Form 20

CORONERS ACT (NORTHERN IRELAND) 1959

Deposition of Witness taken on Tuesday the 25th day of April 2006 at inquest touching the death of CLAIRE ROBERTS, before me MR J L LECKEY, HM Coroner for the District of GREATER BELFAST as follows to wit:-

The Deposition of Dr Brian Herron

of

who being sworn upon his oath, saith

On the instruction of HM Coroner, Mr J L Leckey, LLM - I Dr Brian Herron, Department of Neuropathology, Institute of Pathology, Grosvenor Road, Belfast, Northern Ireland, carried out an examination in relation to the late Claire Roberts. I now produce a copy of my report as Exhibit CR.

TAKEN before me this 25th April 2006

Senior Coroner for Northern Ireland

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DEPARTMENT OF NEUROPATHOLOGY
AUTOPSY REPORT

Autopsy No.: NPPM 114/96

Name :	ROBERTS, Claire		
Age :	9 1/2	Hospital No.:	328770
Sex :	F	Hospital :	R.B.H.S.C.
Pathologist :	Dr. Herron	Ward :	I.C.U.
Clinician :	Dr. Webb/ Dr. Steen	Date of Admission :	22/10/96
Date of Necropsy :	24/10/96	Date of Death :	23/10/96
Time of Necropsy :	11.30 am	Time of Death :	6.25 hrs
Restrictions :	Brain only		

ANATOMICAL SUMMARY — CODES

History of recent diarrhoea and vomiting, cerebral oedema (brain weight 1606 g), brain stem necrosis. Subacute inflammation meninges in perivascular space.
History of epileptic seizures since 10 months of age. Neuronal migration disorder.

CLINICAL SUMMARY

She was well until 72 hours before admission. She had visited her cousin who had vomiting and diarrhoea. She had similar symptoms and 24 hours prior to admission started to vomit. Her speech became slurred and she became increasingly drowsy. She was felt to have subclinical seizures. She was treated with rectal Diazepam, intravenous Phenytoin and intravenous Valproate. She also had Acyclovir and Cefotaxime. Her serum sodium dropped to 121 and there was a query of inappropriate ADH secretion. Her fluids were restricted but she had respiratory arrest at 3 am on 23/10/96. She was intubated and transferred to intensive care where a CT scan showed cerebral oedema. Brain stem criteria was fulfilled at 6 am.

In her past history she had iatrogenic epilepsy since 10 months and mental handicap.

BRAIN DESCRIPTION

The fixed brain weighs 1606 g. There is no cortical venous thrombosis and there is no meningeal exudate. There is symmetrical brain swelling with effacement of gyri. There is uncal prominence but no necrosis.

On sectioning of the brain the presence of diffuse brain swelling is confirmed. There is no evidence of cortical necrosis, either laminar or focal. There is white matter swelling with effacement of the IIIrd ventricle but no evidence of shift at the midline. The paraventricular structures including the mammillary bodies show no evidence of necrosis. There is no basal ganglia or diencephalon lesion. On sectioning of the brain stem there is no evidence of brain stem haemorrhage to suggest Leigh's disease. The cerellum is unremarkable.

HISTOLOGY

Multiple sections from frontal, parietal, temporal cortex, deep white matter, routine sections from basal ganglia, periventricular grey matter, hypothalamus, mammillary bodies, brain stem and cerebellum have been examined.

Cortex and White Matter The sections show that there is focal meningeal thickening and a cellular reaction in the meninges and perivascular space in the underlying cortex. There is no cortical necrosis but in the deep white matter focal collections of neurones are present arranged in a rather haphazard manner.

Basal Ganglia The sections show no pigmentation or calcification and there is generally good neuronal preservation.

Periventricular Grey Matter, Hypothalamus and Mammillary bodies There are focal collections of neuroblasts in the subependymal zone suggestive of a migration problem. There is generally good neuronal preservation and no vascular proliferation is present in the periventricular grey matter and mammillary bodies. However small foci of necrosis are present in the periventricular grey matter which are probably a consequence of cerebral oedema.

Hippocampi The sections show no displaced neurones or Ammon's horn sclerosis. There is some rarefaction and occasional ischaemic neurones are present in the pyramidal cell layer. No tumour has been identified.

Cerebellum The sections show no significant cell loss in Purkinje cell or granule cell layer. There is no cerebellar cortical dysplasia and the dentate nuclei are preserved.

Brain Stem The sections show focal haemorrhagic necrosis. There is no myelinolysis.

COMMENT:

In summary, the features here are those of cerebral oedema with neuronal migrational defect and a low grade subacute meninoencephalitis. No other discrete lesion has been identified to explain epileptic seizures. The reaction in the meninges and cortex is suggestive of a viral aetiology, though some viral studies were negative during life and on post mortem CSF. With the clinical history of diarrhoea and vomiting, this is a possibility though a metabolic cause cannot be entirely excluded. As this was a brain only autopsy, it is not possible to comment on other systemic pathology in the general organs. No other structural lesion in the brain like corpus callosal or other malformations were identified.

11/2/97

CORONERS ACT (Northern Ireland), 1959

Deposition of Witness taken on the day

of 20 , at inquest touching the death of

, before me

Coroner for the District of

as follows to wit: –

The Deposition of DR BRIAN HERRON

of *(Address)*

who being sworn upon h ~~is~~ oath, saith

I found cerebral oedema which is an end stage of many diseases. I had low (hyponatraemia) levels of the low sodium, — but this may be caused by other diseases — but with ~~hyponatraemia~~. There was mild inflammation of the brain. I did not find any virus to cause this though that does not exclude a virus ~~inflammation~~. A pathologist cannot exclude epilepsy. I was not thinking of fluid management but SIADH. The main pathology finding was cerebral oedema with a little inflammation in the brain. In a typical case of encephalitis the degree of inflammation is more severe.

Mr McCrea: I weighed the brain. It was heavier than normal but there had been abnormal development of the brain. 1300grams would have been expected — Clozier was 1606. That is higher than I would have expected. I cannot recall what medical records I had available at the time. I knew of the ~~drop in~~ low sodium level, the query about SIADH secretion and the respiratory arrest.

Mr. Lavery: In addition there was some brain inflammation — possibly a viral infection.

CR – CORONER