ADAM STRAIN (DECEASED)

MEDICOLEGAL REPORT

REPORT OF PROFESSOR P J BERRY
23.3.96

I am Peter Jeremy Berry of [redacted] My qualifications are BA, MB, BChir, FRCP, FRCPath. I am professor of Paediatric Pathology in the University of Bristol, and have been a Consultant Paediatric Pathologist for more than 12 years.

At the request of HM Coroner for Greater Belfast, Mr J L Lecky, LLM I have examined copies of the case notes of Adam Strain referring to his last admission, the report of Dr M Savage (Consultant Paediatric Nephrologist) reports of Dr R H Taylor (Consultant Paediatric Anaesthetist), and the report on equipment used during Adam Strain's transplant operation. I have also examined 15 stained microscope slides taken at the time of Adam Strain's post-mortem examination.

Background:

Adam Strain was 4 years old at the time of his death. He had a history of chronic renal failure and polyuria with recurrent urinary tract infections in infancy. He had undergone multiple urological operations for vesico-ureteric reflux and a fundal plicaton for hiatus hernia. His renal function had deteriorated to a point where peritoneal dialysis was required in 1994. His nutrition was maintained by night time gastrostomy tube feeding and he was taking multiple medications.

As a result of his treatment, and despite his underlying condition he was well grown and normally nourished.

On the 26th November 1995 he was admitted to the Royal Belfast Hospital for Sick Children to receive a kidney transplant. His pre-operative blood tests including electrolytes, haemoglobin, and coagulation studies were satisfactory.

I will not comment on his pre-operative preparation and intraoperative fluid management which are beyond my expertise. However, no major difficulties were encountered during the operation during which his cardiovascular status and oxygenation remained satisfactory. The surgery was complex, but a satisfactory transplant was carried out with an acceptably matched kidney from a 16 year old donor.

Quite unexpectedly Adam Strain failed to breath spontaneously after his operation and he was found to have dilated pupils and bilateral papilloedoma. There was pulmonary oedema by chest x-ray, and an emergency CAT scan showed cerebral oedema with tonsillar herniation. Tests of brain function were carried out on two occasions and confirmed brain death. Ventilatory support was withdrawn at 11.30 am on 28.11.95 on the second post-operative day.

The reports of Dr R H Taylor, Consultant Paediatric Anaesthetist suggest that the problem was pulmonary and cerebral oedema, although the cause was not apparent.

A report on the equipment in use, while indicating deficiencies showed no cause for equipment failure.

The hand-written report of the surgery in the clinical notes indicates no life threatening operative complication and the kidney perfused reasonably. At 12.05 pm the central venous pressure is recorded as + 30 cm. On examination both pupils were fixed and dilated. Both optic discs were indistinct with retinal haemorrhages. Adam was described as "puffy"

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I have not been shown a copy of the provisional post-mortem findings. However the brain weighed 1320 grams. I understand that the brain and spinal chord are awaiting a neuropathological opinion. The heart was donated for valve transplantation.

Microscope slides

Kidney: Sections show a scarred kidney with numerous cysts, interstitial fibrosis and chronic inflammation, tubular atrophy, glomerulosclerosis, prominence of the juxta glomerular apparatus, hyperplastic tubules with circumferential mesenchyme, a single focus of hyaline cartilage, Tamm-Horsfall protein and thickened arterioles. The number of glomerular generations is reduced. Many of the cysts appear to be medullary.

Spleen: There is intense congestion of the red pulp.

Lungs: There is capillary congestion, occasional clusters of lymphoid cells, and a moderate number of intra-alveolar macrophages. Oedema is not conspicuous, and there is no evidence of embolism. A section of larynx shows superficial ulceration associated with intubation, and mild mucus retention in mucous glands.

Liver: Normal lobular architecture is accentuated by post-mortem change or possible mild extension of fine fibrous trabeculae from portal tracts. There are curious foci of clear cell change in hepatocytes scattered throughout the liver substance. I do not know the significance of these nor can I relate them to any underlying disease process. Portal tracts do not show the changes seen in hereditary renal cystic diseases.

Lymphnode: No significant abnormality.

Transplant Kidney: The kidney shows almost complete infarction.

Comment:

From my examination of the histological sections I can confirm that this child had severe renal disease supporting the clinical decision to undertake renal transplantation. I note the clinical history of reflux and recurrent urinary tract infection. Whilst the histological appearance is entirely consistent with cystic renal displasia, the medullary cysts, intense interstitial fibrosis, and the history of polyuria raise the possibility of medullary cystic disease. (This is not relevant to the child's death, but may be important in counselling and can be resolved from the clinical history).

The transplant kidney was infarcted (dead). The extent of the change suggested that this occurred at or before the time of transplantation. This could be resolved by enquiries about the fate and function of the donor's other kidney after transplantation.

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Continuation Sheet 2

The histological material available to me does not include brain, heart, pituitary, adrenal gland, intestine or skeletal muscle. These tissues might have a bearing on the cause of post-operative death, although are unlikely to do so in the circumstances of this case. Sections of bone and parathyroid glands are part of the post-mortem examination of patients with renal failure.

From the material available to me I have been unable to determine an anatomical cause or underlying disease to account for this child's failure to recover from his transplant operation.

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P J BERRY