intravascular coagulation (DIC) in sepsis. They found that significant coagulation activation could be detected after the presence of endotoxin in the circulation. This activation is preceded by an increase in the serum levels of various cytokines, such as tumor necrosis factor and interleukins [32].

Impaired function of the protein C-protein S inhibitory pathway can amplify the activation of coagulation. An imbalance between coagulation and fibrinolysis can lead to plasminogen activator inhibitor type 1-mediated inhibition of fibrinolysis. It may further promote the procoagulant state [32].

9.2 The triplet's illnesses led to vitamin K deficiency

Parneet was admitted to the hospital on Sept. 27th. She lost an average of 3 g/day during the first three days following admission (Table 35). Sukhsaihaj and Imaan were admitted to the hospital on Sept. 28th. Imaan lost an average of 8 g per day during the first three days after admission and Sukhsaihaj did not gain any weight during the first five days after admission (Tables 40, 45).

These babies lost weight in the hospital in spite of the treatments and the care given due to their serious illnesses. Parneet, Sukhsaihaj, and Imaan were suffering from severe anemia at the time of their admission to the hospital on Sept. 27th and 28th. However, they had normal hemoglobin levels on Aug. 4th. They lost 38-47% of their hemoglobin levels in six weeks (Table 46).

These babies suffered from gastroesophageal reflux (GER) and in response, were treated with medication.

Furthermore, Parneet and Sukhsaihaj suffered from infection. These data indicate that the triplets did not gain weight or even lost weight for a significant time prior to their hospitalization due to illness, which led to vitamin K deficiency.

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Table 46. Triplet's hemoglobin levels on August 4th and Sept. 27 through 30, 2007

Measurements	Parneet	Sukhsaihaj	Imaan
Hemoglobin level (g/dL) on August 4	12.9	14.4	13.7
Hemoglobin level (g/dL) at admission on Sept. 27-30	6.8	8.5	8.5
Hemoglobin (g/dL) lost in eight weeks	6.1	5.9	5.2
% of hemoglobin lost in eight weeks	47.3	41	38
Hemoglobin (mg/dL) lost per day	113	107	93

9.3 Vitamin K deficiency causes intracranial bleeding in children

The human 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 occurs in serious illness and treatment with high therapeutic doses of antibiotics for a significant time can lead to vitamin K deficiency and intracranial bleeding in children [7, 9, 33-36].

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. Furthermore; two bone matrix proteins necessary for normal bone metabolism are vitamin K-dependent.

These 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 leads to the production of abnormal vitamin K-dependent factors, which lack gamma-carboxy glutamic acid residues in the NH2-terminal part of their molecules [6-9].

Infants who develop vitamin K deficiency usually suffer from intracranial hemorrhages and bleeding in other locations. The symptoms of vitamin K deficiency in infants may include: seizures; convulsions; drowsiness; feeding intolerance and poor sucking; vomiting; fever; pallor; acute diarrhea; irritability and high-pitched cries. The following clinical studies list the symptoms and the bleeding locations in infants who suffered from vitamin K deficiency:

1) Chaou *et al.* reported late-onset intracranial hemorrhage related to vitamin K deficiency in 32 breast-fed infants (1/2 to 6 months of age). Computerized tomography showed mild to severe intracranial hemorrhages. Most (90.6%) had subarachnoid hemorrhages, either alone or in combination with subdural hemorrhage (37.5%), parenchymal hemorrhage (31.3%), or intraventricular hemorrhage (12.5%) [37].

2) Choo *et al.* conducted a retrospective study of 42 newborns who were admitted to the hospital for spontaneous bleeding. The six most common presenting clinical features were pallor, jaundice, umbilical cord bleeding, tense fontanelle, convulsions and hepatomegaly. Anemia was common, especially in cases with massive intracranial bleeding. Subdural hemorrhage was the most common form of intracranial haemorrhage, followed by subarachnoid haemorrhage.

The overall case fatality rate was 14%. None of the infants had bleeding due to inherited coagulopathy or disseminated intravascular coagulation. All the infants had prolonged prothrombin and partial thromboplastin times, which were corrected by administration of vitamin K at an initial dose of 1-5 mg/daily [38].

3) Aydinli *et al.* conducted a retrospective study that included 11 babies between 30 and 119 days of age, who developed bleeding due to vitamin K deficiency. On examination, tense or bulging fontanelle (73%), anisocoria (36%), weak neonatal reflexes (18%), cyanoses (18%) were the most frequent findings. The locations of the intracranial hemorrhages were as follows: intracerebral (91%), subarachnoid (46%), subdural (27%), and intraventricular (27%). The presenting complaints were seizures (91%), drowsiness (82%), poor sucking (64%), vomiting (46%), fever (46%), pallor (46%), acute diarrhea (27%), irritability and high-pitched cries (18%). [39].

4) Bor *et al.* evaluated 15 infants who developed bleeding in the nervous system and other locations. Their age (mean +/- SD) at onset of symptoms was 62.4 +/- 33.9 days. Signs and symptoms present in the infants were convulsions (47%), feeding intolerance and poor sucking (47%), irritability (33%) and pallor (20%). In physical examination, there was bulging or full fontanel in 10 infants (67%), diminished or absent neonatal reflexes in nine infants (60%) and ecchymosis in three infants (20%) [40].

Neurologic, gastrointestinal and skin hemorrhagic findings were found in 11 (73%), 3 (20%) and 3 infants (20%), respectively. There were both neurologic and skin bleeding symptoms in two infants. The mortality rate in this study was 33%. Before administration of vitamin K, prothrombin time (PT) was 76.1 +/- 43.0 s and partial thromboplastin time (PTT) was 123.4 +/- 68.8 s. Six to 12 h after administration of vitamin K, PT was 15.6 +/- 1.8 s and PTT was 33.4 +/- 1.0 s [40].

5) Hanawa *et al.* reported 543 cases of vitamin K deficiency occurring in infants over 2 weeks of age. They divided these infants into three groups based on the causes that led to vitamin K deficiency. The first group consisted of 427 infants who showed no obvious reasons for vitamin K deficiency. In this group, 387 cases (90.0%) were entirely breast-fed and intracranial haemorrhage was observed in 353 cases (82.7%) of this group. 269 cases (63.0%) developed bleeding episodes between the 1st and 2nd months of age.

The second group included 57 cases, who had bleeding episodes due to vitamin K deficiency associated with obvious hepatobiliary lesions, chronic diarrhea, long-term antibiotic therapy, etc. The third group, consisting of 59 cases in which a hemorrhagic tendency, without any obvious clinical hemorrhage, was discovered by Normotest, at the time of mass screening [41].

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9.4 Bleeding in the brain causes inflammation, necrosis, and gliosis

Parneet's MRI and the CT scan exam performed on Sept. 28 and Oct. 1st revealed the presence of subarachnoid hemorrhage and parenchymal hematoma of mixed stage (old and new bleeding). Dr. D.W. Warren reviewed Parneet's MRI and reported the presence of a contusion (bruise) in parietal region of the brain. He alleged that the brain contusion in the brain resulted from trauma to the head [29].

It seems that Warren overlooked the fact that the contusion in the brain in this case was caused by the bleeding and not by trauma. Blood is an irritant substance and can lead to inflammation and necrosis in the brain. The bleeding observed in Parneet's case on Sept. 28th was old and new. Warren estimated the age of the old bleeding to be 7 days.

I reviewed the medical records of a baby who developed chronic intracranial bleeding as a result of vitamin K deficiency and died of cardiac arrest. The bleeding in the brain caused edema, necrosis, and atrophy involved a large area of her brain [7]. Below are clinical and experimental studies that show bleeding in the brain has caused edema, inflammation, necrosis, and gliosis:

1) Mayer *et al.* studied the development of brain edema following the occurrence of intracerebral hemorrhage (ICH) in 23 individuals. They performed paired consecutive CT and 99mTc-hexamethylpropylenamine oxime single-photon emission computed tomography (SPECT) scans during the acute (18 hours) and subacute (72 hours) phase of ICH. Hematoma and edema volumes were traced and calculated from CT images.

They found that the ICH volume (18 mL) did not change but the mean edema volume was increased by 36% (from 19 to 25 mL, P<0.0001). Perilesional edema on CT always corresponded topographically with perfusion deficits on SPECT [42].

2) Patel *et al.* induced intracranial hemorrhage in rats by the injection of 100 or 200 μL of blood into the subdural space (SDH). Brain edema was measured using the wet/dry weight method. They detected significant increases in water content of the cerebral cortex of the brain at 24 hours after SDH as compared with those of the control animals (control: 0.1+/-0.1 g/g dry weight; 200 μL: 0.8+/-0.3 g/g dry weight; P<0.001) [43].

3) Gong *et al.* conducted a study in rats to evaluate the development of brain edema following the induction of intracerebral hemorrhage (ICH). Immunocytochemistry for polymorphonuclear leukocyte marker (myeloperoxidase, MPO), microglia marker (OX42) and intracellular adhesion molecule-1 (ICAM-1) was performed in control, and 1, 3, 7 and 10 days after the injection of 100 μL autologous blood in the right basal ganglia. They observed an inflammatory response in the brain after ICH at that infiltrating leukocytes and activated microglia may release cytotoxic mediators contributing to secondary brain injury and edema formation [44].

4) Koeppen *et al.* injected 100 μL of autologous whole blood intracerebrally in adult rabbits. They found that the extravasation of blood elicits a cellular reaction in the adjacent surviving tissue where the lesion activates resident microglia and attracts many more phagocytes from the blood stream. The cellular responses to the injections were studied by iron histochemistry and immunocytochemistry for ferritin, the ferritin repressor protein (FRP), the glial fibrillary acidic protein (GFAP), and the complement receptor CR3.

Conversion to hemosiderin began at 5 days after the injection of blood. The lesions caused initial destruction of astrocytes in the perifocal zone as judged by GFAP- and FRP-immuno-reactivity. However, at 5 days, astrocytic processes reentered the perifocal zone and intermingled with microglia and macrophages [45].

10. The likely causes of Parneet's retinal bleeding

Parneet was admitted to Windsor Regional Hospital on Sept. 27th with an apnea. Her eyes were examined two days following admission and the examination revealed the presence of retinal bleeding in the right eye. Dr. D.W. Warren alleged that the bleeding resulted from a traumatic brain injury. My reviewed of the clinical data and published medical studies pertinent to this case indicates that Parneet's retinal bleeding was caused by systemic infections, vitamin K deficiency, and severe anemia.

The clinical data and studies that show Parneet was suffering from infections and vitamin K deficiency at the time of admission are described in Sections 6-9 of this report. Infection, vitamin K deficiency, and severe anemia have caused retinal bleeding in children. For example, Parneet's brother, Sukhsaihaj developed retinal bleeding on Sept. 30th as a result of infections, vitamin K deficiency, and anemia.

His eye exam at 0555 on Sept. 30th showed that he had bleeding under cornea of the right eye at 0555. A yellow green discharge, conjunctival hemorrhage, and retinal bleeding were observed in the right eye at 0620 and 1730. The right eye was treated with polysporin eye drops for 9 days. In addition, I evaluated the medical records of a baby who developed subretinal hemorrhage due to severe anemia, vitamin K deficiency, and Tylenol induced liver injury [26].

Parneet was suffering from a severe anemia on Sept. 27th. She had hemoglobin levels of 6.8 g/dL and hematocrit of 19.4%. Her hemoglobin level and hematocrit values were at 54% of the normal average value. Below are clinical studies that show individuals suffered from severe anemia also developed retinal bleeding and inflammation.

1) Asien *et al.* evaluated the occurrence of clinically apparent retinal changes in 35 anemic individuals and 35 age-and sex-matched healthy control subjects. Retinal photographs of all subjects were obtained and all vascular and extra vascular retinal lesions were recorded. No retinal abnormalities were observed in the control subjects.

Seven (20%) of the anemic individuals exhibited extra vascular lesions (flame-shaped hemorrhages, hard exudates, and cotton-wool spots). The mean hemtocrit reading for these individuals was 24.7%. A significant negative correlation was determined between venous length and the level of hematocrit. It indicates that retinal venous tortuosity is directly related to severity of anemia [46].

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2) Carraro *et al.* conducted a cross-sectional study involving 226 individuals with anemia and/or thrombocytopenia to evaluate the incident of retinopathy among these individuals. 47 healthy age-matched subjects were used as controls. Retinopathy was observed in 28.3% of the individuals as a whole.

The presence of fundus lesions was closely associated with severe anemia (Hb < 8 g/dL) and severe thrombocytopenia (platelet < 50 x 10^9/L). Among the individuals with concomitant anemia and thrombocytopenia, the incidence of retinopathy was 38%. Retinal hemorrhages were found in all of the individuals with concomitant severe anemia and thrombocytopenia [47].

11. The likely causes of the skull fractures observed

Imaan and Sukhsaihaj were admitted at Windsor Regional Hospital because of concerns about child abuse which resulted from Parneet's hospitalization on Sept. 27th. The X-ray exams of their heads performed on Sept. 29th showed fractures of the right parietal bone in Imaan's case and the left parietal bone in Sukhsaihaj's case (Tables 39, 41).

Dr. D.W. Warren reviewed their X-rays and stated that the etiology of the skull fracture in Imaan's case is a traumatic injury. "It is consistent with a mild to moderate blunt force trauma to the area. The nature of this force is consistent with a fall from a moderate height. Fractures of this nature have been seen with falls **from a changing table on to a wood or tile floor**. It is also consistent with a young infant of this age being thrown against a hard surface [29]".

In Sukhsaihaj's case, Warren stated that the fracture in the skull is traumatic in origin. "We find this would be consistent with a greater force to the head than occurred in his brother Imaan. It was again consistent with blunt force trauma to this area. This fracture is more consistent with a more significant blunt force trauma to the skull. This could be in relation **to a fall from a greater height, possibly from the top of stairs, or from a more significant blow to a hard surface** [29]".

Furthermore, Parneet's X-ray exams of Sept. 29th and 30th showed a comminuted fracture of the left posterior parietal and occipital region (Table 33). Warren stated "the extent of this fracture could only have occurred by a full force blow to the skull of this child. **This type of force in a fall I would expect from a height of 3 to 5 floors on to a hard surface** [29]".

Warren alleged that **the forces used to cause the skull fracture in these infants are equivalent to a fall from a changing table on to a wood or tile floor in Imaan's case, the top of stairs on to a hard surface in Sukhsaihaj's case, and a height of 3 to 5 floors up on to a hard surface in Parneet's case**. However, allegedly significant forces involved did not cause neck injuries or even a tiny scratch on the skin of the head in any infant. The triplets were examined in the hospital for 11 days and the treating physicians and nurses observed no evidence of external injury caused by trauma.

The clinical data and medical studies described in this report reveal the following:

1) The skull fractures in these infants resulted from protein and vitamin K deficiencies and infection.

2) The severity of bleeding and skull fracture depends upon the severity of the protein and vitamin K deficiencies, and the degree of infection. The severity of bleeding and skull fracture in Parneet's case is greater than in Sukhsaihaj and Imaan's case. Her weight gain during the first 91 days following birth (26.8 g/day) is less than those of her siblings (30.6-39.6 g/day) and she suffered from systemic infections. The severity of the bleeding and skull fracture in Sukhsaihaj's case is greater than Imaan's case because Sukhsaihaj suffered from infection.

Tanaka *et al.* stated that protein malnutrition increases the fracture risk due to decreased bone mineral density and muscle weakness [48]. Rizzoli *et al.* also reported that protein deficiency contributes to the occurrence of osteoporotic fractures not only by decreasing bone mass but also by altering muscle function [49].

3) The injuries and infections observed in the triplets resulted from the six vaccines given. The severity of the injuries and infections correlate with their hemoglobin levels at the time of vaccination. Imaan was vaccinated on Aug. 29th and Sukhsaihaj and Parneet were vaccinated on Sept. 12th. Imaan did not suffer from infection following vaccination because he had a significantly higher blood hemoglobin level than his siblings.

The expected blood hemoglobin level at the time of vaccination in Imaan's case was 11.4 g/dL. The expected hemoglobin levels in Parneet and Sukhsaihaj's case were 8.5 g/dL and 10.2 g/dL, respectively. The hemoglobin levels of these babies (12.9-14.4 g/dL) were within the normal limits on Aug. 4th because they received red blood cell transfusions. However, their hemoglobin levels were reduced at the rate of 0.1 g/dL per day for the time between Aug. 4th and Sept. 27th (Table 46).

4) The severity of the skull fractures observed in the triplets depends on the level of the intracranial pressure as indicated by the severity of edema and bleeding. The severity of the skull fracture in Parneet's case is greater than in Sukhsaihaj's case or Imman's case. The severity of Sukhsaihaj's skull fracture was greater than that in Imaan's case.

The intracranial bleeding and edema in Parneet's case were more severe than those observed in Imman and Sukhsaihaj's case. Her head circumference on Oct. 1st was 38.5 cm, which is 2 cm more than the expected. She was treated with mannitol and her head circumference was decreased to 37 cm on Oct. 4th. I evaluated the medical records of a baby and a toddler who developed skull fractures which resulted from vitamin K deficiency and increased intracranial pressure due to edema and bleeding [8, 50].

In addition, Fenton *et al.* reported two cases of infants who died as a result of massive intracranial hemorrhage caused by vitamin K deficiency in one infant and disseminated herpes simplex virus infection in the second infant. The radiographic exams of their heads taken prior to death revealed the presence of linear parietal fractures. At autopsy, the parietal bone abnormalities were found not to be fractures, but proved to be an anomalous suture in 1 and a connective tissue fissure in the other [51].

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 glu-

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tamate to gamma-carboxyglutamate (Gla) and 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 [9, 52-54].

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 [53].

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 μg/day) [55].

Furthermore, 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 (chi2 =10.935; p = 0.0273) [56].

12. The likely causes of the rib fractures and bone abnormalities observed

Parneet, Sukhsaihaj, and Imaan's chest X-ray exams performed on Sept. 27-29th at Windsor Regional Hospital (WRH) did not show rib fractures in any of these infants (Tables 34, 39, 41). However, Dr. D.W. Warren from the Children's Hospital of Western Ontario reviewed these X-rays and stated that he found 9, 3, and 2 healed rib fractures in Parneet, Sukhsaihaj, and Imaan's X-rays, respectively (Table 47).

Furthermore, Warren reviewed Parneet, Sukhsaihaj, and Imaan's chest X-rays performed on Oct. 11th at Children's Hospital. He reported additional healed rib fractures involving left 4th in Parneet's case and left and right 10th in Sukhsaihaj's case (Table 47). Warren stated that the rib fractures in these infants resulted from excessive squeezing of the chest.

Warren also stated that the examination of Parneet's legs "revealed increased periosteal thickening of the right and left femurs. There is also evidence of a left tibial chip fracture. These are consistent with excessive force in grabbing the legs of this young infant." Parneet's X-rays taken at WRH in Sept. 27-29th did not show bone abnormalities in the femurs and the tibia.

The triplets were taken into custody by the Children's Aid Society (CAS) on Sept. 28th and stayed in the hospital. The parents had contact with the babies only under supervision. If Warren's allegations are true then his observations must explain how

1) **It is possible that the radiologist(s) at WRH failed to identify 14 fractured ribs on the triplet's chest X-rays;**

2) **Who allegedly squeezed the chests of these infants between September 28th and October 11th to cause the additional rib fractures identified by Warren on October 11th;**

3) **Who allegedly squeezed Parneet's legs to cause the periosteal thickening of the femurs and the tibial chip fracture.**

The medical evidence presented in this report shows that the triplets were suffering from vitamin K and protein deficiency and infection. I explained the roles of protein and vitamin K in bone metabolism. Severe protein and vitamin K deficiency increase the risk for bone fracture. I reviewed the records of babies and a toddler who had multiple rib fractures which resulted from protein and vitamin K deficiency and infection (Table 48).

Table 47. Healed rib fractures identified by Warren on the triplets X-rays

Infant	X-rays of Sept. 27-29	X-rays of October 11th
Parneet	Right posterior 5th, 6th, 7th, 8th Left posterior 5th, 6th, 7th, 8th, 9th	Right 7th, 8th Left posterior 4**th**, 5th, 6th **7th,** 8th
Sukhsaihaj	Right posterior 7th, 8th, 9th	Right posterior 7th, 8th, 9th, 10**th** Left lateral 10**th**
Imaan	Left 5th and 6th	Left 5th and 6th

Table 48. Rib fractures in children caused by vitamin K and protein deficiency

Child & Reference #	Age (Months)	Location of the healed rib fractures
Patrick [26]	3	Right 1st, 6th, 8th, 9th Left 4th, 5th, 7th, 8th
Roman [8]	18	Left 5th, 6th, 7th, 8th, 9th

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13. Conclusions and recommendations

The clinical data and medical studies described in this report reveal the following:

1) The triplets developed serious adverse reactions to the six vaccines given to them on Aug. 29, 2007 in Imaan's case and on Sept. 12th in case of Parneet and Sukhsaihaj. These include infection, gastrointestinal problems, and growth retardation.

2) The severity of the injuries and infections observed in the triplet's case correlates with their hemoglobin levels at the time of vaccination. Imaan did not suffer from infection following vaccination because he had significantly higher blood hemoglobin level than his siblings. The expected blood hemoglobin levels at the time of vaccination in Parneet, Sukhsaihaj, and Imaan's case were 8.5 g/dL, 10.2 g/dL, and 11.4 g/dL, respectively.

3) The subcutaneous and intracranial bleeding observed in the triplets resulted from protein and vitamin K deficiencies and infection. The severity of the bleeding correlates with the severity of the protein and vitamin K deficiencies and the degree of infections.

4) Severe anemia, vitamin K deficiency, and systemic infections are the likely causes of Parneet's retinal bleeding. At the time of admission, she had hemoglobin levels of 6.8 g/dL and hematocrit of 19.4%, respectively.

5) The bone fractures observed in the triplet's case resulted from protein and vitamin K deficiencies. The severity of the skull fractures correlate with the severity of the protein and vitamin K deficiencies, and the degree of the infections, and the levels of the intracranial pressure.

6) Warren's allegations that the triplet's injuries were caused by blunt trauma to the head and child abuse are not medically valid. It seems that he did not use differential diagnosis in this case to consider the role of the adverse reactions to vaccines, infections, vitamin K and protein deficiency in causing the triplet's health problems.

I believe that the following recommendations will help in monitoring the triplet's injuries and healing process. They may also help in preventing the occurrence of similar vaccine related injuries in the future:

1) The clinical data show that the triplets were suffering from severe anemia and thrombocytosis due to bone marrow depression and hyperplasia. Blood analysis should be performed periodically to monitor for these illnesses.

2) Vaccines should not have been given to the triplets while they were anemic and sick. Blood tests should be performed prior to giving vaccines to these children to check for anemia, liver problems, clotting problems, and immune depression. In addition, benefit and risk analysis should be performed prior to vaccination. Like other medications, vaccines are capable of causing serious health problems in children. I believe that giving six vaccines to a premature and sick infant at one office visit is not a medically justified action.

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