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Gaia Young (deceased)
Born 4 March 1996
Died 21 July 2021

Dear Dr Gross

Many thanks for the following:

1. Report dated 30 September 2025
2. Letter dated 14 October 2025

I am grateful to University College London Hospital NHS Foundation Trust (the "Trust") for funding various independent expert reports.

I am the mother of Gaia, my only and much loved previously healthy daughter; she died of an acute and fatal brain illness. My quest has been to investigate her death. I have constructed a website which contains medical and legal documentation concerning her death:

www.truthforgaia.com

I wish to share the information with the world to stimulate curiosity and inquiry into her death.

I appreciate the Trust's and your expressions of sympathy.

Introduction

The central issue is the aetiology of the condition that caused Gaia's death. The investigation of this issue has been difficult. However, it is helpful to understand these difficulties as it may assist the investigation.

The Trust SI report focused on hyponatraemia; hyponatraemia is a well-known non-specific epiphenomenon of any acute brain condition (SIADH) and was not of itself capable of explaining Gaia's death. There was little attempt to investigate any primary encephalopathic process. There was no consideration of ammonia.

Gaia's death was the subject of an inquest in accordance with the Coroners and Justice Act 2009, section 1(2)(a): "cause of death is unknown". Gaia died of an unexplained brain condition.

My consideration of the evidence indicated a fulminant fatal metabolic encephalopathy. I asked also how anyone could "cone" having had a CT head scan two hours previously reported as "No acute intracranial finding". The CT report mandated independent review: please see my submission to the court and the Trust. The Trust and the court disregarded my submission.

The Trust opposed my application for a neurologist to attend and assist the court. The coroner agreed with the Trust. There was no neuroradiological review at the inquest. The medical evidence before the court was largely based on the Trust SI report. The result was an uninformed and uninformative inquest (14 February 2022) – the cause of death was "unclear". The inquest was an opportunity for the Trust to participate in an independent judicial investigation which was concerned specifically with fact and explicitly excluded any consideration of fault.

On 7 March 2022 Dr Ben Killingley consultant physician of the Trust wrote to me after consulting with metabolic diseases specialist colleague and suggested ornithine transcarbamylase ("OTC") deficiency causing hyperammonaemia.

I have encountered senior clinicians who have suggested alternative wide-ranging diagnoses such as water intoxication, serotonin syndrome, and posterior reversible encephalopathy syndrome.

I am concerned that your report has not given sufficient consideration to the aetiology of the condition responsible for Gaia's death.

<u>The autopsy report</u>

You state at paragraph 63:

> "I do not think the autopsy report adds very much to the clinical knowledge. The cause of death was cerebral oedema with herniation of brain structures sufficient to either cut off blood supply or whatever the inflammatory process to cause thrombosis within the main arteries going into the brain."

I respectfully disagree. The reports are immensely helpful and informative; they provide the key to understanding the clinical pathological correlation.

Dr Sheaff's report dated 19 October 2021 included:

> "Ultimately, unfortunately the autopsy cannot establish the underlying cause of the brain oedema and raised intracranial pressure in this case. The brain injury was irreversible and catastrophic but it appears to be a secondary event as no primary pathology was identified within the brain on specialist examination."

Professor Al-Sarraj's report dated 7 October 2021 included:

> "…there is no evidence of any inflammatory cell infiltrate into the brain parenchyma in the meninges. There is no evidence of encephalitis or meningitis.
>
> In summary, the brain shows changes which are secondary to other factors (including hypoxic ischaemic damage or effect of drug use/abuse) but there is no evidence of primary brain pathology which could have caused the respiratory arrest or cerebral oedema."

Whatever caused Gaia's brain swelling killed her. The autopsy reports describe the state of the tissues at death. The striking finding was that there was no primary pathology. The reports do not provide a cause of death but rather the mechanism of death. The autopsy reports do not indicate the *cause* of Gaia's brain swelling but rather describe the *effect* of her brain swelling.

The correlation of clinical and pathological information leads to the following formulation: an encephalopathic process which:

1. originated in the brain.
2. was confined to the brain.
3. caused fulminant lethal brain swelling.
4. did not cause any primary brain pathology.

As far as I am aware, there appears to be only one condition that satisfies these conditions – metabolic encephalopathy. More specifically a urea cycle disorder ("UCD") such as OTC deficiency causing a hyperammonaemia crisis.

My understanding is (forgive me if this known):

1. Ammonia may be regarded as specifically and exclusively toxic to the central nervous system. It may be considered selective endogenous neurotoxin.

2. Ammonia causes intracellular accumulation of glutamine in brain cells – a powerful osmolyte. This leads to cerebral oedema.

3. Ammonia causes hyperventilation and respiratory alkalosis. Gaia's initial blood gas (venous) showed pH 7.514, carbon dioxide partial pressure 3.33kPa.

4. First fatal presentation in adult female of acute hyperammonaemic encephalopathy resulting from OTC deficiency is described in the literature (see Acute Hyperammonemic Encephalopathy Resulting from Late-Onset Ornithine Transcarbamylase Deficiency. M Hershman and others. *Radiology* 2018; 287:353–359).

5. Gaia's clinical history conforms to published descriptions of fatal acute hyperammonaemic crisis in the medical literature.

6. There have also been anecdotal case reports of the outcome of OTC hyperammonaemia crisis. In the press (notably teenage Rohan Godhania deceased, *The Daily Telegraph*, 13 August 2022). Friends provide anecdotes – a boy who presented with floppiness and being unwell. His ammonia was checked and was markedly raised. It was regarded as spurious and was not acted on. He survived and is severely brain damaged.

7. There does not appear to be any viable alternative diagnosis.

8. All the evidence is consistent with OTC mediated hyperammonaemic crisis. None of the evidence refutes OTC mediated hyperammonaemic crisis.

<u>The genetic studies</u>

You state at paragraph 64:

> "A useful thought that this might have represented a genetic metabolic process generating cerebral oedema has been raised, but I could not see for [sic: ?from] the genetic studies that such a diagnosis was likely. For the record, no drugs were detected in the toxicology report or significant amounts of alcohol."

The genetics of OTC deficiency is complex. There are many genes; there are new mutations. A positive genetic match is confirmatory; however, my understanding is that a negative genetic match (absence of evidence) is not exclusionary. It does not represent evidence of absence. My understanding is that a significant percentage of females have *de novo* mutations which can only get detected in specialist research departments.

<u>My comment and conclusion</u>

I have always been open, transparent, and collaborative in my quest for the truth about Gaia's death. My hope is that the Trust reciprocates. The Trust has unrivalled access to medical expertise. Accordingly, I am copying this letter to the Trust. I am also copying it to some clinicians in the Trust: Professor Tom Warner, Dr Robin Lachmann, Dr Ben Killingley, and Dr Elaine Murphy. My hope is that the Trust will circulate my letter in the interests of stimulating clinical curiosity and medical learning.

I consider that the evidence indicates that Gaia died of a hyperammonaemic crisis due to UCD disorder, OTC. This provides a simple, robust, complete, and coherent explanation for Gaia's death:

1. The evidence supports this diagnosis; all the evidence is consistent with it.
2. There is no evidence that refutes this diagnosis.
3. Gaia's clinical history accords with reported proven cases of OTC hyperammonaemia.
4. There is no viable alternative diagnosis.
5. The clinical pathological correlation is not merely a best fit, but a perfect and unique correlation.

Several senior doctors have agreed with me – what else could it be? I appeal to you all to consider OTC hyperammonaemia. If you can find a better fit, then please advise me.

I have previously (through medical contact) approached the editor of a journal of acute medicine to publish Gaia's case study. It was declined: Gaia's case conformed to established medical understanding of OTC – it did not add to the body of medical knowledge.

I thank Dr Ben Killingley again for writing to me on 7 March 2022. I remain baffled as to why the Trust did not put forward the information in his letter before the coroner.

Thank you for your consideration. I look forward to hearing from you and any other interested clinicians.

Yours sincerely

Dorit

Lady Young of Dartington

CC:
Professor David Probert
Professor Tom Warner
Dr Charles House
Ms Cathy Mooney
Dr Robin Lachmann
Dr Elaine Murphy
Dr Ben Killingley