Case 3. LC

I have examined the case notes of LC, including the post mortem report and the report provided by Dr Murray Quinn.

Dr Quinn's letter of 22-06-00 summaries the clinical course following Lucy's admission to the Erne Hospital on 12-04-00. I shall briefly outline the clinical events following her presentation to the Erne Hospital. Lucy was referred to the Erne Hospital by her general practitioner on 12.04.00. The history was of pyrexia, drowsiness, lethargy, floppiness and not drinking, and a diagnosis of a urinary tract infection was queried. The paediatric admission notes confirmed that Lucy had not been feeding well for the past 5 days with pyrexia and vomiting for the past 24 hours, and sleepiness for the past 12 hours. On examination, temperature was mildly elevated and she was noted to have prolonged capillary refill time. The plan was to encourage feeding, check urinalysis, take blood samples for full blood count, urea and electrolytes, glucose, C-reactive protein and blood culture and commence IV fluids after IV cannulation. She was admitted about 7.30 pm in the evening, and around 10.30 pm an IV line was inserted and she was commenced on intravenous fluids, 0.18% sodium chloride. From the nursing notes it appears that venous samples were taken at this stage (blood urea mildly elevated at 9.9 mmol/L and CO_2 reduced at 16 mmol/L.

At 15 minutes past midnight on 13.04.00 she had a large vomit, and at 2.30 am had a large soft bowel motion. At around 3 am the nurses were alerted by the mother, and she was reported to be rigid in her mother's arms. She was not cyanosed and pulse and respirations were recorded as satisfactory. A junior doctor was contacted, Lucy put on her side and oxygen therapy commenced. There was some smacking of her lips and twitching, and rectal diazepam 2.5 mg. was administered. This was followed by a large, watery, offensive stool. Blood pressure was elevated at this time at 144/113, but other observations were within normal limits. Around this time the intravenous fluids were changed to normal saline and run freely into the intravenous line. Around 3.20 am decreased respiratory effort was recorded, an airway inserted and bag mask ventilation commenced.

Intubation was carried out around 4 am but the notes in the chart state that heart rate and oxygen saturation measurements were satisfactory from the time of the respiratory arrest until intubation was carried out. Around this time pupils were noted to be fixed and unresponsive. She was transferred to the Intensive Care Unit in the Erne Hospital and subsequently to Paediatric Intensive Care Unit in the Royal Belfast Hospital for Sick Children. Brain stem tests were carried out in RBHSC, both were

negative and she was extubated at 1300 on 13.04.00. A post-mortem examination was carried out which showed extensive bilateral bronchopneumonia, swollen brain with generalised oedema and early necrosis, with relatively little congestion with some distension of large and small intestine with gas and clear fluid. Rotavirus was detected in stool samples sent from the Erne Hospital on 13.04.00.

The following comments have been made following careful examination of the nursing and medical records from the Erne Hospital, including the post mortem report, and the medical report from Dr Murray Quinn. They are necessarily limited to the information contained in the notes. It is apparent that Lucy's clinical deterioration was unpredicted, rapid and extremely distressing for all concerned. I appreciate that I may have missed some facts, and that my comments are made some time after the events had occurred.

Points

- Vomiting and fever are very common symptoms in young children. In most children, these symptoms are self-limiting and require only supportive measures such as attention to fluid balance, and antipyretic medication.

- Lucy was probably quite ill on admission. She had been off her food for 5 days, with fever and vomiting for 36 hours and drowsiness for 12 hours.

  Clinical examination as documented, was essentially normal, but she did have prolonged capillary refill time indicating a degree of shock.

  Investigation showed increased white cell count, (15,000x 10^9/L) mainly leucocytes, suggesting bacterial infection, urinalysis had protein ++ and ketones ++ and venous $C0_2$ was reduced (16 mmol/L suggesting hyperventilation. Urea was elevated (9.9 mmol/L). These results indicate moderate-severe dehydration with a degree of pre-renal failure. The low $C0_2$ suggests compensated metabolic acidosis (we do not have arterial or venous astrup results). The plan was to encourage feeding, and commence intravenous fluids after cannulation. Given the symptoms and signs, and the prolonged capillary refill time (>2 secs), it would be appropriate to give an immediate fluid bolus of up to 20ml/kg (N Saline, or less commonly, colloid) and then reassess. It was several hours after admission before intravenous fluids were commenced. The difficulty in obtaining intravenous access in young children, and toddlers in particular, is well recognised. The notes do not make clear the possible reasons for the delay in addressing the problem of restoration of circulatory blood volume.

- It is difficult to determine the nature of the episode at around 2.55 am although nursing records indicate some form of seizure activity. At the time respirations ceased, around 3.15am, pupils were fixed and dilated, and thereafter no spontaneous activity was recorded. (Repeat BM was elevated (13.4 mmol/L), BP elevated (144/113), but she was not bradycardic. Repeat U&E showed hyponatraemia (Na = 127 mmol/L), hypokalaemia (K 2.5 mmol/L), and urea had decreased to 4.9 mmol/L.

There are several possible explanations:

(i) Lucy had a febrile seizure (she was pyrexic and at an age when febrile seizures are common) which continued from 2.55am, leading to hypoxia and cerebral oedema. However most children who have febrile seizures suffer no long term sequelae and do not develop cerebral oedema, especially as there was a relatively short-time gap between the first episode (2.55am) and the respiratory arrest (around 3.15am).

(ii) She had a seizure like episode due to underlying biochemical abnormality. Initial sodium was 137 mmol/L, and potassium 4.1 mmol/L at 10.30 pm. At 3.00 am, and after administration of 0.18% NaCl, the repeat sodium was 127, and potassium 2.5.

Biochemical changes are often well tolerated and easily corrected with appropriate fluid replacement, although these results do show a change over a relatively short period of time.

(iii) The episode at 3.15 am was due to cerebral oedema and "coning". My impression from the notes is that Lucy never showed any signs of any recovery after 3.00 am and that this was a pre-terminal event, followed by respiratory arrest around 3.20 am when pupils were noted to be fixed and dilated. BP was elevated at 144/113 but she was not bradycardic. Although intubation did not occur until 4.00 am, nursing and medical records state that oxygenation was maintained and heart rate did not fall below 100.

I agree with Dr Quinn that the administration of rectal diazepam is very unlikely to have been a contributing factor. The dose was correct, and in any case most of the drug was probably expelled shortly after being given by the per rectal route.

- The fluid balance records between admission and the events at 3.00 am are incomplete. 0.18% saline was commenced at 10.30 pm, but the rate is not prescribed on the fluid balance sheet. My interpretation

of the chart is that she received 100 mls/hr 0.18% saline until around 3.00 am when the adverse episode occurred.

Once shock has been corrected with 20 mls/kg N saline (or colloid), APLS guidelines for a child with moderate/severe dehydration would be:-

Fluid deficit = 7.5% dehydration X weight (kg) X 10

i.e. = 750mls

Maintenance fluids (24hrs) = 1000mls

i.e. a total of 1750 to be given over 24hrs

= 70 – 80 mls/hr

The volume given, therefore, does not appear excessive. There is debate about the most appropriate fluid to use.

APLS guidelines; deficit should be replaced with normal saline and maintenance with 0.18% N saline.

For convenience the 2 fluids are often combined and given initially as 0.45% NaCl in 5% dextrose, and the regimen altered on the basis of blood result.

After the respiratory arrest at 3.15am, the fluids were changed to N saline. The clinical notes state that 500mls was given over the next hour.

A volume of 20mg/kg would be indicated in a "shock" situation, although measurements recorded at this time do not suggest circulatory compromise, and her urea had fallen to normal levels.

- There was little warning of the rapid deterioration at around 3.00am. There is nothing in the medical and nursing notes between admission and 3.00 am to indicate that medical and/or nursing staff were unduly concerned. Her temperature remained elevated (above 37.5°C) until 22.30, but not markedly so. She was said to be floppy at 19.30 and asleep at 23.30. BM at 20.30 was 3.6 mmol/L. Although active resusitation was commenced around 3.20am, there was never any response, and the fixed, dilated pupils almost certainly were an indication of severe brain pathology.

- There is the possibility that Lucy had an additional abnormality, which was not detected at post mortem examination. The fact that she had been well prior to this admission (apart from one episode of bronchiolitis) and that there were no documented symptoms or signs to suggest such an abnormality when she was admitted, make this less likely. However, the pathology report does state that she had extensive bilateral pneumonia, but could not confirm whether the pneumonia had preceded, or been caused by the sudden deterioration around 3 am.

Summary

This little girl was admitted to the Erne Hospital in April 2000 and had a respiratory arrest 8 hours later, from which she never regained consciousness. Subsequent results indicate that she had gastroenteritis due to rotavirus (she may also have had bronchopneumonia). Initial investigations indicate that she was quite ill on admission, with a degree of circulatory failure. There was a delay in implementing fluid resuscitation and there are deficiencies in the prescription and recording of volumes of fluids administered. The subsequent events which occurred about 8 hours after admission were likely to have been preterminal and on the basis of cerebral oedema and coning.