9 February 2022

Katharine Kandelaki
Claims & Inquests Manager
Quality & Safety Department
250 Euston Road
London
NW1 2PG

Dear Ms Kandelaki

Gaia YOUNG (deceased) DOB 4 March 1996

I am a consultant neurologist, employed by UCLH NHS Foundation Trust ("UCLH") after my appointment in 2006. Although based at the National Hospital for Neurology and Neurosurgery, part of my role as a consultant has been part of the team of neurologists who provide "on-call" neurology advice to the main trust – including the acute medical services. I was involved in the care of Ms Young as the consultant "on-call" at the time she was admitted to UCLH – and I provide further details below.

I have a clear recollection of the case but in my preparation of the report I have also referred to the electronic medical records ("EPIC") and ambulance records.

CHRONOLOGY:

Ms Young presented to UCLH emergency department (ED) at 23:10 hours 17 July 2021 and was transferred to the Acute Medical Unit (AMU) in the early hours of 18 July 2021.

The neurology team was first involved following referral from the medical team on the AMU. I do not know when the referral was made, but it is likely to have been around midday, following the AMU ward round. The neurology registrar (Dr MacDonald) received the referral and as is normal on-call practice, reviewed the patient directly so that she could discuss the findings of with me afterwards. She assessed Ms Young's case first at ~14:30 hours.

As part of her assessment, Dr MacDonald reviewed the medication records, medical and nursing notes, lab work, and also noted the CT head scan that had been undertaken at 13:07 hours. The report produced by a consultant radiologist (Dr Mahfouz) at 13:40 hours show "*no acute intracranial finding*".

In addition to the CT scan, Dr MacDonald was aware that the first of two attempts at lumbar puncture (LP) had taken place at ~14:15 hours (approximately) but had been abandoned as Ms Young had become very agitated during the procedure. It was documented contemporaneously that the intra-thecal space was not accessed/the dural was not punctured.

Dr MacDonald called me to discuss the case at ~15:15 hours. We used the electronic records system ("EPIC") to simultaneously review all the notes and scanning images. We discussed Ms Young's case in detail. We noted the presentation and lab findings, and that she had been commenced on intravenous anti-biotics and antivirals – but that the diagnosis was not clear. I advised that the range of potential diagnoses was broad and included venous thrombosis and encephalitis.

As part of our review, we discussed the utility of lumbar puncture (LP). On the one hand LP would provide CSF, which could be analysed and offered a good prospect of contributing perhaps decisively to diagnosis (for example, identifying infection or similar). On the other, LP could present risk if performed in the context of raised intracranial pressure. Following review of the scan, my view was the CT could be interpreted to show generalised brain oedema and I considered LP may not be safe to perform due to risk of "coning" if the dura was compromised. In parallel with my call with Dr MacDonald, Ms Young was sedated with 2.5 mg of IV morphine and at 15:15 hours (approximately) the LP was attempted for the second time.

During the second LP attempt Ms Young became unresponsive and in respiratory arrest, and the "crash" team were summoned. The LP was not completed – as again, it is described that the intra-thecal space was not accessed. Ms Young did not recover neurologically following this and was transferred to the intensive care unit at UCLH.

Although I had informal discussions with Dr MacDonald that afternoon to discuss Ms Young's progress (and also to discuss other unwell patients we had been referred) notably at 18:54 hours 18 July 2021, I discussed Ms Young's case with the second of the registrars on-call that day (Dr Farag). We further discussed the need for neurosurgical review and possibility of venous thrombosis. At some point on 18 July 2021 (although I do not know the exact time) the neurosurgeons advised that neurosurgical intervention was not indicated.

Following further CT scanning performed at 20:37 hours, we established that it was unlikely that her deterioration was due to clots/thrombosis.

I "handed over" the neurological aspect of her care to a consultant colleague the following morning but note that later in her admission CSF (obtained on 22 July 2021) showed 4 WCC, 9600 RBC (blood stained, and so protein and glucose were not measured) with no organisms were grown after culture, an auto-immune screen (including anti-neuronal antibodies) was negative, and an infection screen [HIV, NEURO-9 screen, Lyme, Borrelia] was negative.

CONCLUSION:

All that I have understood to date suggests that Ms Young was in good health with no history of neurological illness before 17 July 2021.

She presented to UCLH at 23:10 hours on 17 July 2021 with a short (<6 hour) history of headache, nausea/vomiting, and altered behaviour. She was monitored and provided with IV fluids. Some aspects of her condition improved (ability to answer questions, levels of reported pain) and she remained apyrexial. But overall, she remained unwell and continued to behave oddly over ~11 hours of observation and remained hyponatraemic. Her condition was observed to deteriorate at 10:23 hours on 18 July 2021 when she was found to have become pyrexial. Anti-viral and anti-biotic treatment was started at that time. A further, very significant deterioration occurred at ~15:15 hours on 18 July 2021 when she developed respiratory arrest and from this point there was no neurological recovery.

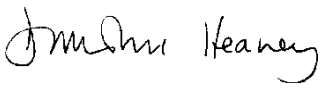
Based on the clinical progression and investigation it seems likely that the patient developed a rapid onset of severe generalised cerebral oedema. This view is supported by the clinical presentation and findings of imaging. The oedema led to coning and brainstem herniation, culminating in the respiratory arrest that occurred at 15:15 hours 18 July 2021. This required a prolonged resuscitation and was complicated by profound hypoxic-ischaemic brain injury.

The cause of cerebral oedema is not apparent. It is extremely unusual for patients to develop malignant cerebral oedema causing such rapid progression to herniation.

STATEMENT OF TRUTH:

I believe the facts stated in this report are true to the best of my knowledge and belief. I understand that proceedings for contempt of court may be brought against anyone who makes, or causes to be made, a false statement in a document verified by a statement of truth without an honest belief in its truth.

SIGNED: **DATE: 9 February 2022**



Dominic Heaney MA FRCP PHD Consultant Neurologist