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April 22nd 2003

John L Leckey LL.M
HM Coroner
Coroner's Office
Courthouse
Old Town Hall Building
80 Victoria Street
Belfast BT1 3GL

Dear Mr Leckey,

Re: Lucy Crawford (Deceased)

Please find, enclosed my report on this little girl.

I have tried to set out the sequence of events leading to her death, as I see them.

I believe she died from hyponatraemia leading to acute cerebral swelling because of failure to administer the correct replacement fluids. You will see from my report I am critical of this and also the failuré to write a proper fluid prescription chart.

I also enclose an account for your kind attention in due course.

Thanks you for asking my opinion on this matter.

Yours sincerely

Edward Sumner
Consultant paediatric anaesthetist

LC-SLT

MEDICOLEGAL REPORT

ON

LUCY CRAWFORD

Deceased

dob: 5th November 1998

Died: 13th April 2000

Prepared for:

John L Leckey LL.M
HM Coroner
Coroner's Office
Courthouse
Old Town Hall Building
80 Victoria Street
Belfast BT1 3GL

By:

Edward Sumner MA, BM, BCh, FRCA
Consultant Paediatric Anaesthetist
[redacted]

April 2002

My name is Edward Sumner and I am a consultant in Paediatric Anaesthesia with an interest in Intensive Care.
I was consultant at the Great Ormond Street Hospital for Children, London, from 1973 until June last year. I am the author of several textbooks on the subject and am the Editor-in-Chief of the Journal, *Paediatric Anaesthesia.*
Currently, I am the President of the Association of Paediatric Anaesthetists of Great Britain and Ireland.

In the preparation of this report I have carefully perused all the medical and nursing notes presented to me by the Erne Hospital, Enniskillen, Northern Ireland.

I understand that my overriding duty is to the Court on matters which are within my expertise. I also believe that the facts I have stated in this report are true and that the opinions I have expressed are correct.

Lucy was born by planned caesarian section for maternal reasons on 5th November 1998, weighing 7lb 2oz. She progressed normally, though she required hospitalisation for 2 days from 18th November 1999 with bronchiolitis caused by Respiratory Syncitial Virus (RSV), but recovered from that.

On 12th April 2000 at 7 30 pm Lucy was again admitted to hospital with a history of being off her food for 5 days, fever and vomiting for 36 hours and being drowsy for the previous twelve hours or so. The history suggests that she vomited everything she had eaten or drunk. At that time Lucy was 17 months old and weighed 9.1kg.
She was up-to-date with all her immunisations.

On examination she was found to have a fever of 38 °C, heart rate of 140 per min and a respiratory rate of 44 per minute.
Capillary refill was greater than 2 seconds, but her tongue was moist. She was noted to be conscious and pink.
A viral illness was diagnosed.

Urine analysis showed ketones ++ and protein ++ (++++in the nurses notes) Blood analysis showed Hb 12.1, white cells 15, sodium 137, potassium 4.1, CO_2 16 (low) and urea 9.9 (high). Blood was also taken for blood cultures and it was decided to provide intravenous fluids after venous cannulation.

Anaesthetic cream (Ametop) was applied at 7 30 pm, though Dr Malik was unable to cannulate a vein, but Dr O'Donohoe achieved cannulation in the left hand at around 10 pm and IV fluids were commenced at 1030 at 100ml per hour of 4% dextrose/ 0.18% saline. The prescription chart for these fluids is page 65, but there is no note at which rate the medical staff wanted the fluids to run.
During the evening, Lucy had taken some sips of fluid and some of this had been tolerated.

The Intake/Output chart on page 64 suggests that 50ml of juice and 100ml Dioralyte had been given orally. She continued to be drowsy and lethargic. Paracetamol 120mg was given at 10pm. It is noted that she vomited at midnight (++) and at 2 30 am she passed a large, runny bowel motion and because of the fear of cross-infection, Lucy was moved into a side room and a stool sample was sent for bacteriological examination (rotavirus was subsequently cultured)

IV fluids were maintained at 100ml per hour of the no 18 solution.

At 2 55 am the nurses were called by Mrs Crawford because Lucy had become rigid in here arms. Dr Malik was called. At that time there was no loss of colour, no cyanosis and the pulse and breathing were satisfactory. Oxygen therapy was commenced. Dr Malik ordered diazepam (2.5mg) to be given rectally, but this provoked a large watery offensive stool. Dr O'Donohoe was also present.
At that time the blood pressure was 144/113 and the pulse rate 160; blood sugar was 13.4 mmol. The intravenous fluids were changed to 0.9% saline and allowed to run in freely – 500ml were given over one hour. Serum electrolytes at the time were: sodium 127, potassium 2.5.

By 3 20 am, there was decreased respiratory effort – an oral airway was inserted and Dr Malik began to "bag" Lucy. At this stage the pupils had become dilated and unresponsive.
Dr O'Donohoe had 2 unsuccessful attempts at tracheal intubation, but this was eventually achieved with a size 4 oral tube by the anaesthetist, Dr Anterson.

At 4pm the benzodiazepine antagonist, Flumazenil 100ug IV was given.

Oxygen saturations were always maintained between 85 and 100% during this period of time. A urinary catheter was inserted and a small amount of clear urine drained.
Lucy was then transferred to the ICU at around 5 am to be transferred later to the Children's Hospital, Belfast.
Mannitol 5g, to reduced cerebral swelling was given IV over 30 minutes.

In a note written on 14th April 2000 after Lucy's death, Dr O'Donohoe states it was his recollection that the fluids had been NaCl 0.18%, dextrose 4% and had been given as a bolus of 100ml and then at a rate of 30 ml per hour after that. He worked out a maintenance fluid protocol at approx 30ml per hour for the transfer. Dr Anterson had suggested 40ml per hour.

Lucy arrived in the PICU in Belfast at 7 45 in the morning of the 13th April, completely unresponsive. Brainstem tests performed at 8 45 and 10 30 am were negative, CT scan showed complete obliteration of the basal cisterns and the electroencephalogram was flat.
She was taken off the ventilator and extubated at 1pm and died shortly after that.

The clinical diagnosis at the time was said to be dehydration and hyponatraemia; cerebral oedema, acute coning and brain stem death.

At postmortem, Lucy weighed 12kg. The lungs were considerably congested and oedematous, especially on the right with well developed bronchopneumonia.

The brain showed features of generalised cerebral oedema with evidence of mild uncal herniation and some grooving in the tonsillar regions.

I would like to make the following comments:

The evidence is that Lucy, a previously healthy little girl died of acute cerebral swelling leading to "coning", the process in which intracranial pressure rises to such a degree that the base of the brain is forced down into the foramen magnum with subsequent brain death.

On admission, Lucy weighed 9.1kg and at post-mortem examination, 12kg and even though inaccuracies do occur in weighing children, she had gained a great deal of weight during her brief hospitalisation – somewhere in the region of 2kg – equivalent to 2 litres of water.

Her circulating blood volume was approximately 700ml. (Approx 80ml/kg body weight)

It is difficult to judge exactly how dehydrated Lucy was on admission to hospital. A capillary refill time in excess of 2 seconds is one sign of approximately 5% dehydration, however, this sign is likely to be hard to interpret in a febrile child. At this level of dehydration, mucous membranes are dry, but it was noted that Lucy's tongue was moist. I think, on balance that she was mildly dehydrated – perhaps somewhat less than 5% and involving a fluid deficit of approximately 350ml.

Lucy's symptoms before hospitalisation were lassitude, fever and vomiting; the diarrhoea came later during her time in hospital.
Vomiting causes a severe loss of both water and electrolytes. Sodium and acid are lost from the stomach in the vomiting and as a compensatory mechanism the kidneys in trying to conserve sodium allow a net loss of potassium.

Intravenous fluid therapy in this situation is aimed at restoring the calculated deficit, replacing ongoing losses and providing normal maintenance fluids while gastro-intestinal function is deranged.
It is common, and good practice in this clinical situation to make a formal assessment of the degree of dehydration, to note the physical signs and then to write down the calculations for prospective intravenous fluid management. This was only done retrospectively after Lucy's death.
What is **absolutely mandatory** is to write a proper fluid prescription on a designated chart stating which fluid is to be given and **at what rate**. This was not done. Dr O'Donohoe thought Lucy was having 30ml per hour of dextrose/saline, but in fact she was having 100ml per hour.

The Intravenous Chart on page 65 must be rather old fashioned since it gives details of subcutaneous fluid administration – a practice abandoned decades ago.

Four percent dextrose/ 0.18% saline is a **totally** inappropriate fluid to make up deficits from vomiting and diarrhoea. The dextrose is immediately metabolised and so this solution is effectively providing only water.
An appropriate fluid would be normal (0.9%) saline with a potassium supplement (eg 10mmol in 500ml), Hartmann's or lactated Ringer's solutions. This could have been as an initial bolus of 100-150ml over the first hour, to cover approximately half the calculated deficit, then the rest of the deficit plus normal maintenance fluids could be given over the next 12-24 hours. The deficit by this stage would be approximately 200ml (8ml per hour) plus normal maintenance of 5ml per kg per hour – a total of approx 50ml per hour, most of which should be saline, though some could be a dextrose containing solution.
Additionally, on-going losses from vomiting and diarrhoea should be replaced by an equal volume of normal (0.9%) saline plus potassium.

In the event, Lucy was given only 4% dextrose/0.18% saline in a volume of 400ml from 1030 pm to 3pm when she became acutely unwell with what was probably a seizure.

Hyponatraemia is defined as a serum sodium level of less than 128mmol.l^{-1}. The second measured sodium level was 127 mmol.l^{-1}, within that definition, but representing a rapid and dramatic fall from the first level measured at 8 50 pm (137 mmol.l^{-1})

It is not clear from the notes exactly what time the second set of electrolytes was taken – the laboratory printout is not timed.* At some time during this period the fluids had been changed from dextrose/saline to normal (0.9%) saline and a very large volume (500ml) given. This volume of saline represents 70% of the circulating blood volume. It is possible that the serum sodium had been lower, but increased during the administration of this huge volume of saline.

* Not Timed?

The dilution of the serum electrolytes by an excess administration of water is clearly shown by the acute reduction in the serum potassium from 4.1 to 2.5 mmol.l^{-1}.

It is also possible that Lucy produced inappropriate Antidiuretic Hormone (ADH) which causes retention of water in the body and is secreted in times of stress and infection.

The tendency to oedema from an excess of administered water would be exacerbated by the vast over infusion of saline at the time of resuscitation.

It is known that an acute reduction in serum sodium by the administration of excess fluids with a low sodium content leads to cerebral oedema by an osmotic effect – the child's brain is particularly sensitive and this is well documented in the medical literature.

Although the skull is a rigid structure,as the brain swells, the intracranial pressure does not rise at once because CSF and blood are displaced from the cranium, but when this mechanism can no longer cope, then the pressure within the skull rises rapidly and the brain is forced down into the foramen magnum – a situation known as "coning". At this stage there would be seizures and vomiting with the rise in intracranial pressure followed by changes to the pupils which become dilated and unresponsive, loss of consciousness and cessation of breathing. Blood pressure is often high at this stage. Brain death follows if steps to reduce the cerebral swelling are not taken immediately, as the intracranial pressure exceeds that of the blood supply.
Lucy's clinical course vividly illustrates this sequence of events.

To conclude and summarise, I believe that Lucy died of acute cerebral oedema leading to "coning" - the forcing of the brainstem down into the foramen magnum in the base of the skull.
She came into hospital somewhat dehydrated from vomiting which causes a loss of water and sodium chloride. There was additional loss from diarrhoea.
She was given an excess volume of intravenous water to replace these losses
I think that the excessive volumes of dextrose/saline (4%/0.18%) in the face of losses of electrolytes from vomiting and diarrhoea caused an acute serum sodium dilution which in turn caused acute brain swelling.
The brain swelling starts insidiously until intracranial compensatory mechanisms cannot cope, at which time intracranial pressure suddenly rises and the coning process begins. This is manifest by seizures, loss of consciousness, the pupils become dilated and unresponsive and there is cessation of respiration.
I think the cerebral oedema was exacerbated by the huge volume of saline given at the time of resuscitation.

exacerbated by excess administration of saline.

References.

Huskisson L Fluid balance: all aspects. In: Paediatric Anaesthesia. Eds: Sumner E, Hatch DJ. London Arnold 1999

Arieff AI. Postoperative hyponatraemic encephalopathy following elective surgery in children. Paediatric Anaesthesia 1998; 8: 1-4