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

Dear Dr Gross (and Baroness Neuberger and Professor Probert)

Thank you for the courtesy and generosity of your letter dated 19 December 2025.

It is a tribute to the independence and integrity of your profession that you disregarded the Trust's instruction to you not to respond to my letter of 9 December 2025. I am concerned and astonished by Trust's wish to stop independent inquiry on clinical issues which may touch upon patient safety. I am copying this letter to clinicians at the Trust so they can consider the conduct of their employer. On 29 June 2022 I received an unsolicited call from a Trust consultant. It was lengthy and the caller was somewhat distraught. The doctor explicitly distanced himself from conduct of the Trust.

You refer to "intense analysis". It is a matter of a proportionate and reasonable inquiry into the aetiology of my beloved daughter's death. There may be implications also for patient safety, Gaia's relatives, and the recipients of Gaia's donated organs. It is a matter of published record that there has been fatality in the recipients of a donated liver where the donor died of encephalopathy due to ornithine transcarbamylase deficiency.

You state:

> "I am not disagreeing with anything you have analysed."

And:

> "If it is accepted by yourself that OTC, hyperammonaemia and encephalopathy is the diagnosis and you have support elsewhere, then it is not for me to doubt that, it is merely indicating that there is a differential diagnosis…"

You have constructed a differential diagnosis as follows:

> "I think we are sitting between your considered diagnosis of OTC, and as yet unknown encephalopathy still to be determined, and an exercise induced encephalopathy."

These three possibilities are considered as follows:

1. "OTC [deficiency]": this is not only the best fit but the perfect fit.

2. "as yet unknown encephalopathy still to be determined"; this is speculative and imagined without any evidential basis.

3. "exercise induced encephalopathy": this is not a unitary pathological entity but a descriptive term that includes multiple aetiologies including stress related OTC. However, I take you to mean hyponatraemic encephalopathy:

   (a) Gaia did not exercise excessively (light cycling with shopping stops) or drink excessively.
   (b) Gaia's sodium fell in response to 3L of Hartmann over 9 hours; this suggested SIADH rather than salt depletion or water intoxication. Gaia's hyponatraemia can be explained completely by SIADH. It was epiphenomenal rather than causative.
   (c) You have previously dismissed hyponatremia: "I personally would not have been concerned about a sodium level of 129." (paragraph 41 of report) and "those figures [123 sodium] were not the cause of her cerebral oedema" (paragraph 54 of report).
   (d) Hyponatraemic encephalopathy is a complex explanation that does not fit the facts.

You state:

> "What we have here is a likely diagnosis sitting within the differential diagnoses, but an absence of total information to say with absolute certainty."

The proposed diagnoses of "unknown encephalopathy" and "exercise induced encephalopathy can be discarded. This leaves OTC deficiency as the only diagnosis.

You state:

> "I could say to you in reverse if this was a scientific discussion, why was this not an exercise induced encephalopathy…"

This has been dealt with at paragraph 3 above.

Neither you nor the Trust have proposed a viable alternative to OTC deficiency.

I accept that the evidence is incomplete. The lack of a blood ammonia level was due to a failure of the Trust to measure ammonia in a case of acute unexplained worsening encephalopathy. The absence of genetic confirmation may be because (I am advised) not all the responsible gene mutations have been identified, or my daughter had a *de novo* variation.

This is not a difficult case. Gaia was healthy; the available clinical and pathological evidence showed the effect of a discrete specific disease process on young adult body where there was no existent pathology. OTC deficiency is a rare condition whose pathophysiology is established. Gaia's case conforms exactly to OTC deficiency – none of the evidence refutes the diagnosis. The available evidence either supports the diagnosis or is consistent with it.

Occam's Razor can be applied to the diagnostic process: *entia non sunt multiplicanda praeter necessitatem*. Why introduce speculation, imagination, and complexity for no good reason?

I ask you and the Trust yet again – if you can find a viable alternative diagnosis to OTC deficiency then please let me know. This is a simple, clear, robust, complete explanation.

It is as if the Trust does not want to find the answer – from firstly objecting to a neurologist at the inquest involving an unexplained brain death – and now wanting to abandon prematurely this independent investigation. If fact cannot be established, then so neither fault. Is this the priority of reputation over safety?

I look forward to receiving any comments you may have - please share with the Trust.

Many thanks for your ongoing assistance.

Yours sincerely

Dorit
Lady Young of Dartington