

Major autopsy findings were of crossed pulmonary arteries macroscopically and pulmonary haemorrhage on histology.

The baby's pre-terminal collapse occurred soon after long line insertion. However, at autopsy there was **no** evidence of air embolism, damage to vessels, bleeding or haemopericardium to suggest that the line insertion had directly led to the deterioration and death. A very small air embolism might not be detectable at autopsy, but then would not be expected to lead to death. Line insertion can cause cardiac arrhythmia, which cannot be assessed at autopsy. Vagal stimulation during line insertion could lead to a bradyarrhythmia, but we would have expected recovery from this to be quite rapid; further clinical opinion on this point may be helpful, as this is not something we can detect anatomically at autopsy. There was no suggestion in the clinical notes of an alternative cardiac arrhythmia.

It is possible that the initial apnoea led rapidly to very extensive pulmonary haemorrhage, which would have made further resuscitation attempts futile.

Crossed pulmonary arteries is a rare anomaly, and in the absence of associated cardiac malformations, has not been reported as a cause of death. The abnormal positioning of the vessels does not cause haemodynamic disturbances. The path of the long line would have not been close to the pulmonary arteries, and they were not obstructed. Therefore, it is not thought that this anomaly is causative of the baby's deterioration or death.

Microbiology and virology tests were negative and acylcarnitine profile and Oil red O stain revealed no evidence of a fatty acid oxidation disorder.

Toxicology revealed caffeine in concentration consistent with therapeutic use.

We are in agreement that the cause of death remains Unascertained.



There were significant abnormalities in different organ systems:

Lungs - chronic lung disease of prematurity.

Brain – white matter hypoxic damage with periventricular leukomalacia.

Heart – fibrotic areas in myocardium indicating previous ischaemia/infarction.

Kidneys - acute tubular necrosis.

Although there was no additional specific acute event identified at autopsy, the physiology in such babies with multi-organ sequelae of prematurity is finely balanced, and the final cause of death is often multi-factorial. Cardiac arrhythmia in a scarred myocardium is always a possibility, but cannot be detected at autopsy and would require clinical monitoring to detect.

We are in agreement that the cause of death is

- 1a. Hypoxic ischaemic damage of brain and chronic lung disease of prematurity due to
- 1b. Extreme prematurity.



No cause was found at autopsy for the initial collapse. The report discusses at some length the concept of "Sudden Unexpected Postnatal Collapse".

There was a ruptured subcapsular haematoma of the liver. The subcapsular haematoma is likely to have occurred as a consequence of hypoxia after the baby's collapse and deterioration. Chest compressions during resuscitation may also have contributed to its formation. Once there has been significant bleeding into the abdomen, this would have significantly contributed to the failure to resuscitate with subsequent death, hence the immediate cause of death was submitted as:

- 1a. Haemorrhage to the peritoneal space due to
- 1b. Rupture of subcapsular haematoma, due to
- 1c. Prematurity.

However, the cause of the initial collapse remains unexplained.

Toxicology was not performed, and therefore medication overdose cannot be excluded.



There were no significant findings at autopsy, and the concept of "Sudden Unexpected Postnatal Collapse" was also discussed in this autopsy report. Prematurity was the only abnormal factor of note in this baby. We discussed that the cause of death (which is given on "the balance of probabilities" to the Coroner) could also have been submitted as unexplained/unascertained, and this would be a subjective decision that would vary between pathologists.

Toxicology was not performed, and therefore medication overdose cannot be excluded.

Referral of this family to Clinical Genetics to discuss potential genetic causes of sudden unexpected postnatal collapse would be reasonable.