Analysis of causes that led to baby Jackie Ray’s developmental delay and intracranial bleeding

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1. Summary of the case and findings

Jackie Ray Lewis III is a black male infant from Nacogdoches County, Texas. He had a seizure on May 31, 2007 while he was with his father and two siblings (2-5 years of age) at home. The father reported the incident to the baby’s mother when she returned from work and she took the baby to Nacogdoches Memorial Hospital (NMH) for examination. Jackie was 93 days old.

Jackie was examined at the ER and no signs of trauma were identified. A CT scan exam of his head performed at 1.25 hours following his admission to the hospital (FAH) showed only a small area of bleeding in the left frontal region. A second CT head exam taken at about 40 hours FAH revealed that the baby had subdural hemorrhages and brain edema. A brain MRI exam performed at 89 hours FAH showed the baby’s subdural bleeding was bilateral and at various ages.

The treating physicians alleged that vigorous shaking (Shaken Baby Syndrome) caused the baby’s bleeding. The baby’s father was accused of causing his son’s injuries. My investigation in this case reveals that the baby received 7 vaccines at two months of age, while he was ill. He suffered from developmental delay, anemia and femoral abnormalities. His head circumference (HC) was 38.7 cm on the day of vaccination and it decreased to 37.3 cm at 32 days post vaccination. The baby’s HC growth rate during the 2 months prior to vaccination was 2.8 cm/month.

His rate of weight gain was also reduced by 32% following vaccination as compared to the rate prior to vaccination. Vitamin K deficiency was the likely cause of the baby’s bleeding and femoral abnormalities. The allegations of Shaken Baby Syndrome and child abuse made in this case are false.

Abstract

A 3 month-old black male infant had a seizure and his mother took him to the hospital for examination. The baby was examined at the ER and no signs of trauma were identified. A CT scan exam of his head performed at 1.25 hours following his admission to the hospital (FAH) showed only a small area of bleeding in the left frontal region. A second CT head exam taken at about 40 hours FAH revealed that the baby had subdural hemorrhages and brain edema. A brain MRI exam performed at 89 hours FAH showed the baby’s subdural bleeding was bilateral and at various ages.

The treating physicians alleged that vigorous shaking (Shaken Baby Syndrome) caused the baby’s bleeding. The baby’s father was accused of causing his son’s injuries. My investigation in this case reveals that the baby received 7 vaccines at two months of age, while he was ill. He suffered from developmental delay, anemia and femoral abnormalities. His head circumference (HC) was 38.7 cm on the day of vaccination and it decreased to 37.3 cm at 32 days post vaccination. The baby’s HC growth rate during the 2 months prior to vaccination was 2.8 cm/month.

His rate of weight gain was also reduced by 32% following vaccination as compared to the rate prior to vaccination. Vitamin K deficiency was the likely cause of the baby’s bleeding and femoral abnormalities. The allegations of Shaken Baby Syndrome and child abuse made in this case are false. © Copyright 2008, Medical Veritas International Inc. All rights reserved.

Keywords: anemia, brain edema, child abuse, developmental delay, diphtheria-tetanus toxoids-acellular pertussis vaccine, head circumference, Haemophilus influenzae type b vaccine, Hepatitis B vaccine, inactivated polio vaccine, pneumococcal conjugate vaccine, seizure, subdural bleeding, vaccine, Shaken Baby Syndrome, vitamin K deficiency.

1. Summary of the case and findings

Jackie Ray Lewis III is a black male infant from Nacogdoches County, Texas. He had a seizure on May 31, 2007 while he was with his father and two siblings (2-5 years of age) at home. The father reported the incident to the baby’s mother when she returned from work and she took the baby to Nacogdoches Memorial Hospital (NMH) for examination. Jackie was 93 days old.

Jackie was examined at the ER and no signs of trauma were identified. A CT scan exam of his head performed at 1.25 hours following his admission to the hospital (FAH) showed only a small area of increased attenuation in the left frontal region that raised suspicions about hemorrhage. Jackie was given IV fluids and put on O2 breathing and transferred to Texas Children Hospital (TCH) on June 1, 2007 for further evaluation.

Jackie’s CT scan exam performed at about 40 hours FAH revealed that Jackie had subdural hemorrhages and brain edema. In addition, a brain MRI exam performed at 89 hours FAH showed his subdural bleeding was increased significantly. The subdural hemorrhages were bilateral and at various ages.

The treating physicians alleged that vigorous shaking (Shaken Baby Syndrome) caused Jackie’s subdural bleeding. Jackie was discharged from the hospital on June 5th and released into custody designated by the Child Protective Services (CPS). Jackie’s father was accused of causing his son’s injuries, arrested, and indicted by a grand jury.

The father’s defense attorney requested that I evaluate the medical evidence in baby Jackie’s case to find the likely causes that led to his bleeding and his health problems. 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 baby Jackie’s medical records and the articles cited in this report using differential diagnosis. Approximately 200 hours were required to evaluate the medical evidence, perform an analysis, and write this report. My findings in this case include:

1) Jackie received 7 vaccines at his pediatrician’s office on April 30, 2007 while he was ill. Jackie was vomiting and had nasal congestion. His mother took him to Nacogdoches Memorial Hospital for examination. Jackie was vaccinated with Hepatitis B vaccine, diphtheria-tetanus toxoids-acellular pertussis (DTaP); inactivated polio vaccine (IPV); Haemophilus influenzae type b (Hib); and pneumococcal conjugate vaccine (PCV). He was 62 days old (Section 3).

2) Jackie suffered from developmental delay following the 7 vaccines on April 30th. His head circumference (HC) on April 30th was 38.7 cm and it decreased to 37.3 cm on June 1st. It was reduced by 1.12 cm/month during the 32 days following vaccination. Jackie’s HC growth rate during the 62 days prior to receiving the vaccines on April 30th was 2.8 cm/month. It is expected that Jackie’s HC would be at least 40 cm on June 1st.

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In addition, Jackie gained weight at the rate of 33.3 g/day during the 2 months prior to the receiving 7 vaccines on April 30th and his weight gain rate decreased to 22.7 g/day (32% reduction) during the 32 days following vaccination. The reduction in Jackie’s weight gain rate indicates that his food intake was reduced significantly following vaccination (Section 3).

3) The majority of Jackie’s subdural hemorrhages occurred following his admission to the hospital (FAH) on May 31st as shown by his CT head and MRI brain exams. A CT head exam performed at 1.25 hours FAH showed only a small area of bleeding in the left frontal region. A second CT head exam performed at about 40 hours FHA showed the subdural hemorrhages involving more areas of the brain and brain edema. Furthermore, An MRI exam of his brain performed at 89 hours FHA revealed that the subdural bleeding had increased significantly. The subdural bleeding was bilateral and at various ages (Section 4).

These clinical data do not support the allegations that Jackie’s bleeding occurred prior to his admission to the hospital on May 31st and resulted from vigorous shaking and abuse. In addition, Jackie was examined in the hospital at 5 minutes FAH and no signs of trauma were identified.

4) The likely cause of Jackie’s developmental delay and subdural bleeding was vitamin K deficiency as described in Section 5 of this report. Vitamin K deficiency in infants has led to developmental delay, intracranial hemorrhages, and bleeding in other locations. For example, Demirören et al. described the outcome of 19 infants with intracranial hemorrhage (ICH) due to vitamin K deficiency. Ten infants were followed for a mean period of 26.9 +/- 22.6 months. The follow-up findings were developmental delay (40%), microcephaly (30%), epilepsy (30%), blindness (20%), strabismus (20%), spastic tetraparesis (10%), spastic hemiparesis (10%), growth retardation (10%), and hydrocephaly (10%).

The localizations of the ICHs were as follows: Parenchymal (47%), subarachnoid (47%), subdural (42%), and intraventricular (26%). The most frequent presenting complaints were convulsion (58%), vomiting (47%), and irritability (47%). The most frequent examination findings were coma (74%), fontanel bulging (68%), and absence of pupil reaction (42%)

In addition, Aydinli et al. conducted a retrospective study that included 11 babies between 30 and 119 days of age, who developed intracranial bleeding due to vitamin K deficiency. These children were followed for a period of 6 to 48 months (mean: 21 +/- 13 months). Seizure disorders (73%), severe psychomotor retardation (46%), cerebral palsy (46%) and microcephaly (46%) were observed.

The localizations of the intracranial hemorrhage 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 cry (18%)

5) Bone abnormalities are also biomarkers of vitamin K deficiency. Jackie’s knee performed on June 12th showed both femurs were intact. It is likely that the abnormalities in Jackie’s femurs were reversed due to receiving adequate amount of vitamin K between June 3rd and June 12th.

Some of the bone matrix proteins necessary for normal bone metabolism are vitamin K-dependent. Vitamin K is a coenzyme for glutamate carboxylase that mediates the conversion of glutamate to gamma-carboxyglutamate (Gla) 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 (Section 5).

6) Jackie’s health problems were induced by the 7 vaccines given to him on April 30, 2007. These vaccines contain various antigens, heavy metals, antibiotics, and preservatives. Additive and synergistic actions among these components causing serious health problems can occur even in healthy children and adults.

For example, reports sent to the USA 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.

Jackie was ill when he was vaccinated and vaccines should not be given to a sick child. I have evaluated cases of infants and a toddler who died as a result of adverse reactions to vaccines. I also evaluate cases of children and adults who developed serious health problems from vaccines (Section 6).

7) The allegations of Shaken Baby Syndrome and child abuse made in this case are false and Jackie’s father is innocent.

2. Jackie’s health condition and growth rate during the 62 days prior to receiving 7 vaccines on April 30, 2007

2.1 Jackie’s health condition at birth and clinical tests performed

Jackie is a black male infant. He was born at 37 weeks of gestation via vaginal delivery on February 27, 2007 at Nacogdoches Memorial Hospital, Texas. Labor was induced due to leaking amniotic fluid and pain. His Apgar score at 1 and 5 minutes was 9.

Jackie’s weight was 2537 g (10-25 percentile). His length and head circumference were 46 cm (10-25 percentile) and 33 cm (25-50 percentile), respectively. He was vaccinated with hepatitis B vaccine following birth on February 27th.

A blood test performed on February 28th revealed that Jackie was not anemic or suffering from infection (Table 1). He also had a normal blood glucose level of 58 mg/dL (reference range: 40-79 mg/dL). Jackie passed his hearing test on March 1, 2007 and he was discharged from the hospital without receiving 0.5 mg vitamin K (IM) to decrease his risk for developing intracranial bleeding and bleeding in other locations [1].

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2.2 Jackie’s exam at one week of age
Jackie was discharged from the hospital on March 1, 2007. He was fed breast and formula milk and had no postpartum complications. He developed mild jaundice on March 6th. He had a total blood bilirubin level of 7.7 mg/dL (reference range: 0.0-12.0 mg/dL) and a direct bilirubin level of 0.3 mg/dl (reference range: 0.0-12.0 mg/dL). His body weight was 2.55 kg.

2.3 Jackie’s exams on March 20th and April 11, 2007
Jackie’s check up exam and the newborn screening tests performed on March 20th were normal (Table 2). His physical exam was normal. He had a total blood bilirubin level of 7.7 mg/dL (reference range: 0.0-12.0 mg/dL) and a direct bilirubin level of 0.3 mg/dl (reference range: 0.0-12.0 mg/dL). His body weight was 2.55 kg.

2.4 Jackie’s exam on April 17, 2007
Jackie suffered from an allergic reaction on April 17th and was given 7 vaccines in his pediatrician’s office on April 30th (Table 4). The clinical data collected during the times prior and post vaccination show that the vaccines caused developmental delay and other health problems in Jackie’s case (Section 3.1 & 3.2).

Table 2. Newborn screening tests performed in Jackie’s case

<table>
<thead>
<tr>
<th>Screening type</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amino Acid Disorders</td>
<td>Normal</td>
</tr>
<tr>
<td>Fatty Acid Disorders</td>
<td>Normal</td>
</tr>
<tr>
<td>Organic Acid Disorders</td>
<td>Normal</td>
</tr>
<tr>
<td>Galactosemia</td>
<td>Normal</td>
</tr>
<tr>
<td>Biotinidase deficiency</td>
<td>Normal</td>
</tr>
<tr>
<td>Hypothyroidism</td>
<td>Normal</td>
</tr>
<tr>
<td>Congenital adrenal hyperplasia</td>
<td>Normal</td>
</tr>
<tr>
<td>Hemoglobinopathies</td>
<td>Normal</td>
</tr>
</tbody>
</table>

3. Jackie’s health problems developed following the receipt of 7 vaccines on April 30, 2007
Jackie appeared sick on April 30th. He was vomiting and had nasal congestion. His mother took him to Nacodoches Memorial Hospital for a check up. An upper gastrointestinal tract study was performed and no reflux was identified during Jackie’s examination.

Table 3. Jackie’s weight, length, and head circumference measurements taken between birth and April 30, 2007

<table>
<thead>
<tr>
<th>Date</th>
<th>Age (days)</th>
<th>Weight (g) &amp; (percentile)</th>
<th>Length (cm) &amp; (percentile)</th>
<th>HC (cm) &amp; (percentile)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2/27/07</td>
<td>Birth</td>
<td>2537 (10th-25th)</td>
<td>46.0 (10th-25th)</td>
<td>33.0 (25th-50th)</td>
</tr>
<tr>
<td>3/20/07</td>
<td>21</td>
<td>2868 (10th-25th)</td>
<td>50.8 (10th-25th)</td>
<td>34.3 (25th-50th)</td>
</tr>
<tr>
<td>4/11/07</td>
<td>43</td>
<td>3750 (15th)</td>
<td>52.7 (10th-25th)</td>
<td>36.8 (25th-50th)</td>
</tr>
<tr>
<td>4/30/07</td>
<td>62</td>
<td>4602 (25th)</td>
<td>55.3 (15th)</td>
<td>38.7 (10th-25th)</td>
</tr>
</tbody>
</table>

*First injection was given following birth on February 27, 2007

3.1 Jackie’s developmental delay
Jackie’s head circumference (HC) on April 30th was 38.7 cm and it decreased to 37.3 cm on June 1st (Table 5). It was reduced by 1.12 cm/month during the 32 days following the 7 vaccines on April 30th. Jackie’s HC growth rate during the 62 days prior to receiving the vaccines on April 30th was 2.8 cm/month (Table 6). It is expected that Jackie’s HC would be at least 40 cm on June 1st [1, 2].

Brandt evaluated head circumference growth rate in cm/month from the prenatal period until the age of 18 months. Measurements were made in 60 appropriate for gestational age (AGA) preterm infants of very low fetal age and 68 full term infants. They found a period of rapid head circumference growth -- a growth spurt -- extends from the 31st postmenstrual week until the 6th month after term. In the following months the velocity curve flattens. If the age is not corrected for prematurity, the peak of the velocity curve becomes flat and spread with a mean growth velocity of 3.0 cm in the first, of 3.4 cm in the second and of 2.5 cm in the third month [3].
It appears that Jackie had about a 3 cm reduction in his HC during the 32 days following vaccination. In addition, during the 2 months prior to receiving 7 vaccines on April 30th, Jackie gained weight at the rate of 33.3 g/day. His weight gain rate decreased to 22.7 g/day (32% reduction) during the 32 days following vaccination (Tables 5,6). The reduction in Jackie’s weight gain rate indicates that his food intake was reduced significantly following vaccination [1, 2].

Jackie’s length increased at the rate of 5.3 cm/month during the 32 days following receiving vaccines on April 30th, which is 20% higher than the rate of 4.5 cm/month observed during the 2 months prior to vaccination (Tables 5, 6). Jackie’s weight measured on May 31st and June 1st indicates that he was suffering from edema. His weight on May 31st and June 1st were 6000 g and 5327 g, respectively. He lost 673 g in one day (Table 5). These data indicate that Jackie’s higher than normal length increase rate observed following vaccination resulted from fluid retention and does not represent a higher rate of growth.

Table 5. Jackie’s weight, length, and head circum. (HC) measured before and after receiving vaccines on April 30

<table>
<thead>
<tr>
<th>Date</th>
<th>Age days</th>
<th>Weight (g) &amp; (percentile)</th>
<th>Length (cm) &amp; (percentile)</th>
<th>HC (cm) &amp; (percentile)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2/27/07</td>
<td>Birth</td>
<td>2537 (10th-25th)</td>
<td>46.0 (10th-25th)</td>
<td>33.0 (25th-50th)</td>
</tr>
<tr>
<td>4/30/07</td>
<td>62</td>
<td>4602 (25th)</td>
<td>55.3 (10th-25th)</td>
<td>38.7 (25th)</td>
</tr>
<tr>
<td>5/31/07</td>
<td>93</td>
<td>6000</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6/01/07</td>
<td>94</td>
<td>5327 (10th-25th)</td>
<td>61.0 (50th)</td>
<td>37.5 (&amp;3rd)</td>
</tr>
</tbody>
</table>

Table 6. Jackie’s weight gain and growth rates during the periods prior and post vaccination on April 30, 2007

<table>
<thead>
<tr>
<th>Measurements</th>
<th>02/27-04/30 (62 days)</th>
<th>05/01-06/01 (32 days)</th>
</tr>
</thead>
<tbody>
<tr>
<td>HC growth (cm)</td>
<td>5.7</td>
<td>-1.2</td>
</tr>
<tr>
<td>HC growth rate (cm/month)</td>
<td>2.8</td>
<td>-1.1</td>
</tr>
<tr>
<td>Weight increase (g)</td>
<td>2065</td>
<td>725</td>
</tr>
<tr>
<td>Weight increase rate (g/day)</td>
<td>33.3</td>
<td>22.7</td>
</tr>
<tr>
<td>Increase in length (cm)</td>
<td>9.3</td>
<td>5.7</td>
</tr>
<tr>
<td>Increase in length rate (cm)</td>
<td>4.5</td>
<td>5.3</td>
</tr>
</tbody>
</table>

*Period prior to receiving 7 vaccines on April 30th.

3.2 Symptoms observed during the 24 hours prior to Jackie’s hospitalization

Jackie appeared irritable on the night of May 30th. He was congested and coughing. He also appeared irritable in the morning of May 31st. He refused to take his pacifier and did not sleep well. He took 10-15 minutes naps. Jackie’s mother went to work at 1500 and left the baby with his father. Jackie’s father put the baby in his crib around 1700 after feeding him and the baby slept until 2000. The baby woke up crying and his father put him on the couch. He noticed that Jackie was not responding as usual but he was still breathing. The father tried to wake Jackie up by moving him back and forth and moving his arm for a few minutes. The baby responded but appeared sleepy.

The father laid Jackie on the bed until his mother returned home from work after 2100. The mother took the baby by car to the Nacogdoches Memorial Hospital ER for evaluation when she heard the story. It took about 5 minutes to reach the hospital. She noted that Jackie appeared sleepy and he was not moving much, but he was breathing and had no color change [2].

4. Jackie’s hospitalization on May 31–June 5, 2007, clinical tests, diagnosis, and treatments given

Jackie was brought by his mother to Nacodoches Memorial Hospital (NMH) at 2206 on May 31, 2007. The mother told the physician at the ER that Jackie had a seizure at home. He was jerking and his eyes rolled back. Jackie was 93 days of age.

Jackie was examined at 2211 and no signs of trauma were identified. The baby was sleeping and appeared uncomfortable. He had a pulse rate of 111/min, a respiratory rate of 36/min, and a rectal temperature of 99.1°F. His blood O2 saturation level was 97%. His weight was 6.0 kg [2].

A peripheral IV was started at 2249 and Jackie blood was drawn. His blood analysis showed that he was suffering from anemia. His red blood cell count and hemoglobin level were 71% and 81% of the low normal limit values. His platelet count was 123% of the upper limit value (Table 7). He had a blood glucose level of 152 mg/dL (Table 8).

A chest X-ray exam performed at about 1 hour following Jackie’s admission to the hospital (FAH) was normal. Jackie’s head CT scan exam performed at 1.25 hours FAH showed only a small focus of increased attenuation in the left frontal region that raised suspicions about hemorrhage (Table 9).

Jackie’s treatment with IV fluid started at 009 and June 1st. He was given D5 1/2 normal saline IV at 22 cc/hour. Jackie’s weight at 1500 on June 1st was 5.33 kg. His length and head circumference were 61 cm and 37.5 cm, respectively [2, 4].

Jackie was transferred from Nacogdoches Memorial Hospital (NMH) by Nacogdoches County EMS to Texas Children Hospital (TCH) on June 1, 2007 for further evaluation. They left NMH at 1613 and arrived to TCH at 1836. During transportation, Jackie was on O2 breathing and given IV fluids. He had a pulse rate of 110/min, a blood pressure of 80/50 mm Hg, and a respiratory rate of 24/min [2, 4].

A CT scan exam performed at about 40 hours FAH revealed that Jackie had subdural hemorrhages and brain edema. In addition, a brain MRI exam performed at 89 hours FAH showed that the subdural bleeding was increased. The subdural bleeding was bilateral and at various ages (Table 9).

Jackie’s skeletal survey performed at 1054 on June 2nd revealed a slight bilateral irregularity of the medial distal femoral metaphysis. Jackie’s eyes were examined at 1249 on June 2nd and appeared normal. No retinal hemorrhage was observed in either eye.

Electroencephalogram exam was performed in Jackie’s case at 1443 on June 5th and revealed abnormal results. The findings suggested the presence of potentially epileptiform abnormalities. A focal disturbance in the right temporal region was also noted.

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The treating physicians alleged that Jackie’s subdural hemorrhages were resulted from nonaccidental trauma. Jackie was discharged from the hospital on June 5th. He was released into custody designated by the Child Protective Services (CPS). The clinical data collected in both hospitals are presented in Section 4.1-5.

4.1 Evidence of anemia and hyperglycemia
A blood analysis performed at 43 minutes following his admission to the hospital showed that he was suffering from anemia and hyperglycemia. His red blood cell count and hemoglobin level were 71% and 81% of the low normal limit value. His platelet count was 123% of the upper limit value (Table 7). He had a blood glucose level of 152 mg/dL (Table 8).

4.2 Jackie’s chest X-ray exam
A chest X-ray exam performed at 2301 on May 31st was normal. Jackie’s lungs were well expanded and clear of acute infiltrates. The heart size and the mediastinal structures were within the normal limit.

4.3 Skeletal survey and knee X-ray
Jackie’s skeletal survey performed at 1054 on June 2nd revealed a slight bilateral irregularity of the medial distal femoral metaphysis. A knee X-ray exam performed at 1403 on June 3rd also showed mild irregularity of the medial aspect of the distal femur.

4.4 Jackie’s eye exam
Jackie’s eyes were examined at 1249 on June 2nd and appeared normal. No retinal hemorrhage was observed in either eye.

4.5 Jackie’s CT head and MRI brain exams
A CT head exam performed at 1.25 hours following Jackie’s admission to the hospital (FAH) showed only a small focus of increased attenuation in the left frontal region that raised suspicion about hemorrhage. A second CT head exam performed at about 40 hours FAH showed subdural hemorrhages involving more areas of the brain and brain edema. In addition, a brain MRI exam performed at 89 hours FAH revealed that the subdural bleeding increased. The subdural bleeding was bilateral and at various ages (Table 9).

4.6. Jackie’s electroencephalogram exam
Electroencephalogram exam was performed in Jackie’s case at 1443 on June 5th and revealed abnormal results. It showed a focus of slow activity in the left temporal region and occasional spike activity in this region. An independent spike and slow wave activity were also present in the left central and right occipital regions.

<table>
<thead>
<tr>
<th>Test type and time</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>CT Scan May 31st at 2321</td>
<td>A small focus of increased attenuation in the left frontal region suspicious for a small hemorrhage. No evidence of midline shift, mass effect, or extraxial fluid collection.</td>
</tr>
<tr>
<td>CT Scan June 2nd at 1443</td>
<td>Hyperdense subdural blood seen superiorly and to the left along the falx with posterior and downward extension again along the falx and along both tentorial leaflets.</td>
</tr>
<tr>
<td>MRI June 4th at 1105</td>
<td>Bilateral thin cerebral hemispheric subdural hematoma at various ages.</td>
</tr>
<tr>
<td>MRI June 4th at 1105</td>
<td>The majority of hematomas adjacent to the left cerebral hemisphere with the exception of posterior hematoma were chronic hematomas. The hematomas identified posteriorly adjacent to the left hemisphere were early subacute.</td>
</tr>
<tr>
<td>MRI June 4th at 1105</td>
<td>Hematoma identified along the majority of the right hemisphere was mostly compatible with the late subacute blood.</td>
</tr>
<tr>
<td>MRI June 4th at 1105</td>
<td>Hemispheric subdural hematomas were identified about the cerebellar hemispheres.</td>
</tr>
</tbody>
</table>

Table 8. Jackie’s serum analysis performed at 2249 on May 31

<table>
<thead>
<tr>
<th>Measurements</th>
<th>Values</th>
<th>Reference range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose</td>
<td>152</td>
<td>70-110 mg/dL</td>
</tr>
<tr>
<td>Sodium</td>
<td>135</td>
<td>135-145 mmol/L</td>
</tr>
<tr>
<td>Potassium</td>
<td>4.4</td>
<td>3.5-5.0 mmol/L</td>
</tr>
<tr>
<td>Chloride</td>
<td>105</td>
<td>98-108 mmol/L</td>
</tr>
<tr>
<td>Calcium</td>
<td>10.1</td>
<td>8.7-10.2 mg/dL</td>
</tr>
<tr>
<td>CO₂</td>
<td>23</td>
<td>24-32 mmol/L</td>
</tr>
<tr>
<td>Anion Gap</td>
<td>11</td>
<td>5-16 mmol/L</td>
</tr>
<tr>
<td>BUN</td>
<td>10</td>
<td>8-22 mg/dL</td>
</tr>
<tr>
<td>Creatinine</td>
<td>0.5</td>
<td>0.8-1.3 mg/dL</td>
</tr>
<tr>
<td>Total protein</td>
<td>6.4</td>
<td>6.4-8.2 g/dL</td>
</tr>
<tr>
<td>Albumin</td>
<td>4.2</td>
<td>3.2-5.0 g/dL</td>
</tr>
<tr>
<td>Globulin</td>
<td>2.2</td>
<td>2.8-3.2 g/dL</td>
</tr>
<tr>
<td>Total bilirubin</td>
<td>0.6</td>
<td>0.0-1.5 mg/dL</td>
</tr>
<tr>
<td>Alkaline phosphatase</td>
<td>280</td>
<td>108-310 IU/L</td>
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<tr>
<td>SGOT</td>
<td>39</td>
<td>5-45 IU/L</td>
</tr>
<tr>
<td>SGPT</td>
<td>22</td>
<td>10-40 IU/L</td>
</tr>
<tr>
<td>Osmolality</td>
<td>272</td>
<td>275-295 mOsm/kg</td>
</tr>
</tbody>
</table>

Table 9. Jackie’s CT and MRI brain exams

<table>
<thead>
<tr>
<th>Measurements</th>
<th>Values</th>
<th>Reference range</th>
</tr>
</thead>
<tbody>
<tr>
<td>RBC</td>
<td>3.2</td>
<td>4.5-5.3 x 10⁹/µL</td>
</tr>
</tbody>
</table>
5. The likely causes of Jackie's subdural bleeding and abnormalities in the femurs

The clinical data and the medical studies described below show that: 1) The majority of Jackie's subdural hemorrhages occurred following Jackie’s admission to the hospital on May 31st; 2) Jackie’s brain edema was induced by the bleeding; 3) his subdural bleeding and femoral abnormalities were caused by vitamin deficiencies, especially vitamin K; and 4) vaccines given to Jackie on April 30, 2007 caused vitamin and protein deficiencies and developmental delays.

5.1 Progress of Jackie's subdural hemorrhages

The CT head and the MRI brain exams taken during the 89 hours following Jackie’s admission to the hospital (FAH) showed that the majority of the subdural bleeding occurred during his stay in the hospital. His first CT head exam performed at 1.25 hours FAH revealed that the bleeding was limited to a small area in the left frontal region. His second CT head exam taken at about 40 hours FAH revealed that the subdural hemorrhages involved a wider area than that of the first exam (Table 9).

Furthermore, his brain MRI exam performed at 89 hours FAH revealed that the subdural bleeding had increased significantly. The subdural bleeding was bilateral and at various ages (Table 9). The MRI result indicates that the subdural bleeding occurred over the course of several days.

These clinical data do not support the allegations made in this case that Jackie’s bleeding occurred prior to his admission to the hospital on May 31st and resulted from vigorous shaking and abuse. In addition, Jackie was examined in the hospital at 5 minutes FAH and no signs of trauma were identified. The likely cause of Jackie’s bleeding was vitamin K deficiency. Developmental delay and bone abnormalities are also biomarkers of vitamin K deficiency and these conditions were observed in Jackie’s case (Section 5.2).

5.2 Vitamin K deficiency in infants has caused developmental delay and bleeding

Jackie suffered from developmental delay following receiving the 7 vaccines on April 30, 2007. His head circumference (HC) on April 30th was 38.7 cm and it decreased to 37.3 cm on June 1st (Table 5). It is expected that Jackie’s HC would be at least 40 cm on June 1st.

Furthermore, Jackie’s weight gain rate decreased by 32% following the vaccines on April 30th as compared to the rate prior to receiving vaccines (Table 6). These data indicate that his food intake was reduced significantly following vaccination and it led to the deficiency of vitamin K and other essential nutrients such as iron. Jackie was also suffering from anemia.

Vitamin K deficiency in infants has led to developmental delays, intracranial hemorrhages, and bleeding in other locations. For example, Demirören et al. described the outcome of 19 infants with intracranial hemorrhage (ICH) due to vitamin K deficiency. Ten infants were followed for a mean period of 26.9 +/- 22.6 months. The follow-up findings were developmental delay (40%), microcephaly (30%), epilepsy (30%), blindness (20%), strabismus (20%), spastic tetraparesis (10%), spastic hemiparesis (10%), growth retardation (10%), and hydrocephaly (10%) [5].

Furthermore, Aydinli et al. conducted a retrospective study included 11 babies between 30 and 119 days of age, who developed intracranial bleeding due to vitamin K deficiency. These children were followed for a period of 6 to 48 months (mean: 21 +/- 13 months). Seizure disorders (73%), severe psychomotor retardation (46%), cerebral palsy (46%) and microcephaly (46%) were observed. Hydrocephalus developed in three (27%) babies [6].

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 has led to the production of abnormal vitamin K-dependent factors, which lack gamma-carboxyglutamic acid residues in the NH2-terminal part of their molecules [7-14].

In addition to developmental delay and intracranial bleeding, 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 cry. The following clinical studies list the symptoms and the bleeding locations and frequency in infants who suffered from vitamin K deficiency.

1) Pooni et al. evaluated 42 infants who developed intracranial hemorrhage (ICH) and bleeding in other sites as a result of vitamin K deficiency. The majority of these infants (76%) were 1-3 months. They found that 71% of these infants presented with intracranial hemorrhage, the most common site being intracerebral and multiple ICH. Visible external bleeding was noted in 1/3rd of the infants. Three infants died [15].

2) Chaou et al. reported 32 cases of infants (0.5-6.0 months of age) who developed intracranial bleeding due to vitamin K deficiency. Computerized tomography showed mild to severe intracranial hemorrhage. Most (90.6%) had subarachnoid hemorrhage, either alone or in combination with subdural hemorrhage (37.5%), parenchymal hemorrhage (31.3%), or intraventricular hemorrhage (12.5%). In three (9.4%) the infratentorial region was involved [16].

3) Demirören et al. described the clinical and laboratory findings of 19 infants with intracranial hemorrhage (ICH) due to vitamin K deficiency. The mean age at onset of symptoms was 49 +/- 18 days. The localizations of the ICHs were as follows: Parenchymal (47%), subarachnoid (47%), subdural (42%), and intraventricular (26%).

The most frequent presenting complaints were convulsion (58%), vomiting (47%), and irritability (47%). The most fre-
quent examination findings were coma (74%), fontanel bulging (68%), and absence of pupil reaction (42%) [5].

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

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 [17].

5) 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. The localizations of the intracranial hemorrhage 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 cry (18%) [6].

6) Nishio et al. examined 84 cases from the medical literatures of intracranial hemorrhage in children due to vitamin K deficiency. Hemorrhage sites were identified by CT scan in these children. Subarachnoidal hemorrhage was found in 72 cases (85.7%), subdural hemorrhage in 41 cases (48.8%), intracerebral hematomas in 36 cases (42.9%) and intraventricular hemorrhage in 9 cases (10.7%) [18].

7) Doneray et al. described the clinical and demographic features of 16 cases with late vitamin K deficiency bleeding. Ages of infants were between 30 and 130 days. Intracranial hemorrhage was the most common bleeding site (37.5%), and two children (12.5%) died because of it. The most common presenting finding was pallor (62.5%) [19].

5.3 Vitamin K deficiency causes abnormalities in bones and bone fractures

Some of the bone matrix proteins necessary for normal bone metabolism are vitamin K-dependent. Vitamin K is a coenzyme for glutamate carboxylase that mediates the conversion of glutamate to gamma-carboxyglutamate (Gla) 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 Ca$^{2+}$ and incorporate these ions into the hydroxypatite crystals [7, 20-22].

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 [21].

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) [23].

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 in the vitamin K2-treated group (chi2 = 10.935; p = 0.0273) [24].

I have also evaluated the medical records of several babies who developed subdural bleeding, bone fractures, and bone abnormalities and differential identified vitamin K deficiency as the primary cause of these health problems [9-14]. Jackie’s skeletal survey performed at 1054 on June 2nd revealed a slight bilateral irregularity of the medial distal femoral metaphysis. As knee X-ray exam performed at 1403 on June 3rd also showed mild irregularity of the medial aspect of the distal femur.

However, Jackie’s X-ray exam of the knee performed on June 12th showed both femurs were intact [25]. It is likely that the abnormalities in Jackie’s femurs were reversed due to receiving adequate amount of vitamin K between June 3rd and June 12th.

6. Adverse reactions to vaccines given to Jackie on April 30, 2007

Jackie was vomiting and had nasal congestion on April 30th. His mother took him to Nacodoches Memorial Hospital for examination. He was 62 days old. His pediatrician gave him 7 vaccines on April 30th after he was discharged from the hospital. These vaccines include Hepatitis B vaccine, diphtheria-tetanus toxoids-acellular pertussis (DTaP); inactivated polio vaccine (IPV); Haemophilus influenzae type b (Hib); and pneumococcal conjugate vaccine (PCV).

Jackie had a seizure and appeared sick on May 31, 2007 and his mother took him to the hospital for examination. CT head and MRI brain exams revealed subdural hemorrhage. His blood analysis revealed that he was anemic.

Jackie’s head circumference measurements indicate that he suffered from developmental delay following vaccination. His
The twins had received the second dose of oral polio, DPT and the first dose of hepatitis B vaccines. They developed fever on the first day of the vaccination and were given Tylenol (acetaminophen) [37].

The database from the 1994 National Health Interview Survey (NHIS) in the USA that included 6,515 children less than six years of age who received the hepatitis B vaccine were analyzed to evaluate the vaccine related adverse reactions. Hepatitis B vaccine was found to be associated with prevalent arthritis (odds ratio (OR) = 5.91, 95% confidence interval (CI) = 1.05-33.14), incident acute ear infections (OR = 1.60, 95% CI = 1.00-2.58), and incident pharyngitis/nasopharyngitis (OR = 1.41, 95% CI = 0.95-2.09) [38].

Wise et al. evaluated 4,154 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 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 [39].

7. Conclusions

My review of the medical evidence pertinent to baby Jackie’s case revealed the following:

1) Jackie suffered from developmental delay following receipt of the 7 vaccines on April 30th. His head circumference (HC) on April 30th was 38.7 cm and it decreased to 37.3 cm on June 1st. Jackie’s HC growth rate during the 62 days prior to receiving the vaccines on April 30th was 2.8 cm/month. It is expected that Jackie’s HC would be at least 40 cm on June 1st.

2) The majority of Jackie’s subdural hemorrhages occurred following his admission to the hospital on May 31st as shown by the CT head and MRI brain exams. The likely cause of the bleeding in Jackie’s case was vitamin K deficiency. Vitamin K deficiency has been known to cause subdural bleeding in infants.

3) Vitamin K deficiency was the likely cause of Jackie’s femoral abnormalities observed on June 2nd and 3rd, 2007.

4) Jackie’s health problems were induced by the 7 vaccines given to him on April 30, 2007. Jackie was ill when he was vaccinated and vaccines should not be given to a sick child.

5) The allegations of Shaken Baby Syndrome and child abuse made in this case are false and Jackie’s father is innocent.
References


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