Medicolegal Report on Claire Roberts
D.O.B: 10th January 1987

By

Dr R M Bingham FRCA

This report has been compiled using the following documents:

- Photocopies of the clinical notes from Claire's admission to the Royal Hospital for Sick Children, Belfast on 21st October 1996.
- Statement compiled by Mr Alan Roberts following a meeting with Dr N Rooney, Dr H Stein, Dr A Sands and Professor Young
- Statement from Dr Heather Steen compiled on the 16th March 2005

BACKGROUND TO CASE

Claire Roberts was admitted to the A & E department of the Royal Hospital for Sick Children at 19.15hrs on the 21st October 1996 with a history of malaise for 1 day and hourly vomiting and drowsiness since the same afternoon.

Claire had a history of developmental delay and moderate learning difficulty and she attended Torbank Special School[1]. She also had hospital admissions for seizure activity between the ages of 6 months and 4 years. She was treated for these with anti-epileptic medication but this was discontinued when she had no further fits.

From the admissions notes it would appear that when well, Claire could construct meaningful sentences, walk, run and climb stairs unaided. She fed herself with supervision but needed help with dressing. It was noted that she favoured her left side.

On examination following her admission her temperature and pulse rate were normal but it was noted that her speech was slurred and that she was hardly speaking. Her pupils and fundii were normal and there was no neck stiffness[2]. There is a note that her muscle tone was raised and the left sided tendon reflexes were brisker than those on the right[3]. Later, on the ward this was reversed and it was also noted that she had "cogwheel rigidity" of the right arm and ankle clonus[4].

On admission to the ward there is a statement that she was "not responding to parents voice" but she was "responding to deep pain". There is no mention of Claire's state of hydration, which I assume means that there were no concerns. Certainly her heart rate was normal and increased heart rate is one of the earliest sign of dehydration.

A presumptive diagnosis of "viral illness" was made, blood samples[5] were taken and intravenous fluids were started at a rate of 64ml/hr.

On reassessment at midnight Claire was "slightly more responsive" and there were no signs of meningism so it was decided to observe her overnight and re-assess in the morning. The results of the blood tests were also available and were normal except for a raised white cell count[6] and a low serum sodium (132mmol/l).

The following day there was a ward round with Dr Sands where Claire's continued lack of responsiveness was noted with the comment "vagueness/vacant (apparent to parents)" and it was agreed to contact Dr Gaston; Claire's Community Paediatrician.

A diagnosis of "non-fitting status"[7] was made and a dose of diazepam[8] was prescribed.

Later the same day (16.00) Claire was reviewed by a neurologist who noticed that she appeared to have improved following the diazepam. It is noted that Claire withdrew from a painful stimulus

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[1] I think this is the correct name; the writing is difficult to read from the photocopy.
[2] Abnormal fundii (the optic nerves at the back of the eye) are associated with brain swelling. Neck stiffness is a symptom of meningitis.
[3] There is a reference here to a GP letter, which I do not have.
[4] All these are signs of abnormalities in the control the central nervous system (CNS) exerts over the peripheral nervous system. These could be longstanding, related to Claire's developmental delay or signs of CNS irritability related to the current illness.
[5] Full blood count, serum electrolytes, blood group and antibody screen and viral titres.
[6] This is consistent with an infective process.
[7] A condition in which there is epileptic electrical activity in the brain but no muscle twitching.
[8] A tranquillising drug which is an effective anti-epileptic.

and opened her eyes to voice prompts but did not talk. On examination there were "reduced movements right side?" and her reflexes were symmetrically brisk. This was interpreted as "probably long standing but this needs to be checked with notes". Her fundii were again normal. The summary states "The picture is of acute encephalopathy most probably postictal in nature[9]. I note no biochemistry profile". A loading dose of the anti-epileptic drugs phenytoin and midazolam were prescribed.

Claire was re-assessed by the neurologist at 17.00 following the drugs. Her level of consciousness had deteriorated so that she only responded to pain. It was decided to start anti-biotic and anti-viral drugs in case Claire had meningo-encephalitis[10] although this was not felt to be a likely diagnosis. Another anti-convulsant (sodium valproate) was also prescribed.

The nursing notes and observations at this time also note that she was only responding to painful stimuli. At 21.00 an episode of screaming and drawing up of arms was noted on the "Record of Attacks Observed" and at this time a reduction in the GCS[11] from 8 to 6 occurred.

At 23.30, the results from a previous blood test were available. The serum sodium level was extremely low (121mmol/l). The SHO contacted his Registrar who suggested reducing the rate of fluid administration to 2/3 of its current value. Urine was sent for osmolality testing[12].

At 03.00 Claire had a sudden respiratory arrest and developed fixed and dilated pupils[13]. She was resuscitated with mask ventilation with oxygen followed by tracheal intubation[14]. She was transferred to the ICU where she was put on a mechanical ventilator given mannitol[15] and an infusion of dopamine[16]. Examination revealed fixed and dilated pupils, bilateral papilloedema[17] and no response to painful stimuli. A CT scan showed severe swelling of the brain and no focal abnormalities.

Brain stem death tests at 06.00 showed that the criteria for brain stem death were fulfilled. Serum sodium levels at the time of the brain stem testing were 133mmol/l on the intensive care unit blood gas analyser and 129mmol/l on the laboratory sample.

A second set of tests performed at 18.25 confirmed brain stem death and ventilation was discontinued.

OPINION ON CAUSATION

On admission, Claire's initial diagnosis was of an unspecified "viral illness" but the slight improvement in her level of consciousness led the team to a plan of observation and expectant management. It appears to me that there was difficulty in deciding how much of Claire's condition was related to her underlying developmental delay and how much was due to the acute illness; this is highlighted by the comment "vagueness/vacant (apparent to parents)" noted following the ward round on the morning after her admission. This difficulty may have contributed to the delay in recognition of the serious nature of her condition.

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[9] Brain injury (probably temporary) as a result of the status epilepticus.
[10] CNS infection.
[11] Glasgow Coma Score; an internationally accepted measure of depth of coma – the lower the number the deeper the coma.
[12] A measure of its concentration as a cause of low sodium is the production of urine of inappropriately high concentration.
[13] A sign of severe swelling of the brain with an increase in the pressure within the skull.
[14] A tube passed into her lungs to aid mechanical ventilation.
[15] A drug which reduces brain swelling
[16] A drug which increases blood pressure in an effort to improve the blood flow to the swollen brain
[17] Swelling of the fundii or optic nerves, which had previously been normal.

The admission diagnosis is reasonable however and acute encephalopathy (viral or ictal) is a likely cause of the presenting illness. Although the serum sodium was initially low and officially defined as hyponatraemia[18] I think it is unlikely that this was the cause of her presenting symptoms. Serum sodium levels in this range are reasonably common in children admitted to hospital in acute illness[19] and symptomatic hyponatraemia is usually associated with much lower sodium levels than this. In addition hyponatraemia is known to accompany both acute encephalopathy and nausea and vomiting.

The decision to start Claire on intravenous fluids was reasonable, as she did not appear well enough to hydrate herself. The choice of fluid was 0.18% saline with 5% glucose, which has been the standard fluid, used for maintenance fluid therapy in children for 50 years. The volume prescribed was in line with widely used recommendations for full maintenance fluid therapy. There were, however reasons why Claire may have required fluid restriction. These include a low level of metabolism as she had reduced consciousness and a possibly reduced urine output due to the secretion of anti-diuretic hormone[20], which often accompanies both encephalopathy and nausea and vomiting. A record of poor urine output could confirm this but in fact there are several notes of her passing urine recorded on the fluid charts and on one occasion it was noted that there was a large volume.

Despite the fact that Claire may have been receiving more fluid than she required, it is not clear why her serum sodium fell to such a low level. The intravenous fluid volume charted in the notes would not be sufficient to account for this, even in the absence of any water excretion by the kidneys. It is possible she was receiving water by other means such as drinking but there is little record of this in the ward notes and her reduced conscious level would make it unlikely. Another possibility is that she was passing urine with very high sodium content but although there is a record of a urine osmolality sample[21] being sent at 23.30 on the 22nd October I have not been able to find the results. Finally, it is possible that the result was inaccurate as the sodium levels in the ICU at 06.00 on the 23rd October were much higher (133mmol/l (blood gas analyser) or 129mmol/l (laboratory)).

Assuming the result was accurate and irrespective of the mechanism for the fall in serum sodium, it is likely that this was the cause of the deterioration in Claire's condition on the evening of the 22nd October; a sodium level of 121mmol/l is known to cause brain swelling and convulsions which can progress to respiratory arrest and death. It is not however possible to completely exclude the chance that the sodium level was inaccurate and that acute encephalopathy was involved in - or even central to - the deterioration.

IN CONCLUSION:

The understanding of complex medical problems is always much easier with hindsight; in particular in this case there has been much recent publicity in both the lay and medical press which has led to a better appreciation of the dangers of hyponatraemia in children and helped to clarify the cause of this tragedy. Much of this information has only been available in the last five years.

I feel that Claire's initial diagnosis and management was reasonable. A viral illness was a common and likely diagnosis and although her serum sodium was low it was not excessively so. Her fluid prescription was in line with the practice of the time and although current guidance would be to use fluid with higher sodium content in this situation, this advice did not exist in 1996.

[18] A serum sodium level of less than 136mmol/l
[19] Pediatrics 2004: 113;1279-1284
[20] A hormone which reduces the amount of water the kidneys excrete.
[21] A measurement of the electrolyte content of the urine

I think there was also confusion about Claire's usual neurological status, which complicated her evaluation and led to an underestimate of the severity of the condition.

The initial and subsequent anti-convulsant treatment was logical, given the working diagnosis and it is unlikely it would have worsened the consequences of hyponatraemia although it may have masked the symptoms.

The hyponatraemia was probably an associated feature of Claire's condition rather than the primary illness. It was most likely to have been a result of the combination of raised levels of anti-diuretic hormone together with the intravenous infusion of fluid with low sodium content although the volumes infused do not fully account for the sodium becoming so low.

I think it is most likely that hyponatraemia was the cause of the neurological deterioration on the evening of the 22nd October culminating in the respiratory arrest at 03.00 on the 23rd. It is not, however possible to completely exclude the possibility that the serum sodium result was an isolated artefact and the deterioration was due to acute encephalopathy.

It is difficult to be certain whether this tragedy could have been prevented but assuming hyponatraemia was the cause, it is likely that identification of a low sodium level when the absence of a biochemical profile was noted, followed by the institution of a fluid restriction regime, would have ameliorated its consequences. It is also possible that aggressive treatment at 21.00, when Claire's coma score reduced from 8 to 6, may have been effective. Although the measures taken at 23.30, when the sodium result was available, were of the correct type they were too little and too late.

Statement

I understand that it is my duty to help the Court with those matters, which are within my expertise, and In relation to which my advice has been sought, and I have complied with that duty.
I believe that the facts I have stated in this report are true and that the opinions I have expressed are correct.

Dr Robert Bingham
Consultant Paediatric Anaesthetist

14 April 2005

Short CV of Dr R M Bingham

Current appointment: Consultant Paediatric Anaesthetist; Great Ormond Street Hospital NHS Trust. London (1985-present)

Relevant positions held:

Chairman; Resuscitation Council (UK). (2000-2003)

Vice-Chairman; Resuscitation Council (UK). (1998-2000)(2003-2004)

National Director; Paediatric Advanced Life Support courses (1993-1999)

Clinical Director; of Anaesthesia and Theatres GOS Trust (1997-2000)

Lead Clinician for Anaesthesia GOS Trust (1994-1997) and 2001

Medical Advisor (paediatrics); London Ambulance Service (1995-present)

Primary Examiner; Royal College of Anaesthetists (1996-present)

Royal College of Anaesthesia representative; Paediatric and Congenital Cardiac Services review (2001)

Council Member; Association of Paediatric Anaesthetists of Great Britain and Ireland (2004-present)

Publications:

Book Chapters

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