Form 20

CORONERS ACT (NORTHERN IRELAND) 1959

Deposition of Witness taken on the , at inquest touching the death of LUCY CRAWFORD, before me MR J L LECKEY Coroner for the District of GREATER BELFAST as follows to wit:-

The Deposition of DR J G JENKINS

of who being sworn upon HIS oath, saith

I Dr J G Jenkins MD FRCP FRCPCH FRCPI Senior Lecturer in Child Health and Consultant Paediatrician have been asked to prepare this report for Mr J L Leckey Coroner for Greater Belfast. This report is based on material made available including hospital notes relating to admission of this child to Erne Hospital in November 1999 and April 2000. The first admission was for bronchiolitis and is not directly relevant to the problems, which occurred during the subsequent admission in April 2000. At that time Lucy was admitted to hospital with a history of fever and vomiting for 36 hours associated with drowsiness for 12 hours. There was no history of cough and her chest was clear on examination. The history and clinical findings has been thought by the GP to suggest urinary tract infection but it was felt on admission that this was more likely to be a viral illness. Initial blood tests were performed and Dr Malik attempted to commence IV fluids but was unable to do so and so called Dr O'Donohoe (the Consultant Paediatrician). IV fluids were then commenced. The nursing notes record observations at 19.30, 22.30 and 23.30 during which time the high temperature gradually came down to 37.4 There is then a gap in the observation sheet with no apparent entry until an episode of sudden collapse which occurred around 3.00 am. It appears that the mother called nursing staff as Lucy had passed diarrhoea and then became rigid. Dr Malik was called and felt that this could be a febrile convulsion so administered Diazepam. He discussed the case with Dr O'Donohoe who then came directly to hospital arriving at 3.20 am. At around this time Lucy's condition further deteriorated as she stopped breathing and

required respiratory support. The on-call Anaesthetist was called at 3.40 am and Dr Auterson arrived shortly after 3.50 am and assisted with the resuscitation including intubation and transfer to the Intensive Care Unit in the Royal Victoria Hospital for Sick Children later on the same morning. The doctors involved seem clear that there was no episode of cardiac arrest or circulatory instability during this period but it was noted that the pupils became fixed and dilated and did not respond to ventilation or the administration of Mannitol. Subsequently tests in Belfast revealed evidence of the brain stem death and post mortem examination was performed. This showed bronchopneumonia and cerebral oedema with evidence of herniation of the brain. The Pathologist is unable to comment as to whether the bronchopneumonia had been present from admission to Erne Hospital or had occurred in association with the collapse and resuscitation. Further specimens have shown rotavirus infection suggesting that the initial admission was likely to be due to rotavirus gastroenteritis. Urine cultures showed no significant growth.

COMMENT

This child's admission to Erne Hospital was very typical of gastroenteritis in this age group. This is often associated with the high temperature and vomiting with or without diarrhoea and young children can become very unwell. The standard treatment is to administer fluids either orally or (if there is significant dehydration or vomiting) by the intravenous route. The solution used is one which is commonly used in Paediatric practice to provide maintenance fluids in these circumstances as it replaces small amounts of electrolytes but also gives Dextrose which is required by young children who are unable to take calories orally during the acute phase of the illness. Initial physical findings were suggestive of poor peripheral circulation with delayed capillary refill time >2 seconds. The GP noted that the mucosae were moist but there is little specific detail in the admission note regarding evidence of dehydration. However, the urea was 9.9 which is slightly elevated suggesting a mild degree of dehydration but with normal electrolytes at that time. This would again be very typical

of the condition and would not normally indicate anything other than appropriate fluid replacement with careful monitoring and nursing observation. However, in this situation the intravenous fluids for replacement of any losses should contain a higher content of sodium (eg "normal saline" – 0.9% NaCl – sodium chloride). In these circumstances it is always very difficult to understand an episode of sudden collapse. Sudden onset of convulsions is most commonly due to high temperature in young children and this was considered. However, the features were not typical and the temperature had in fact improved since admission. It is unclear as to what alternative diagnoses were considered at this time but the blood test for electrolytes was appropriately repeated immediately. This showed a significant fall in sodium for 137 to 127 and in potassium from 4.1 to 2.5, together with an increase in glucose from 4.5 to 10.9. These changes do raise the question as to the fluid management in the period from insertion of the IV line at 23.00 to collapse at around 3.00 am. Unfortunatly there appears to have been confusion between the staff involved as to the fluid regime ordered by the consultant. In addition it is difficult to interpret the record made by nurses on the fluid balance chart and no totals have been calculated for this period. It will be most important to determine from the staff involved exactly how much of each type of fluid was given at each stage throughout this time period, and following the change of fluids to normal saline through until the child arrived in the Paediatric Intensive Care Unit in Belfast. Other aspects of this tragic case demonstrate a rapid and effective response by the medical staff concerned. In particular both the Consultant Paediatrician and Consultant Anaesthetist appear to have been available within a very short time period of being called and to have done their best in the difficult circumstances involved in caring for a child of this age in an adult intensive care setting for stabilisation and transfer in the absence of a Paediatric transfer service in Northern Ireland. Over recent years concerns have increasingly been expressed regarding the use of 0.18% saline in Dextrose as a standard solution for intravenous use in young children, and a number of cases of symptomatic hyponatraemia have been identified, some resulting in death or cerebral damage. It has been suggested that a

more appropriate solution would contain a higher level of sodium and this has recently been the subject of discussions involving the Department of Health, Social Services and Public Safety and production of guidelines. However, it must be emphasised that this is a recent development and that at the time of Lucy's admission to hospital many Paediatric Units were continuing to use the solution which was initially given in this case. Although the sodium level of 127 is not in itself usually associated with severe problems, it is likely to be the rate at which the sodium falls rather than the absolute level, which can cause problems in this setting. While no definite conclusions can be drawn regarding the cause of the child's deterioration and subsequent death, there is certainly a suggestion that this was associated with a rapid fall in sodium associated with intravenous fluid administration causing hyponatraemia and cerebral oedema. In these circumstance clear documentation is needed regarding the fluid type and rate prescribed, together with clear records as to the exact volumes of each fluid which were in fact received by the child throughout the time period concerned.

TAKEN before me this

Coroner for the District of Greater Belfast

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Their has not appear to have happened.
A formula should be devised by the
doctor or to the fluid require. The form
should be written down though notwithst
the actual calculation, I do not believe
is always necessary. I agree it is mandat
to write a proper fluid prescription
designated that stating what fluid is to
given and at what rate. I agree that the
care Lucy received was sub-standard. "I
solution is completely wrong to make up
deficits. That had not been well-clear
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of children at that time. I prepared my
report on 7th March 2002. At the time
Lucy's death 18 solution was commonly u

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for both maintenance & depletion in some circumstances. At 2000 there were different views expressed in relevant books. For Lucy I would have given 9 solutrin or a bolus of 100 ml followed by 18 solutrin. There is no other explanation for what happened to Lucy than excessive dilute fluid. It was an extremely grave state of affairs that the rate of infusion was not written down. The doctor who decides on the fluid regime is responsible for writing up the prescription - fluid to be used & the rate of infusion. Normally nurses record fluid details on a separate sheet. In this case their record is difficult to interpret. I agree that the rate & fall of the sodium level is as important or more important than the sodium level. I agree that the rapid fall in sodium was the cause of the cerebral oedema in Lucy. I base that on the other investigations referred to by Dr. Hamilton.

Mr Good: I was a member of the Working Party set up by the Dept. There was a literature search on a world-wide basis. The literature did not provide clear guidance. There was a range of approaches to a child with mild dehydration. The aim of the Working Party was to provide clarity - guidelines to be applied in different cases. One "absolute" definition was not possible. We found 18 solutrin was commonly used & there was ignorance of all its implications. I agree that the level of dehydration for Lucy was 5-7.5%. I would

TAKEN before me this 19th day of February 2004.

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have assessed Lucy for circulatory compromi – an initial bolus of 100 ml of solution, followed very possibly by 18 solution. If there was significant ongoing losses extra amoun of 9 solution. I am not surprised that 18 solution was used at the time. The regime of Dr Evans at para 41 reflects his personal approach. I believe it is a different approa to the problem – a simpler solution. His is averaging approach. On the available inform I have no criticism of the resuscitation procedures. Any further losses would have required careful assessment. John [signature]

P.T.C

013-032-122

TAKEN before me this 19th day of February 2004

Coroner for the District of Greater Belfast

I agree that the cause of death should be 1(a) Cerebral Oedema (b) Acute Dilutional Hyponatraemia (c) Excess Dilute Fluid II Gastroenteritis.

Mr Fee: I cannot say if the interval between admission and the successful insertion of the IV line was too long. The doctor should make an assessment of the level of dehydration. I saw nothing in the notes to show that this had been done. It is important that a formal assessment of the degree of dehydration is made. That does not appear to have happened. A formula should be devised by the doctor as to the fluid required. The formula should be written down though not always the actual calculation. I do not believe that is always necessary. I agree it is mandatory to write a proper fluid prescription on a designated chart stating what fluid is to be given and at what rate. I agree that the care Lucy received was sub standard. No 18 solution is completely wrong to make up deficits. That had not been well clarified in the paediatric literature at the time of the death of Lucy. There was a poor understanding of this area in the treatment of children at that time. I prepared my report on 7th March 2002. At the time of Lucy's death No 18 solution was commonly used for both maintenance and deficits in some circumstances. At 2000 there were different views expressed in relevant texts. For Lucy I would have given 9 solution as a bolus of 100 mls followed by 18 solution. There is no other explanation for what happened to Lucy than excessive dilute fluid. It was an extremely grave state of affairs that the rate of infusion was not written down. The doctor who decided on the fluid regime is responsible for writing up the prescription - fluid to be used and the rate of infusion. Normally nurses record fluid details on a separate sheet. In this case their record is difficult to interpret. I agree that the rate of fall of the sodium level is as important or more important than the sodium level. I agree that the rapid fall in sodium is the cause of the Cerebral Oedema in Lucy. I base that on the other investigations referred to by Dr Hanrahan.

Mr Good: I was a member of the Working Party set up by the Department. There was a literature search on a worldwide basis. The literature did not provide clear guidance. There were a range of approaches to a child with mild dehydration. The aim of the Working Party was to provide clarity – guidelines to be applied in different cases. One absolute definition was not possible. We found No 18 solution was commonly used and there was ignorance of all its implications. I agree that the band of dehydration for Lucy was 5-7.5%. I would have assessed Lucy for circulatory compromise – an initial bolus of 100 ml No 9 solution followed very possibly by No 18 solution. If there were significant ongoing losses extra amounts of No-9 solution. I am not surprised that No 18 solution was used at the Erne. The regime of Dr Evans at paragraph 41 reflects his personal approach. I believe it is a different approach to the problem – a simpler solution, his is an

averaging approach. On the available information I leave no criticism of the resuscitation procedures. Any further losses would have required careful assessment.