IN THE NEWCASTLE UPON TYNE CORONERS COURT

GRO-A DECEASED

INQUEST DATED : GRO-A 1987

NOTES OF EVIDENCE

Verdict : Death by Misadventure

GRO-A

Evidence of identification and background as per statement.

DR. D.J. SCOTT

Gives cause of death as per statement, produces Post Mortem Report.

M.H. SNOW

Produces report referred to also says:-

On balance of probabilities the route of infection is blood transfusion, but that is without testing the then given blood.

CITY OF NEWCASTLE UPON TYNE (TO WIT)

THE EVIDENCE OF WITNESSES severally

taken and acknowledged on behalf of our Sovereign

Lady the Queen, touchi	ng the death of GRO-A		
at the Coroner's Court,	Bolbec Hall, Westgate Road		
in the said City of News	castle upon Tyne, the GRO-A day of	GRO-A 19 ⁸⁷ , be	efore
PATRICK ANTHONY	CYRIL CUFF, one of Her Majesty's	Coroners for the said City of	f Newcastle upon
Tyne sitting at Newcastl	e upon Tyne.		
GRO-A upo	on his oath saith.		
I live at	GRO-A	Newcastle upon Tyne	
The body of the of 21 years, who live	deceased seen after death by red with me at the above addr	me, is that of my wif ess.	e GRO-A , aged

For as long as I have known her, GRO-A was always in good health, had no disabilities, and participated in a lot of sports.

She contracted a throat infection in about June of this year, which resulted in an abcess forming which made eating difficult.

Over the next two months the infection persisted, which resulted in her admission to Newcastle General Hospital at the beginning of September 1986.

GRO-A s condition continued to deteriorate over the last few weeks whilst in hospital, where she remained until her death on Tuesday GRO-A 1986.

David John SCOTT upon his oath saith.

I am a Consultant Pathologist in the Department of Pathology at Newcastle General Hospital.

Acting upon the instructions of H.M. Coroner for the City of Newcastle upon Tyne I performed an autopsy upon the body of **GRO-A** aged 21 years in the post mortem room of the hospital on **GRO-A** 1986.

I had been given a history as shown in my full post mortem report (produced), and was in possession of the clinical notes in respect of the deceased.

In my opinion her death was due to

- 1a. PNEUMOCYSTIS CARINII PNEUMONIA, due to
- b. HUMAN IMMUNODEFICIENCY VIRUS INFECTION.

PM. NO.:	GRO-A
and the second se	

Performed by Dr. D. J. Scient at the request of HIL Coroner, Newcastle upon Tyno.

Notes on the Pos	t Mortem of:-	
GRO-A	<u>AGED</u> : 21 ye	ars
ADDRESS:	GRO-A	, Nevcastle upon Tyne
DATE OF DEATH:	GRO-A 1986	DATE OF PM: GRO-A 1986

HISTORY

Admitted on 3 September 1986 for investigations for a six week history of persistent tonsillar and pharyngeal abscess. Since January of this year she had been treated for recurrent candidiasis. Her first child had been born five years previously and a blood transfustion had been given at the delivery. Her second chil' had been born two years ago following a normal delivery. Investigations showed that she had a low lymphocyte count with high IGA levels. Tests for HTLV-111 antibody were confirmed as being positive on 18 September 1986. On 17 September 1986 she had a biopsy of the tonsillar lesion and this showed no specific features in particular no definite evidence of lymphoma. On 22 September 1986 she showed signs of deteriorating respiratory function and a definative diagnosis of pneumocystis pneumonia was made on 25 September 1986. She was transferred to the ICU for elevitive ventilation. Her condition continued to deteriorate over the next few weeks and she died on **GRO-A** 1986.

EXTERMAL EXAMINATION

There were no obvious abnormal features externally.

INTERNAL EXAMINATION

Central Mervous System

The brain was not examined as there was no indication of CNS involvement.

Cardiovascular System

The pericardial sac was normal. The heart was of normal dimensions externally. The right and left ventricle were of normal thickness and on sectioning the myocardium appeared normal. The tricuspid, pulmonary and aortic values were normal. Pale friable vegetations were present on the atrial surface of the free margin of the mitral value and these measured up to 3 mm in diameter. There was no structural abnormality of the mitral value. The coronary arteries were normal.

Respiaratory System

Larynx and trachea: An area of ulceration was present immediately distal to the vocal cord. The trachea was otherwise normal. The pleural cavities were normal Both lungs were heavy and uniformly consolidated. On section the consolidated lung tissue showed a light grey appearance on the cut surface. The appearances were visible throughout all lobes and were entirely consistent with pneumcystic pneumonia.

Gastrointestinal System

Frances (______) = (______) = (_____) = (_____) = (_____) = (____) = (____) = (____) = (____) = (____) = (____) = (____) = (__) = (_) = (__) = (__) = (__) = (__) = (__) = (__) = (__) = (__)

GRO-A continue.

Peritoneal covity normal. Occophague and large intestines were normal. The liver appeared slightly enlarged but on sectioning appeared entirely normal. Gallbladder and bile ducts were normal. Pencreas normal.

Genito-Unincry System

The 'diveys were of normal size and appeared normal and appeared normal on out section. Unoters and bladder were normal.

Lemnhoid System

The spleen was slightly enlarged and on sectioning there were two recent inferets close to the interior pole, the largest measuring ? on in diameter. Inlarged congested lymph nodes were present in both axillae.

Indocrine System

Thyroid was normal. Adrenals were normal.

Summary of Lesions

- 1. Pneumocystis pneumonia
- 2. Indocarditis probably non bacterial
- 3. Splenic inferct
- 4. Lymphadenopethy



Dr D J Scott

PM HISTOLOGY

Lungs - All sections show pneumocystis pneumonia with large numbers of intra alveolar organisms. There is widespread intra alveolar, interstitial and peribronchiolar fibroblastic proliferation. Pneumocytes are hyperplastic. Scattered organising emboli are present. Heart - The vegetations are composed of fibrin showing early organisation. There is no cellular response. The myocardium shows a mild focal myocarditis. Toxoplasma organisms not seen. Lymph nodes - These show severe cortical depletion. Spleen - An infarct is confirmed. Oesophagus - Normal. Liver - Normal. Cause of Death - 1 a Pneumocystis carinii pneumonia 1 b Human immunodeficiency virus infection

Dr D J Scott GRO-C

GRO-A

P.M. No. GRO-A

COMMENT

Mrs**GRO-A**died frm a type of penumonia which is usually associated with conditions in which the body's immune system is severely impaired. The cause of the immune deficiency in Mrs**GRO-A**was infection with Human Immunodeficiency Virus which is thought to be the causative agent of the acquired immune deficiency syndrome.

GRO-C

Dr. D. Scott.

Snow tant Physician 5 and 6

NEWCASTLE HEALTH AUTHORITY NEWCASTLE GENERAL HOSPITAL

WESTGATE ROAD, NEWCASTLE UPON TYNE, NE4 6BE

TELEPHONE TYNESIDE (091) 273 8811

MHS/SH/X406821

9 March 1987

Mr P W Stobie Police Constable and Coroner's Officer Coroner's Court Bolbec Hall Westgate Road Newcastle Upon Tyne NE1 1SE

Dear Mr Stobie

GRO-A - died Newcastle General Hospital GRO-A 1986

The above named came under my care on 3 September 1986 having been referred from the Department of Haematology, Freeman Hospital to the ENT Department, Newcastle General Hospital who in turn sought my opinion.

The patient's history began approximately 11 months previously with recurrent vaginal infections which included candida, herpes and chlamydia.

Six weeks before admission the patient developed a tonsillar abscess which subsequently discharged, soreness of the mouth and difficulty swallowing. During this period she lost about 2 stones in weight.

On admission under my care the patient was thin and had a temperature. There was severe oral thrush and a large ulcer around the right tonsil. There were enlarged lymph nodes in the neck. Vaginal thrush was present.

Preliminary investigations revealed anaemia and a low white blood cell count. In view of the nature of the presenting infections HIV antibody status was checked and studies of immunological status were carried out.

Treatment was commenced for both the candida infection (with initially Amphoteracin lozenges and Clotrimazole pessaries and subsequently with Ketaconazole) and the infected tonsillar ulcer (with Penicillin and Metronidazole).

After initial improvement the patient was allowed home on 12 September 1986 for a few days while awaiting results but was readmitted on 16 September 1986 with increasing difficulty swallowing.

At this stage results showed immunological changes typical of those seen in AIDS and initial HIV antibody testing was positive. This positive result was confirmed on retesting on subsequent occasions. As the tonsillar abscess was not-healing a biopsy was taken which showed no diagnostic features on histology. Treatment with Acyclovir on the basis that this was likely to be due to herpes simplex and subsequently the ulcer began to heal.

On 17 September 1986 the patient developed a further fever but no new signs were present and chest xray and bacteriological investigations were negative.

9 March 1987

Mr P W Stobie

On 22 September 1986 the development of swinging fever, increased pulse rate and the appearance of chest xray shadowing compatible with an atypical pneumonia a diagnosis of pneumocystis carinii pneumonia (PCP) was made and treatment with high dose intravenous Cotrimoxazole was commenced. The diagnosis was confirmed by fibre-optic bronchoscopy and bronchoalveolar lavage.

At the time of diagnosis of PCP the patient was hypoxic and despite antibiotic and oxygen therapy the patient's lung function deteriorated and chest xray appearances deteriorated to the extent that artificial ventilation with high concentration oxygen became necessary on 25 September 1986 and she was transferred to the Intensive Care Unit. Despite all therapeutic attempts the patient's lung function continued to decline and she died at 0700 hours on **GRO-A** 1986.

With regard to the route of acquisition of the HIV infection responsible enquiries were made of the patient and her husband regarding at risk activities. The patient denied sexual contact with individuals of known high risk groups and the only admitted possible risk factor was of a 2 unit blood transfusion following the birth of her first child.

Her husband denied any history of homosexuality, bisexuality or any sexual activity with any at risk individual. He had not been transfused. He had been tattooed but this was 12 years previously. He denied any history of drug use.

On testing her husband and children for HIV antibodies, her husband and youngest child were found to be positive. The infection of her youngest child indicates that she was positive at the time of his birth.

On testing blood samples which had been stored since the patient's attendance at ward 34 in the autumn of 1985 for investigation of vaginal infections it was shown that the patient was positive at that time. Only a small sample from that time was available from her husband and this was only sufficient for a single test of HIV antibody which appeared to give a negative result.

In view of the history of blood transfusion and the absence of other known risk factors Dr A Collins, Director, Regional Blood Transfusion Service was informed so that enquiries regarding the donors of the blood received by the patient could be made. As far as I am aware one donor has been shown to be HIV negative and the other is untraceable.

In summary the patient had AIDS confirmed by a characteristic opportunistic infection, typical abnormalities of immune status and positive HIV antibodies. There were no admitted risk factors other than the patient's blood transfusion. The infection of her youngest child supports the fact that she was infected before his birth. The testing of stored blood samples point towards her husband's infection being more recent than that.

Yours sincerely

GRO-C

M H Snow Consultant Physician