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Director
Alphy Maginness
Assistant Directors
Donna Scott
Hilary Wells

Our Ref: INQ T50/9/15.ds

Date: 23 January 2003

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Dr J G Jenkins MD FRCP FRCPCH
Consultant Paediatrician
United Hospital H&SS Trust
Antrim Area Hospital
45 Bush Road
ANTRIM
BT41 2RL

() Get hyponat. file.
(? AOC article re JG's salm)*

Dear Sir

RACHEL FERGUSON (DECEASED)

I refer to the above matter and enclose herewith copy report, which has been received from Dr Declan Warde, the Consultant Paediatric Anaesthetist retained to advise the Trust. I would be gratefully obliged if you could consider Dr Warde's report and provide me with any further comments which you have which might assist the Trust. In view of the imminent date of the hearing of this Inquest, I would be gratefully obliged to receive any further comments which you may have as a matter of urgency. Please do not hesitate to contact me if you wish to discuss this matter further.

Yours faithfully

June Connell, Gerald W. Alinden

June 2001 first alerted locally.

D Scott.

D SCOTT
Assistant Director of Legal Services

Direct Line [Redacted]
E-Mail Address - scott@ [Redacted]

Enc

*5/2/03
after Dr. Sumner
- ? time 9.30*

*check re ? fax
today + then post. ⁵ faced posted 29/1/03*

Thank you

JJ 29/1/03.

Each department providing inpatient care for children should involve medical staff with relevant paediatric experience in the development of local guidelines and policies for fluid management, and in the management of individual patients who develop electrolyte abnormalities.

9ds43zs



Solicitors

- Elizabeth Adair
- Wendy Beggs
- Derek Burgoyne
- Marie-Claire Carey
- Ann Cassidy
- Eileen Finnegan
- Brian Greenaway
- Audrey McClean
- Carol McClean
- Siobhan McCrory
- Kathryn Minnis
- Jonathan Taylor
- June Turkington

Ⓟ 27/1/03

MEDICOLEGAL REPORT

Re:, deceased.

Prepared for:

By:

January 2003

My name is and I am a Consultant Paediatric Anaesthetist with an interest in Paediatric Intensive Care.

I have been a Consultant in the since 1986 and have served at various times since then as Chairman of the Department of Anaesthesia and Director of the Paediatric Intensive Care Unit in that institution. I am the author of several articles and book chapters on Paediatric Anaesthesia / Intensive Care. I served as representative.....
.....
.....

In preparing this report at the request of, I have carefully perused all the medical and nursing notes and statements presented to me, together with the reports of Drs

I believe that the facts I have stated in this report are true and that the opinions I have expressed are both correct and within my area of expertise.

..... was born on and died on

Having been fit and well until earlier that evening, she was brought to the Accident and Emergency Department of Hospital at 8 p.m. on She was complaining of sudden-onset and increasingly severe abdominal pain since 4.30 p.m. approximately. She had eaten dinner at 5.10 p.m. but had no appetite subsequently. She complained of nausea but had not vomited. Her temperature was normal. Her weight, perhaps estimated, was 26 kg. Clinical examination, which revealed right iliac fossa tenderness, guarding and rebound tenderness was consistent with a diagnosis of acute appendicitis.

Preoperative haematology and biochemistry were normal. In particular, her serum sodium was 137 mmol.l⁻¹ (normal laboratory range for Hospital 135 -145 mmol.l⁻¹). Urinalysis showed proteinuria +++.

....., obtained consent for surgery. At 8.20 p.m. was given Cyclimorph 2mg by intravenous (i.v.) injection for relief of pain. She was subsequently admitted to Ward ., kept fasting and commenced on i.v. fluids to maintain adequate hydration prior to surgery. initially prescribed Hartman's solution. He later changed this to Solution 18 (sodium content 30 mmol.l⁻¹) at 's request after she informed him that his initial prescription was not in keeping with common practice on the ward. Solution 18 was infused at 80 mls/hour until went to the Operating Theatre at which point in time it was discontinued, 60 mls having infused.

No premedication was administered. Consent for rectal analgesia was obtained from’s mother in the Operating Theatre area. Anaesthesia was induced at 11.30 p.m. approximately. Intravenous Hartman’s solution was commenced prior to induction. The exact amount infused in theatre is in some doubt. Dr’s retrospective note of on the anaesthetic record indicates that this was 200 mls while her statement of refers to a volume of 300 mls. The infusion was discontinued prior to’s return to Ward .. The anaesthetic and surgical techniques were routine and surgery proceeded uneventfully. was somewhat slow to awaken afterwards, presumably because of opioid administration, but was awake by 1.15 a.m. on

Following her return to Ward .., the Solution 18 infusion was recommenced at 80 mls/hr. Later on was noted to be afebrile and free of pain. She vomited at 8 a.m. and had a large vomit at 10 a.m., at which time she also passed urine (volume not measured). She vomited “++” at 1 p.m. and again at 3 p.m. At 9 p.m. she vomited coffee grounds “++”, had three more small vomits at 10 p.m., a small coffee ground vomit at 11 p.m., and a “mouthful” of vomit at 00.35 a.m. on June 9th. She had been given i.v. injections of the anti-emetics Ondansetron and Cyclizine at 6 p.m. and 10.15 p.m. respectively. The vomitus volume was not measured and there was no nasogastric tube in place. At 9.15 p.m. noted that her colour was “flushed to pale” and that she was complaining of headache.

Throughout the day the prescribed Solution 18 was infusing. I have calculated from the fluid balance chart that received 1840 mls from the time she left recovery until midnight on, and a further 320 mls between that time and 4 a.m. on, a total of 2160 mls. There is no record of any urine output other than at 10 a.m. does not appear to have taken any oral fluids or solids although, Surgical Senior House Officer had been happy for her to have small amounts of clear fluids orally from morning onwards.

At 3 a.m. June 9th was informed by that was fitting. She asked, who was in the ward, to attend urgently. Rectal and i.v. diazepam were administered, and were successful in controlling the seizure. Oxygen was administered via a face mask.’s vital signs and airway were satisfactory. Dr suspected an electrolyte abnormality as a likely cause of the fit and asked Dr, Surgical Pre-registration House Officer, to send blood to the laboratory for urgent analysis for electrolyte profile, calcium, magnesium and full blood count. Prior to obtaining the electrolyte results he went to the Neonatal Intensive Care Unit to discuss the scenario with Dr, Paediatric Second Term House Officer. During their discussion he was bleeped and informed that’s condition had deteriorated. Dr went to Ward .. The electrolyte results were now available and she noted that the sodium was 119 and the potassium 3 mmol.l⁻¹. She asked the Surgical Junior House Officer to urgently repeat the electrolytes and to send blood for culture and gas analysis. On clinical examination she found that looked very unwell, was unresponsive with dilated and non-reacting pupils and that her breathing sounded “rattly”. Her oxygen saturation was normal and her heart rate 160/minute. She had a petechial rash around her face, neck and upper chest, her trunk appeared flushed and her limbs were floppy. She

spoke to Dr, Consultant Paediatrician, by telephone and asked him to come to the ward immediately. She asked Dr to come and assist her – he inserted a second i.v. line and gave i.v. antibiotics in view of the petechial rash. Shortly afterwards the arterial oxygen saturation fell to 70% and became apnoeic. Dr commenced bag and mask ventilation and the Anaesthetic Registrar (.....) was fast-bleeped at 4.00 – 4.15 a.m. He arrived quickly and intubated without the need for any drugs. The second electrolyte analysis revealed sodium 118 and magnesium 0.59 mmol.l⁻¹. Intravenous fluid therapy was altered to 0.9% sodium chloride at 40 ml/hr and was given intramuscular magnesium sulphate because of her low magnesium level.

At 5.30 a.m. approximately she was brought to the X-Ray Department for an emergency CT Brain Scan. The examination revealed evidence of cerebral oedema. There was a suggestion of associated subarachnoid haemorrhage although this was subsequently considered to be simply secondary to reduced brain density. A repeat examination was performed to outrule possible subdural empyaema.

Rachel was transferred to the Intensive Care Unit for continuing care and later that morning to the Paediatric Intensive Care unit at the She died at 12.09 p.m. on June 10th 2001.

An autopsy was performed by Drs and on The major findings were of cerebral oedema and diffuse hypoxic ischemic necrosis in the cerebral cortex. Specialist opinion was sought from as to the likely cause of the cerebral oedema. It was concluded that died from cerebral oedema secondary to hyponatraemia.

SUMMARY AND COMMENTS

..... was a previously fit and healthy girl who was admitted to hospital suffering from acute mild appendicitis. Following appendicectomy, she experienced severe and protracted vomiting despite the administration of anti-emetics. During this time she was receiving Solution 18 intravenously. Some 27 hours postoperatively she developed seizures secondary to acute cerebral oedema, itself secondary to acute hyponatraemia. She died approximately 60 hours following surgery.

Vomiting following appendicectomy is very common. Causes include administration of opioid drugs, traction on the peritoneum during surgery and side-effects of certain anaesthetic agents e.g. nitrous oxide. However vomiting as severe and sustained as that

→ experienced by is rare – it is not clear why this occurred. It may be that in the later stages rising intracranial pressure was a contributory factor. Vomit contains 70-100 mmol.l⁻¹ of sodium – this is less than that contained in plasma. However, if the sodium and other electrolytes lost through vomiting are replaced with fluids containing minimal electrolytes, such as Solution 18, , the net effect is sodium depletion with resultant hyponatraemia.

The “Syndrome of Inappropriate Antidiuretic Hormone secretion (SIADH)” has long been known to be associated with stress (e.g. anaesthesia and surgery), vomiting and pain. It leads to accumulation of fluid in the extravascular space and inhibition of excretion of excess free water – this syndrome is the most likely explanation for’s apparently low urine output in the postoperative period. While accurate “fluid balance” (volume administered by all routes vs volume excreted by all routes) records are not available, I strongly suspect that was in significant “positive balance” (administered fluid in excess of that excreted) by the time she became acutely ill. In addition, the administered fluid (Solution 18) was low in sodium whereas much of that excreted (vomit) had a significantly higher concentration. The inevitable end-result was acute hyponatraemia.

Administration of low-sodium solutions to children in the perioperative period is not uncommon. Their use, I believe, stems largely from the fact that it has been known for many years that sodium excretion in the presence of sodium loading is far less efficient in infants and young children than in adults. “Ward policies” regarding i.v. fluid administration in children’s wards were developed, in part at least, to ensure that children were not given too much sodium, which can itself lead to major problems. Unfortunately such policies rarely took maturation of body organs with age into account. In reality, assuming appropriate volumes were given, the perioperative fluid and electrolyte requirements of, a previously healthy nine year old child, were far closer to those of a full-grown adult than to those of an infant or young child. In recent years there has been a steady move away from the perioperative use of such hypotonic fluids in children. There are a number of suitable alternatives available including 0.45% sodium chloride (“half-strength saline”, sodium content 77mmol.l⁻¹) in 2.5% or 5% glucose

Excess gastrointestinal losses in the postoperative period should be replaced with 0.9% sodium chloride (normal saline) with added potassium. If a hypotonic fluid such as Solution 18 is used for maintenance requirements, this necessitates the administration of two different i.v. fluid solutions simultaneously, which may be difficult. An acceptable alternative approach is to use a single fluid with intermediate electrolyte content e.g. Ringer’s Lactate or Hartman’s Solution (sodium 130 mmol.l⁻¹) for all perioperative requirements with added glucose and electrolytes as indicated by regular measurement of blood glucose and serum electrolytes. I note that prescribed Hartman’s Solution preoperatively but that this was not administered because of ward policy, and also that the anaesthetist used this fluid in the operating theatre.

Appropriate fluid and electrolyte management in the postoperative period in a patient with abnormal losses cannot, in my opinion, be achieved without electrolyte

measurement and accurate estimation of fluid balance. Gastrointestinal losses could have been measured either by passage of a nasogastric tube to drain the stomach contents, or by ensuring that vomited into a container with volume markings. Urinary output could have been measured by bladder catheterisation or other means.

In my opinion, died as a result of developing acute cerebral oedema secondary to acute hyponatraemia, which was itself caused by a combination of severe and protracted postoperative vomiting, SIADH, and administration of intravenous fluid with a low sodium content. The relative contribution of each of these factors is impossible to quantify. Many cases of hyponatraemic encephalopathy occur because initial non-specific symptoms such as nausea, vomiting or headache are ignored; an alternative view is that many cases occur because the initial symptoms are non-specific. Nevertheless, a high degree of vigilance, accurate fluid balance recording and regular electrolyte evaluation in postoperative patients with these symptoms may help prevent future tragedies.

REFERENCES

Viliunas V. Postoperative hyponatraemia. WFSA Distance Learning Paper No: 19 (reprinted from Australian Anaesthesia 1998)

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

Liu L. Fluid management. In: A practice of Anesthesia for Infants and Children, 2nd Ed. W B Saunders Co., 1992

ADDITIONAL COMMENTS

.....'s medical management from the time she began fitting was, in my opinion, in most respects entirely appropriate. I believe that many doctors of Dr 's relative lack of seniority would not have suspected from the outset that an electrolyte abnormality was the root cause of the problem. One could question why, upon receipt of the initial electrolyte results (revealing sodium 119 mmol.l^{-1}), Dr did not immediately alter the i.v. fluid therapy to 0.9% sodium chloride but instead asked for a repeat estimation. Whether or not this would have made a difference to the ultimate outcome we do not know, but it may have been beneficial. Some would argue that faced with a symptomatic patient with acute severe hyponatraemia it would have been appropriate to be more aggressive and to commence treatment with hypertonic (3%) sodium chloride combined with a diuretic such as frusemide. However this solution may not have been readily available and once again one can only speculate as to the possible effect.

results available 04.30
changed to N Sahi 04.30

(Dr. Date's report
- called 04.15-04.30
arrived < 5 mins later)

[Handwritten signatures and notes in purple ink, including a large bracketed signature and several smaller signatures with illegible text.]