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IN THE CORONERS COURT FOR NORTHERN IRELAND

CORONER DOUGAN

IN THE MATTER OF AN INQUEST TOUCHING UPON THE DEATH OF ANNA MARGARET MARY McCORMICK

Mr Aidan Sands KC (instructed by Mr Eamon O'Connor, Coroners Service for Northern Ireland) on behalf of the Coroner
Mr David Reid (instructed by Mr Steven Millar, Directorate of Legal Services) on behalf of the South Eastern Health and Social Care Trust (SEHSCT)
Ms Lauren Cheshire (instructed by Mr Jonathan Goode, McCartan Turkington Breen Solicitors) on behalf of the Next of Kin

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Introduction

[1] The inquest proceeded in Laganside Courts on 28, 29, 30 May 2025 and 13 June 2025. During the inquest, I received evidence from a number of witnesses, and I carefully considered further statements, together with voluminous notes, records and reports which were admitted pursuant to Rule 17 of the Coroners (Practice and Procedure) Rules (Northern Ireland) 1963. While it is not feasible to set out all the evidence in these findings, I wish to make clear that I have duly considered all evidence presented before reaching my conclusions.

[2] The deceased, Anna Margaret Mary McCormick, born on 22 May 1946 of 7 Skipperstone Road, Bangor, died on 10 June 2019 in the Ulster Hospital, Dundonald.

Summary of events

[3] The deceased presented to the Accident and Emergency Department of the Ulster Hospital at 22.33 hours on 9 June 2019 with a three-day history of diarrhoea, vomiting, reduced oral intake and increasing confusion. She was first assessed in the early hours of 10 June 2019. Initial investigations identified electrolyte abnormalities, including hyponatraemia and significant hypokalaemia, together with a raised white cell count. A working diagnosis of gastroenteritis with associated dehydration and electrolyte disturbance was made. She was treated with medication and intravenous

fluids. Over the course of the morning and early afternoon, her condition progressively deteriorated.

[4] By late afternoon, the deceased had developed a markedly reduced level of consciousness, hyperpyrexia, cardiovascular instability and abnormal respiratory observations. As her clinical condition worsened, a number of potential diagnoses were considered, including severe infection, neuroleptic malignant syndrome and serotonin syndrome. It was determined that she was not a candidate for escalation to intensive care. She continued to deteriorate despite treatment, including broad-spectrum antibiotics, antiviral therapy and supportive care, and was pronounced dead at 21.05 hours on 10 June 2019, approximately twenty-two and a half hours after her arrival at hospital.

Scope of the inquest

[5] It is well-established that an inquest is a fact-finding exercise. It does not determine civil or criminal liability. The applicable standard of proof is the civil standard, namely the balance of probabilities.

[6] A provisional scope document was presented to the Properly Interested Persons (PIPs) prior to the inquest commencing. None of the PIPs suggested any additions, deletions or other adjustments. The scope document read as follows:

> "The following is a preliminary definition of the scope of the inquest proceedings;
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> 1. This inquest will examine the four basic factual questions as required by Rule 15 of the Coroners (Practice and Procedure) Rules 1963;
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> a. the identity of the deceased,
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> b. the place of death,
>
> c. the time of death, and,
>
> d. how the deceased came by her death.
>
> 2. The identity of the Deceased is not in dispute. The place and time of death can be ascertained on the available evidence.
>
> 3. The inquest will examine all issues concerning the cause and the timing of death.
>
> 4. In relation to how the Deceased came by her death, the Coroner will examine the events of 9 and 10 June 2019, and in particular;
>
> a. The precise causes of the Deceased's death;

b. The diagnosis and medical treatment received by the Deceased while an in-patient in Accident & Emergency and the Medical Assessment Unit Ward in the Ulster Hospital, Dundonald;

c. The consequences of the above matters and whether they caused or contributed to the Deceased's death.

5. At the discretion of the Coroner, subject at all times to the relevant legislation provisions and in accordance with the principles set out in R v Inner West London Coroner ex parte Dallaglio [1994] 4 All ER 139, the inquiry may stretch wider than strictly required and examine other circumstances as seem necessary to the Coroner."

[7] I am satisfied that this inquest has addressed all the relevant issues and that, where possible, I have reached a finding in respect of the matters which come within the scope of this inquest.

Review of the evidence

Joanne McMullan

[8] Mrs Joanne McMullan, daughter of the deceased, gave evidence to the inquest. Mrs McMullan told the inquest that her mother had worked as a seamstress before having to give up in around 1980 as a result of rheumatoid arthritis. She then became a housewife and looked after her and her sister Lynne.

[9] In 2004, the deceased's husband passed away, and Mrs McMullan moved in with her, and she became her mother's carer with assistance from her sister who organised her medications. The deceased's health was generally good apart from arthritis. However, around 2012 she developed bowel cancer and was successfully treated.

[10] Around 2014, she began to suffer the effects of fibromyalgia, but this only had a minor impact on her health, and there were no other health concerns for her until about three years prior to her death, when her mobility began to deteriorate and she began using a rollator at home and a wheelchair when out.

[11] Within the last two years of her life, the deceased was mostly housebound as a result of anxiety and bathroom visits. She attended hospital appointments and managed her own medication at home. Her medication was delivered weekly by the local Clear Pharmacy in a pill box or organiser. Mrs McMullan explained that her mother was "*absolutely meticulous with her medication*". She added that her mother "*did respect her medication and, because of her very strong religious beliefs, prayed over everything that she took*". The deceased's prescription was of eleven types of medications, including tramadol (maxitram), and venlafaxine (vensir XL). Mrs McMullan stated that she may have had occasional medications in a cupboard

such as paracetamol. When asked if the deceased would ever have taken additional medication when her pain levels increased, Mrs McMullan replied that she would only have taken paracetamol in addition to her prescribed medication.

[12] Over the last year, the deceased's appetite became reduced, and her food portions got less and less. Mrs McMullan stated that she lived on crusty bread and lemon curd, which she loved with a cup of tea.

[13] Mrs McMullan explained that on Thursday 6 June 2019, the deceased began belching, almost like a reflux belch, every 5–10 minutes. She could not eat much from this point on.

[14] On Friday morning, she had some loose bowel movements and again on the Saturday morning, 8 June 2019. Mrs McMullan confirmed that the deceased last took her prescribed medication from her pill box on the Saturday.

[15] Mrs McMullan stayed in her mother's room on Saturday night and into Sunday morning. She woke about 07.30 hours, and her mother was restless and asked her to call the Out of Hours Doctor.

]16] Mrs McMullan spoke with the Doctor who prescribed medication that was to be collected that day when the pharmacy opened.

[17] From around midday, Mrs McMullan began to notice the deceased become more agitated and disorientated than normal. She was repetitive and she did not want to be left alone.

[18] Mrs McMullan collected the new medication at 13.30 hours, and the deceased was given it soon after. It took around 30 minutes to take effect, and the belching stopped for a short time, before it returned.

[19] Mrs McMullan gave evidence that, during the course of the day, the deceased stated, "*I'm really scared Joanne, I don't want to die*." Mrs McMullan indicated that this was out of character, as the deceased had consistently expressed to her family, based on her faith, that she was not afraid of death. Mrs McMullan considered this comment to be indicative that the deceased was not behaving as was usual for her.

[20] Mrs McMullan confirmed that neither she nor her husband had been prescribed venlafaxine or tramadol. She stated that, to her knowledge, the deceased took her prescribed medication as directed and did not omit doses in a manner that might have resulted in the accumulation of surplus medication. Mrs McMullan indicated that she was unable to provide an explanation for the elevated levels of tramadol, cyclizine and venlafaxine identified in the deceased's blood at post-mortem. She added that "*I know within my own heart that my mum wouldn't take anything more than she knows that she was allowed*".

[21] Mrs McMullan's husband, a hospice nurse, checked the deceased's temperature later in the day and it was sufficiently high, so they telephoned the Out of Hours Doctor again.

[22] The Doctor sent an ambulance, and the deceased was taken to the Emergency Department in the Ulster Hospital, Dundonald. Mrs McMullan's sister and niece attended and kept Mrs McMullan updated.

[23] At around midday on Monday 10 June, Mrs McMullan was informed by her niece that the deceased was in a bad way, and she was concerned about her and asked her to come up as soon as possible. She arrived at the hospital around 15.30 hours.

[24] The deceased was in a small room in the Emergency Department. The room was incredibly warm, and Mrs McMullan described how the deceased was shaking uncontrollably and breathing erratically. She had dried blood around her mouth and was unaware that Mrs McMullan was present. About 10 minutes, later she was transferred to the Medical Assessment Unit (MAU).

[25] Upon arrival at the MAU, there was immediate concern from the medical staff, and Mrs McMullan recalled someone say, "'*what is this patient doing here? She is far too ill to be moved*'".

[26] From this point, medical staff treated the deceased, and they received good communication from staff. The family raised concerns about her treatment in the Emergency Department. It was only when the family were consulted about resuscitation and religious beliefs, that they began to realise the seriousness of the deceased's condition. At around 20.30 hours, the deceased received the last rights and around 20.45 hours they were advised to make the most of their time with her. She then passed away shortly after.

[27] Mrs McMullan raised concerns regarding the overall care and treatment provided to her mother in the Accident and Emergency (A&E) Department. In particular, she highlighted issues with the maintenance of intravenous access, including periods without IV fluids and electrolyte replacement, and concerns that cardiac monitoring was not consistently maintained. She further questioned the clinical assessment of her mother's condition, noting symptoms such as fever, confusion, agitation, involuntary movements and altered mental state, and expressed concern about delays in treatment and aspects of medication management.

[28] Mrs McMullan also described making repeated requests for assistance which she felt were not acted upon, during which time she observed a deterioration in her mother's condition. She raised concerns regarding her mother's movement within the hospital, including transfer from the resuscitation area and subsequently to a ward, despite her being extremely unwell.

[29] Mrs McMullan also questioned medication management, including the potential for adverse drug interactions, and whether serotonin syndrome ought to have been considered at an earlier stage and whether medication effects contributed to the presentation.

[30] Mrs McMullan expressed the view that earlier recognition of her mother's deterioration, together with greater consideration of the family's concerns, may have altered the outcome.

Lynne Calder

[31] Mrs Lynne Calder, daughter of the deceased, gave evidence to the inquest. She described her mother as being heavily reliant on prescribed medication. In February 2019, the deceased attended an appointment with Dr Niranjan Chogle, Consultant in Anaesthesia and Pain Medicine, at which a plan was made to wean her from gabapentin and introduce an alternative.

[32] Mrs Calder stated that she was responsible for managing the deceased's medications and confirmed that these were supplied in a weekly pill organiser by the pharmacy. In March, the deceased had been unwell and was prescribed cyclizine for nausea; this course was not completed, and several tablets remained in a cupboard where other medications were also stored. Mrs Calder confirmed that the deceased was compliant with her prescribed medication and that she was familiar with all medications taken. She stated that she was unable to explain the elevated levels of certain drugs identified at post-mortem.

[33] Mrs Calder reported that her mother had been unwell for several days prior to hospital attendance, displaying symptoms including vomiting, persistent belching, confusion, was jittery, and had reduced mobility. On the morning of Sunday 9 June, the deceased spoke with an Out of Hours GP and was prescribed cyclizine as she felt she had some ease from them. She stated that her mother did take the leftover cyclizine tablets that were prescribed to her in March. Mrs Calder confirmed that following contact with the Out-of-Hours GP a second time due to worsening confusion and pyrexia, an ambulance was arranged and she was admitted to A&E on 9 June 2019.

[34] Mrs Calder and her daughter Megan McCormick attended hospital with the deceased. On arrival at approximately 22.30 hours, the deceased was placed in a treatment room where she was described as agitated, intermittently confused, making unusual comments which were out of character, and seeking to return home.

[35] Mrs Calder stated that assistance to obtain a wheelchair or trolley was difficult to secure, requiring repeated requests. The deceased required frequent toileting, and her condition deteriorated with increasing confusion and agitation. Blood tests identified significantly low potassium levels, following which she was transferred to the resuscitation area for monitoring and intravenous replacement.

[36] While in resuscitation, the deceased was described as restless with involuntary movements, like tremoring, and in apparent discomfort during observations. Mrs Calder reported that she assisted repeatedly with toileting, requiring staff assistance to disconnect intravenous equipment. A urinary catheter was requested but not inserted. The deceased continued to require frequent toileting and was noted to have a green bowel movement.

[37] Mrs Calder recalled the deceased being assessed by a Consultant, Dr Warnock, on the morning of 10 June, who made a provisional diagnosis of gastroenteritis. Mrs Calder stated that she informed Dr Warnock of the deceased's mild agitation and muscle tremors. She explained that she used words to the effect of "*could you look at my mum's legs and arms, and could you please explain that because my mum looks like a drug addict and she looks like she's coming off something and I'm really concerned that this could be something to do with gabapentin*". Dr Warnock replied to her that he would look into it but that he did not think so and that it could also be signs of low potassium.

[38] Later that morning, following a brief absence by Mrs Calder, the deceased was assisted to the toilet by staff, after which she became exhausted and in pain. During transfer back to bed, the intravenous cannula was dislodged and not immediately replaced despite reported difficulty in obtaining venous access. As a result, there was a period during which no intravenous fluids or potassium replacement were administered.

[39] Mrs Calder described further deterioration in the deceased's condition, including increased agitation, confusion, fluctuating temperature, and abnormal limb movements. She reported ongoing concerns raised with staff regarding the deceased's condition.

[40] The deceased was subsequently moved from the resuscitation area to a small side room, which Mrs Calder considered unsuitable for her needs. She remained without intravenous access for a period until a line was inserted later that afternoon. During this period, Mrs Calder reported difficulty obtaining medical review and treatment, including for agitation, pain, and pyrexia.

[41] Medication including lorazepam and loperamide was administered. By this stage, Mrs Calder described her mother as "*delirious*". She described the deceased at this stage as severely agitated, confused, and unresponsive to family members.

[42] Medical staff later reviewed the deceased, noting a high temperature, abnormal vital signs, and reduced consciousness. Intravenous antibiotics and paracetamol were commenced. At approximately 15.30 hours, the deceased was transferred to the MAU in a significantly deteriorated condition and subsequently became unconscious.

[43] Mrs Calder reported that ward staff expressed concern regarding the timing and condition of the deceased's transfer from A&E to MAU. She considered there to have been deficiencies in care, including delays in treatment, failure to respond to deterioration, and poor communication.

[43] The family raised concerns regarding care prior to and during admission, including an apparent discrepancy in the Out-of-Hours GP prescribing record of 9 June 2019. They stated that the deceased had deteriorated prior to hospital attendance, with pyrexia and confusion, which they felt was not adequately reflected in the clinical records.

[44] The family expressed the view that there were delays in recognising the seriousness of the deceased's condition and commencing appropriate treatment, including for possible infection or a medication-related cause. They additionally raised concerns regarding the combination of prescribed medications and the potential for serotonin syndrome, which they considered was not identified at an early stage, nor was it ever explained to them as a possibility by Dr Chogle. It was also asserted that repeated requests for medical review were made during the admission, and that the deceased's progressive deterioration was not acted upon with sufficient urgency.

[45] Mrs Calder described her mother as a woman of deep and unwavering faith, drawing comfort and strength from her beliefs in every aspect of her life. Even in her final years, when her mobility was limited, she remained the heart and anchor of her family — their "*matriarch*". She carried a quiet trust in those who cared for her, including the medical professionals she believed would always do right by her, just as she reassured her daughters in their own times of need. To her family, she was a source of wisdom, steadiness and reassurance, always seeming to have the right words and the right guidance. Her presence brought comfort and unity, and she remains deeply loved and greatly missed by her family.

Megan McCormick

[46] Ms Megan McCormick, granddaughter of the deceased, gave evidence to the inquest, which was accepted into evidence under Rule 17. Megan McCormick attended A&E with the deceased on the evening of 9 June 2019. She observed that the deceased was agitated and intermittently confused but initially able to engage in conversation. She described difficulty obtaining a wheelchair to assist with toileting despite multiple requests.

[47] Ms McCormick reported that the deceased was later transferred to the resuscitation area due to low potassium levels and a rising temperature. On returning to the hospital the following morning, Ms McCormick observed that the deceased remained agitated and distressed. She was informed that the intravenous cannula had been dislodged and that attempts to re-establish access had been unsuccessful, resulting in the absence of intravenous fluids.

[48] The deceased was subsequently moved to a small side room where Ms McCormick described her condition as deteriorating, with increasing confusion, agitation, and rising temperature. She noted that the room lacked space and ventilation and that the deceased continued to require frequent toileting in unsuitable facilities.

[49] Ms McCormick stated that she and her mother raised repeated concerns with staff regarding the deceased's worsening condition and requested medical review and treatment.

[50] By early afternoon, Ms McCormick described the deceased as severely agitated, largely unresponsive, and exhibiting abnormal movements. She reported that her condition continued to deteriorate despite further requests for assistance. A

doctor later attended, and the deceased was transferred to the MAU at approximately 15.30 hours, at which point she was unconscious. She did not regain consciousness thereafter.

Dr Niranjan Chogle

[51] Dr Niranjan Chogle, Consultant in Pain Medicine and Anaesthesia, gave evidence to the inquest, which was admitted into evidence by way of Rule 17. He saw the deceased on 13 February 2019 following GP referral for optimisation of complex chronic pain.

[52] Dr Chogle recorded a longstanding history of widespread pain consistent with fibromyalgia and seronegative arthritis, together with multiple comorbidities, including previous bowel surgery, hypertension, obesity, depression and reduced mobility. The deceased described significant functional impairment, poor quality of life and increasing reliance on carers.

[53] At the time of review, the deceased was prescribed a high level of analgesic medication, including gabapentin at 2.4g daily for over a decade and tramadol 200mg twice daily, in addition to amitriptyline, anti-inflammatory medication and paracetamol, with limited ongoing benefit.

[54] Dr Chogle expressed concern regarding the extent of polypharmacy and the limited efficacy of the current regimen. He recommended a structured revision of medication, with gradual weaning of gabapentin in the first instance, followed by planned reduction of tramadol, in keeping with standard practice of reducing one medication at a time.

[55] He advised introduction of an SNRI, specifically venlafaxine, as an alternative for management of fibromyalgia-related pain, explaining that such medications may assist both pain and mood symptoms. A cautious approach was recommended, with low initial dosing and slow titration.

[56] Non-pharmacological measures were also discussed, including supportive therapies appropriate to her limited mobility. The agreed treatment plan was communicated to the deceased's GP for implementation, and a follow-up review was planned after six months.

[57] Dr Chogle addressed queries arising from the deceased's family, confirming his involvement was limited to medication management following his consultation with the deceased in February 2019.

[58] Dr Chogle explained that venlafaxine, an SNRI, was introduced as an alternative treatment for pain management. This was commenced cautiously using a low initial dose with gradual titration, including a slower than usual increase schedule, to assess tolerance and minimise adverse effects. He acknowledged that combinations of such medications (venlafaxine and tramadol) carry a recognised risk of serotonin toxicity but described the severe form of serotonin toxicity as rare. He stated that the risk had to be balanced against the greater risks associated with

ongoing high-dose polypharmacy, including respiratory depression and other adverse outcomes. Considering all the factors he proposed a cautious introduction of venlafaxine as the gabapentin was weaned off.

[59] Dr Chogle indicated that it was his standard practice to inform his patients of potential side effects and early warning symptoms of increased serotonin activity, including nausea, diarrhoea and feeling unwell, and advises that such symptoms should be reported to a GP. He confirmed that the agreed medication plan was communicated to the deceased's GP for implementation and ongoing management.

Dr Michelle Godfrey

[60] Dr Michelle Godfrey, then a GP Specialty Trainee working in the out-of-hours service, gave evidence to the inquest. She had a single telephone consultation with the deceased on 9 June 2019 between 08.14 and 08.41 hours.

[61] During the consultation, Dr Godfrey obtained a history from both the deceased and her daughter, noting symptoms of reflux since Wednesday 5 June, recent diarrhoea which had largely resolved, nausea (for which she tried cyclizine), reduced appetite and a recent fall on Thursday 6 June without injury. The deceased was described as alert, orientated, able to speak in full sentences, with no respiratory distress, abdominal pain or confusion reported at that time.

[62] Dr Godfrey was aware that the deceased was weaned off gabapentin and prescribed an anti-depressant. She was able to access the deceased's prescriptions on the Northern Ireland Electronic Care Record (ECR) to view that the deceased was prescribed venlafaxine and tramadol amongst other medications. She agreed that these were commonly prescribed drugs for those with chronic pain. It was put to Dr Godfrey that the 2023-24 Business Services Organisations General Pharmaceutical Services for Northern Ireland Annual Statistics showed that 20.5% of the population were prescribed anti-depressants. Dr Godfrey agreed that it would be common in practice and agreed that in elderly people with other co-morbidities or chronic pain that would be common, as the statistics showed that 30% of those over 65 years of age were prescribed anti-depressants.

[63] Dr Godfrey formed the clinical impression of resolving gastroenteritis with a flare of reflux symptoms, alongside discomfort related to recent reduction in gabapentin.

[64] She advised an increase in lansoprazole dosage and continuation of cyclizine, and additionally prescribed ondansetron. She issued a prescription for these three medications and advised to also continue with Gaviscon. She provided safety-netting advice, including that the deceased should be reviewed if symptoms failed to settle or worsened, and advised follow-up with the Out of Hours GP.

[65] Dr Godfrey did not consider a home visit necessary at that time, as she was reassured by the clinical presentation described. She had no further involvement in the deceased's care.

[66] Dr Godfrey stated that, based on the symptoms reported during her consultation, she did not suspect serotonin syndrome.

Dr Lydia Hanna

[67] Dr Lydia Hanna, a GP in the out-of-hours service, gave evidence to the inquest. She had a single telephone consultation on the evening of 9 June 2019 at approximately 21.10 hours. She spoke with the deceased's daughter, who reported that the deceased had deteriorated since an earlier consultation, with ongoing retching, marked confusion, reduced mobility and a high temperature.

[68] On the basis of this history, Dr Hanna formed the view that there was concern for a serious infection, including possible sepsis, and advised urgent hospital assessment.

[69] As the family indicated that the deceased was not fit for transport by car, Dr Hanna arranged a 999 ambulance for transfer to the Emergency Department in the Ulster Hospital.

Dr Claire Bradley

[70] Dr Claire Bradley, then an ST5 trainee in respiratory and general internal medicine, gave evidence to the inquest. She clerked the deceased at approximately 04.00 hours on 10 June 2019 while working a night shift in the Emergency Department in the Ulster Hospital.

[71] Dr Bradley recorded that the deceased had been unwell since the preceding Thursday with diarrhoea and vomiting, which had largely settled by the time of assessment, although she continued to experience persistent belching and was unable to tolerate solid food, while managing fluids. There were no reported abdominal pain, respiratory symptoms, or confusion at the time of review.

[72] A detailed medical history was documented, including rheumatoid arthritis, fibromyalgia, previous bowel cancer resection, and other comorbidities. A comprehensive medication list was noted, including tramadol, venlafaxine, amitriptyline, anti-inflammatory medication and other regular treatments.

[73] On examination, the deceased was recorded as haemodynamically stable with observations including a temperature of 38°C, pulse 100 bpm, and normal oxygen saturations. Clinical examination identified ongoing belching but no abdominal tenderness, normal bowel sounds, clear chest and no evidence of peripheral oedema. She was documented as not confused.

[74] Investigations demonstrated significant electrolyte disturbance, including hypokalaemia (low potassium at 2.6mmol/L) and hyponatraemia (low sodium at 128), with white cell count mildly elevated and normal CRP. Imaging, including chest and abdominal X-rays, did not demonstrate an acute pathology.

[75] Dr Bradley's clinical impression was of gastroenteritis with associated low sodium, low potassium and a metabolic alkalosis. Her management plan was for rehydration and replacement of potassium with IV fluid and potassium, encouragement of oral intake, and repeat blood tests to monitor electrolytes.

[76] She had no further involvement in the deceased's care after completion of her shift.

[77] When asked about the deceased's medications during their hospital admission, Dr Bradley explained that she used ECR to obtain the deceased's drug history. She was therefore aware of the deceased's regular medications, including tramadol (maxitram) and venlafaxine, and had prescribed these in the medication chart in accordance with the documented history. She outlined that, once prescribed, the administration of medications is carried out by nursing staff, typically during routine drug rounds such as morning and evening rounds, or at other appropriate times as required. The nurses act on the instructions set out in the medication chart when administering medications. She confirmed that this was the process followed in the deceased's case.

[78] According to the deceased's medication chart contained within the hospital records, she was prescribed and administered venlafaxine (recorded as vensir) and also appears to have received maxitram (tramadol) at approximately 10:00 hours on 10 June 2019. In relation to the scheduled doses of maxitram at 22:00 hours and the following morning, an "8" was recorded, indicating a prescribed omission. This notation was subsequently struck through, together with other medications including vensir, indicating that these prescriptions were cancelled in their entirety. Dr Bradley could not assist on when this occurred.

[79] When questioned as to whether it was possible that the deceased may have inadvertently received an increased dose of medication, such as tramadol or venlafaxine, Dr Bradley acknowledged that medication errors can occur in hospital settings, including prescribing errors. However, she stated that, in this instance, she had prescribed the correct dosages on the medication chart. She further noted that both maxitram and venlafaxine are supplied in specific dose strengths, and that it would represent a significant error for multiple tablets at those strengths to be administered unintentionally.

Dr Ashley Warnock

[80] Dr Ashley Warnock, Consultant Acute Physician, gave evidence to the inquest. He had a single involvement with the deceased during a consultant ward round in the Emergency Department on the morning of 10 June 2019.

[81] The deceased was admitted in the Emergency Department on 10 June 2019 under the care of Dr Toner, Consultant Respiratory Physician, the consultant on call. Dr Warnock was assisting her with the morning post-take ward round in the Emergency Department. Dr Warnock examined the deceased in the resus area of the Emergency Department at 08.30 hours. Dr Jill Fulton Core Trainee (CT1) and

Dr Keith McPartland ST4 Acute Medicine Registrar were in attendance and Dr McPartland acted as scribe.

[82] Dr Warnock noted that the deceased had presented the previous evening with symptoms of diarrhoea, vomiting and persistent belching, and had been admitted under the medical team with a working diagnosis of gastroenteritis. Initial assessment identified a temperature of 38°C and significant electrolyte disturbance, including hyponatraemia, hypokalaemia and metabolic alkalosis.

[83] A management plan was in place which included intravenous fluids with electrolyte replacement with intravenous saline and potassium chloride.

[84] During his review, Dr Warnock noted that the deceased's clinical observations were normal and her NEWS score was 1. Her temperature was normal at 37.2°C having been 38°C at the time of admission. Dr Warnock made a provisional diagnosis of electrolyte disturbance and pyrexia due to a likely viral gastroenteritis. His management plan was to continue IV fluids and electrolyte replacement, to reassess electrolyte levels and test stool samples. A bed on the medical wards was awaited.

[85] Dr Warnock explained his plan to the deceased and her daughter. Her daughter, Mrs Calder, mentioned that the deceased had some mild agitation and muscle tremor over the previous days. She queried if this could have been due to withdrawal from gabapentin or the commencement of venlafaxine. Dr Warnock stated that the clinical team did consider whether the deceased's presentation might be attributable either to withdrawal from previously prescribed medication or to an adverse reaction to newly introduced medication. Having reviewed ECR, Dr Warnock was satisfied that gabapentin had been appropriately reduced and withdrawn and that venlafaxine had been commenced and titrated in accordance with standard practice. He noted that the deceased had been taking venlafaxine for several weeks or months, and not at an excessive dose, and therefore considered it unlikely to be the cause of an acute adverse reaction. In his view, the deceased's presentation was more consistent with an electrolyte disturbance.

[86] In terms of clinical features observed at approximately 08.30 hours, Dr Warnock recalled the presence of mild agitation and a muscle tremor. He described this not as pronounced or abnormal movements, but rather as a relatively minor twitching of the muscles. He did not recall observing the more significant or "*writhing*" movements that were described later in the day. At that stage, he did not consider the observed signs to be strongly indicative of a serious neurological syndrome.

[87] Dr Warnock confirmed that the possibility of an accidental overdose of medication was considered. This was explored through discussion with the deceased's daughter. He was informed that there were no empty medication packets in the home and that the deceased's medication was dispensed via a pill organiser, which would have limited her access to excess quantities. On that basis, and in the absence of any other evidence, he stated that the likelihood of overdose was

considered low. He acknowledged that he did not specifically recall asking the deceased herself about the possibility of excess ingestion and believed the discussion was primarily with the daughter.

[88] Dr Warnock accepted that the assessment of overdose risk relied significantly on the account provided by the deceased's daughter, whom he regarded as a reliable historian, and he was content to rely on that information at the time. He acknowledged, however, that such reliance cannot provide absolute certainty, as patients or relatives may not always be aware of, or disclose, the full circumstances.

[89] In response to later queries raised by the deceased's family, Dr Warnock explained that antibiotic treatment is not routinely indicated in suspected viral gastroenteritis and that the initial management was appropriate to the working diagnosis, namely IV fluids and electrolyte replacement.

[90] Dr Warnock told the inquest that serotonin syndrome is a very rare condition, usually occurs within 24 hours of commencing or changing a medication such as venlafaxine, or in cases where an excessive dose or overdose of the medication has been taken. He had only encountered one other case in the past 19 years of practice. He explained that Mrs Calder felt very strongly that the deceased would not have taken any extra tablets and that she was on venlafaxine for longer than 24 hours. Dr Warnock stated that, when he assessed the deceased, her clinical picture was more in keeping with electrolyte disturbance due to GI fluid loss. He did not believe it was a case of an obvious diagnosis being missed. He further stated that, as the clinical picture evolved later in the day, and the deceased's condition worsened, serotonin syndrome treatment was subsequently considered by the treating team. He was not contacted about the deceased's later deterioration in the early afternoon. Dr Warnock explained that the persistent pyrexia at that point would have caused him to suspect serotonin syndrome although he agreed that broad-spectrum antibiotics were also appropriate to cover an infective cause which was being considered.

[91] When reflecting on the clinical features present at the morning ward round, Dr Warnock stated that there were no clear or overt signs strongly suggestive of serotonin syndrome. He explained that he would have expected more pronounced neuromuscular features, such as persistent muscle rigidity rather than a mild tremor. However, with the benefit of hindsight, he accepted that there were subtle features which could be seen as consistent with that diagnosis, including the muscle tremor and an isolated raised temperature. He emphasised, however, that these features were non-specific and equally consistent with what he considered to be the more likely diagnosis of gastroenteritis with associated electrolyte imbalance, including low sodium and potassium levels.

[92] Dr Warnock explained that clinical decision-making on a consultant ward round involves identifying the most likely diagnosis based on the available evidence and proceeding with appropriate treatment for that condition, while continuing to review the patient's progress. In this case, the working diagnosis was gastroenteritis

and electrolyte disturbance, and he considered it appropriate to treat on that basis at the time.

[93] Dr Warnock accepted that serotonin syndrome could have been considered as a differential diagnosis, but maintained that it was not, in his view, a likely diagnosis at that stage. He further stated that the initial management would not have differed significantly, as treatment would primarily have been supportive in nature, including monitoring physiological parameters and correcting abnormalities such as dehydration and electrolyte imbalance. He noted that, at that point, there was no specific intervention required beyond such supportive care.

[94] In relation to the post-mortem findings indicating excess medication in the bloodstream, Dr Warnock stated that, while it is theoretically possible for excess medication to have been administered in hospital, he considered this unlikely. He referred to the safeguards in place, including prescribing records, nursing administration procedures requiring documentation, and, in the case of controlled drugs such as tramadol, the requirement for two staff members to be involved in access and administration. Whilst acknowledging that errors can occur, he considered them unlikely on the balance of probabilities.

[95] Dr Warnock accepted that, if excess medication was present, ingestion prior to hospital admission was a possibility, but he emphasised that he had no objective evidence to confirm or exclude this and did not feel able to reach a definitive conclusion on that issue.

[96] In relation to the measurement of creatine kinase (CK), Dr Warnock stated that this was not a test that would routinely have been performed unless there was a specific suspicion of conditions such as serotonin syndrome. He noted that the CK level later recorded, 3386, was elevated but not markedly so and that CK alone is not diagnostic of serotonin syndrome.

[97] Dr Warnock confirmed that he remained on duty and available later that day but was not asked to review the deceased again. Whilst he indicated that he would have preferred to have been made aware of the deceased's deterioration, he considered that the actions taken by Dr Fulton in escalating to a senior colleague, Dr McPartland, were reasonable in the circumstances.

[98] Dr Warnock stated that he was unable to provide a definitive explanation for how toxic levels of tramadol, cyclizine, together with venlafaxine were reached. He indicated that the matter had been considered within clinical team discussions, including morbidity and mortality meetings, but no clear cause had been identified. He noted that the elevated levels could potentially be due to excess ingestion either prior to or during hospital admission, or less likely an abnormality in metabolism, although there was little clinical evidence to support impaired liver function as a contributing factor. Dr Warnock further observed that the simultaneous elevation of all three drugs was unlikely to result from one medication influencing the levels of the others, suggesting instead that each would have had to be present in excess independently.

Dr Jill Fulton

[99] Dr Jill Fulton, then a Core Trainee (CT1) doctor in General Medicine, gave evidence to the inquest. She reviewed the deceased on 10 June 2019 at approximately 08.30 hours in the Emergency Department as part of the consultant's, Dr Warnock's, ward round.

[100] The deceased was recorded as having presented with vomiting, diarrhoea and some confusion, with a working diagnosis of gastroenteritis and hypokalaemia (low potassium at 2.6mmol/L, with normal range between 3.5mmol/L – 5.3mmol/L). Intravenous fluids with potassium replacement had been commenced, and the plan from the ward round was to continue IV fluid replacement therapy, stool sampling and repeat blood tests to check if potassium levels had returned to normal range.

[101] Dr Fulton advised the inquest that, at the time of this assessment, neither serotonin syndrome nor an adverse drug reaction was being considered. She recalled that the deceased's family raised a query regarding the weaning of gabapentin in the preceding months. ECR was reviewed, and the clinicians were satisfied that the medication had been reduced in a gradual and appropriate manner.

[102] Dr Fulton further stated that serotonin syndrome would typically arise in the acute phase following the introduction of a new medication, usually within days to, at most, a number of weeks, rather than after a period of several months.

[103] At approximately 13.00 hours, Dr Fulton was informed by nursing staff that the deceased had become increasingly agitated, attempting to remove medical equipment and get out of bed. She did not assess the deceased in person. She prescribed oral lorazepam for agitation and loperamide for ongoing diarrhoea. She assumed the morning ward round direction for a stool sample had been actioned. She stated that she did not recall being informed of difficulties with swallow and stated that that information would have been important. When asked why she did not assess the deceased in person, Dr Fulton explained that she had spoken with the staff nurse but did not recall being specifically requested to review the deceased. It was noted that the family had expressed concerns that the deceased appeared confused. She had formed the view that the deceased may have been developing delirium. This was considered in the context of her vulnerability, having regard to her age, past medical history, polypharmacy, the presence of electrolyte abnormalities, and the likelihood of dehydration given her history of vomiting and diarrhoea. On that basis, Dr Fulton proceeded on the assumption that the deceased had become delirious and prescribed medication, accordingly, considering that her presentation was potentially interfering with the delivery of necessary medical care.

[104] At around 14.00 hours, Dr Fulton reviewed the deceased at the request of nursing staff. They stated that the family wished to speak to her. When she assessed the deceased, she observed a significant change in the deceased's presentation. Dr Fulton told the inquest that she would have expected that nursing staff escalate

this to her regardless of the family expressing concerns. Dr Fulton noted reduced engagement, difficulty maintaining eye contact and involuntary writhing movements. The deceased's family expressed concern regarding her deterioration.

[105] Dr Fulton considered a possible diagnosis of delirium and arranged further blood tests to assess if the low potassium level had been corrected or if any biochemical abnormality was contributing to the presentation.

[106] Dr Fulton agreed that there was a steady deterioration in the deceased's presentation, as her NEWS score at 09.30 hours was 1 and at 14.30 hours it was at 4 coupled with her agitation and confusion.

[107] Due to her concern regarding the writing movements and agitation, Dr Fulton escalated the case to the medical registrar, Dr McPartland, who attended and undertook further assessment. She assisted in obtaining further blood samples. She did not recall having a discussion with Dr McPartland about serotonin syndrome, but he did direct specific blood tests such as CK and thyroid function, as he did not have final diagnosis.

[108] Dr Fulton was not present for the entirety of the review but understood that Dr McPartland considered a diagnosis of delirium or central nervous system infection. He commenced treatment including paracetamol, broad-spectrum antibiotics and antiviral therapy.

[109] At approximately 15.30 hours, Dr Fulton was asked by a staff nurse whether the deceased could transfer to the MAU. As she had been reviewed by Dr McPartland and a management plan was in place, and treatment in progress, Dr Fulton deemed that it would be appropriate for the deceased to transfer to the MAU without assessing her in person. She had no further involvement in the deceased's care.

[110] When asked whether it was appropriate that the deceased was transferred to the MAU at that time, when she had a recorded NEWS score of 15 on arrival to the MAU, it was 7 when she left, at 15.30 hours, Dr Fulton explained that the deceased had been waiting for a bed for quite some time which was not ideal and she was aware she was unwell and escalated her concerns to Dr McPartland and there was a management plan in place.

[111] Dr Fulton accepted that, at the time of the deceased's transfer, she did not fully appreciate the clinical implications of a NEWS score of 7. She acknowledged that such a score should have prompted immediate escalation, senior medical review, and consideration of transfer with appropriate critical care support.

[112] She confirmed that she did not request a critical care team to accompany the deceased during her transfer and was unaware of who specifically carried it out. It was put to her, and she accepted in hindsight, that transfer by a porter, and auxiliary nurse was inappropriate given the deceased's condition. She further accepted that she did not check the deceased's NEWS score at the time of transfer, nor did she

consult with Dr McPartland or confirm whether outstanding blood test results were available before agreeing to the move.

[113] Dr Fulton explained that her decision to permit the transfer was based on the understanding that a management plan had been established and initiated, and that a period of time was required to assess the patient's response to treatment. She agreed that reassessment of the patient was necessary but did not specifically verify her clinical status prior to transfer.

[114] In relation to diagnosis, Dr Fulton stated that she had not previously encountered serotonin syndrome at the time. She considered the presenting features to be more consistent with gastroenteritis and delirium, which she regarded as more common in a patient of that age. She noted that serotonin syndrome is rare, often difficult to confirm, and typically suspected in the context of medication changes or overdose, which were not apparent in the deceased's case.

[115] Dr Fulton outlined that treatment for serotonin syndrome is predominantly supportive, including symptom management such as sedation for agitation, intravenous fluids, and respiratory support where required. She indicated that, had she suspected serotonin syndrome at the time, she would have escalated the case to a senior clinician, recognising that it is not a condition she had experience managing independently.

[116] Overall, Dr Fulton accepted that aspects of her management, particularly in relation to the transfer and lack of escalation, would be viewed differently in hindsight. She added, "*on reflection, perhaps it wasn't an ideal time, but I suppose at that point I thought it was an opportunity to transfer this lady because obviously her care couldn't really continue in the emergency department*."

Dr Keith McPartland

[117] Dr Keith McPartland, then a ST3 Medical Registrar, gave evidence to the inquest. He first reviewed the deceased on the post-take ward round at approximately 08.30 hours on 10 June 2019 in the Emergency Department, led by Dr Warnock.

[118] At that time, the deceased was orientated and conversing normally. A working diagnosis of gastroenteritis with associated electrolyte disturbance was made, and she was receiving intravenous fluids with potassium replacement. Dr McPartland stated that he had no concerns that the deceased had taken excess medications or serotonin syndrome at this time.

[119] At approximately 14.27 hours, Dr McPartland was asked to review the deceased by Dr Fulton, following clinical deterioration, increasing agitation and "*writhing movements*". Dr Fulton advised that the deceased had been unwell for approximately one hour and she had prescribed lorazepam.

[120] On assessment, Dr McPartland noted the deceased's temperature was at 40°C, had a GCS of 12/15 and was saying inappropriate words. Due to agitation, she was not fully cooperative with examination, although moving all limbs without focal neurological deficit. He described the limb movements as much more pronounced and slightly different in character to those noted at the morning ward round. He described how all four limbs were moving, with the upper limbs flexed as opposed to a tremor, which he recorded as rigor.

[121] Dr McPartland's working diagnosis was delirium driven by pyrexia, from a possible infective source. Given the acute drop in consciousness level and agitation he was concerned about a possible central nervous system infection.

[122] Dr McPartland initiated a management plan, including intravenous fluids, paracetamol, and broad-spectrum antimicrobial therapy (including antibiotics and antivirals), and arranged blood tests, blood cultures and blood gas analysis. He also requested additional investigations, including a CK level and thyroid function tests, to exclude rarer causes such as serotonin syndrome/neuroleptic malignant syndrome. He told the inquest that this was a possibility in his mind at the time. When asked why he did not record that in his note as a possible diagnosis, he stated that, on the balance of probability, given the other clinical findings, the diagnosis of delirium, rigors and a central nervous system infection were in his opinion more likely. He added that there were no positive results at that time from the CK sample, so it was not something he felt he needed to document at that stage. When it was put that the deceased, was at this time, presenting with classic serotonin syndrome symptoms, he replied that the symptoms were there but that the fever, and rigors could equally be explained by alternative pathologies. Dr McPartland stated that there is no single blood test that will diagnose serotonin syndrome and so there were no other specific blood tests that could be done at that stage.

[123] Dr McPartland stated that, even with the benefit of hindsight, there was no clinical indication to order CK levels at an earlier stage. He explained that CK is a non-specific test, which may be elevated for a variety of reasons, and that the degree of elevation does not reliably correlate with a patient's clinical condition. A patient may have significantly elevated CK levels while remaining relatively stable, or only mildly elevated levels while being clinically unwell.

[124] Dr McPartland agreed that CK can be raised in cases involving muscle breakdown, including rhabdomyolysis, but noted that it may also be elevated following seizures, which themselves can occur in the context of brain infections. Accordingly, he considered that alternative pathologies could account for any elevated CK in the deceased's case.

[125] Dr McPartland concluded that, based on the presenting symptoms and history, there was no clinical justification to request CK testing prior to his involvement at approximately 14.30 hours.

[126] Dr McPartland stated that he could not recall whether any enquiry was made of the next of kin regarding the possibility that the deceased had taken excess

medication prior to admission. He explained that, as part of routine ward practice, prescribed medications are reviewed and administered doses are recorded by nursing staff but acknowledged that records would not capture medication taken by a patient at home.

[127] Dr McPartland accepted that toxicology results contained in the post mortem report subsequently demonstrated elevated levels of cyclizine, tramadol and venlafaxine. In terms of clinical management, Dr McPartland stated that treatment of suspected serotonin syndrome would primarily be supportive and conservative. This would include cessation of any offending medications, control of temperature, and the use of benzodiazepines for agitation. He noted that these measures had been instituted by Dr Fulton and were appropriate.

[128] Dr McPartland later became aware that the deceased had been transferred to the MAU at approximately 15.30 hours without his knowledge. On receiving abnormal results, including elevated CK and abnormal thyroid function tests, he contacted the MAU to communicate his concerns to the team involved in her ongoing care.

[129] When asked what alternative treatment options might have been available had excess medication been known at the time of his review at 14.30 hours, he reiterated that management in such cases is typically limited to withdrawal of the offending agents and supportive care, noting that his prior experience of similar cases involved known or witnessed overdoses.

[130] When asked why he did not discuss his possible diagnoses with consultants Dr Warnock or Dr Toner, Dr McPartland stated that, he had completed his assessment and commenced treatment, and it would be usual practice to observe a patient's response before escalating. He indicated that, had the deceased failed to respond or deteriorated, he would have contacted a consultant at that stage. He further stated that, upon subsequently becoming aware that the deceased had been transferred and that the CK level was elevated, he promptly escalated the matter to a senior member of the team responsible for the deceased's care.

Dr Fiona O'Kane

[131] Dr Fiona O'Kane, Consultant in Acute Medicine, gave evidence to the inquest. She first reviewed the deceased at approximately 16.00 hours on 10 June 2019 shortly after the deceased was transferred from the Emergency Department to the MAU.

[132] On arrival, the deceased had a NEWS score of 15 (from 7) and a significantly reduced level of consciousness (GCS 8/15). Observations included marked hyperpyrexia (41.6°C), tachycardia (137 bpm), respiratory compromise with fluctuating respiratory rates, and reduced oxygen saturation requiring high-flow oxygen.

[133] Dr O'Kane described a significant deterioration in the deceased's condition over a short period, noting an escalation which she characterised as substantial.

Following the transfer, she made enquiries and was informed by Sister Marks that, during the transfer from the Emergency Department to the ward, concerns had arisen among the porter and an auxiliary nurse. At one stage, they considered whether the patient should be returned to the Emergency Department due to a perceived change in her condition but ultimately decided to continue to the ward. Dr O'Kane interpreted this as indicating that the deceased's condition had altered during the course of the transfer.

[134] Upon the deceased's arrival on the ward, Dr O'Kane immediately attended and commenced an assessment using the standard A, B, C (airway, breathing, circulation) approach. She stated that it became apparent very quickly from verbal handover information that there had been a marked and rapid deterioration in the deceased's condition. She was informed that the deceased had previously been able to walk to the toilet with assistance, which contrasted sharply with her presentation on arrival at the ward. From this, Dr O'Kane formed the view that there had been a dramatic change in the deceased's clinical state prior to or during transfer.

[135] Dr O'Kane commenced immediate resuscitative assessment and management, including optimisation of oxygenation, fluid resuscitation for hypotension, and insertion of a urinary catheter. Blood investigations were undertaken, including sepsis bloods, and close monitoring was instituted.

[136] She reviewed prior treatment and noted administration of lorazepam in the Emergency Department. Reversal agents, including flumazenil and naloxone, were administered without clinical effect on her consciousness.

[137] In relation to the deceased's medication chart, Dr O'Kane explained to the inquest that the notation "8" recorded beside the later doses of maxitram (tramadol) referred to the evening dose and the following day and indicated a prescribed omission. This entry had subsequently been struck through, with accompanying notation interpreted as "hold," together with a signature, believed to be that of Dr Devine.

[138] It was understood that this amendment reflected a clinical decision taken at a later stage to withhold the medication when serotonin syndrome was being considered. The medications were thereafter cancelled in their entirety following the deceased's transfer to the MAU.

[139] Notwithstanding the alterations to the chart, it was the understanding of Dr O'Kane that maxitram had been administered earlier that morning, and this informed subsequent clinical management, including the administration of naloxone.

[140] On review of prior medical input, Dr O'Kane noted that the Dr McPartland, had identified significant pyrexia and agitation earlier that afternoon and had initiated antimicrobial therapy for suspected infection, including antibiotics and antivirals. Dr O'Kane continued and expanded this treatment with broad-spectrum antibiotics and steroid cover due to the deceased's background steroid use.

[141] Dr O'Kane confirmed that a CK test had been requested by Dr McPartland, likely in response to the presence of rigors. She was not directly aware of the request at the time it was made but understood that Dr McPartland had telephoned the ward and communicated this to a junior doctor, Dr Beck, who relayed the information to Dr Scott, a specialty doctor, and subsequently to her.

[142] Dr O'Kane was unable to specify precisely when the CK result became known, as she was moving between the deceased and her family during this period. She indicated that the result would have been discussed following the initial phase of resuscitation, at a time when her focus had shifted to ensuring that the deceased had achieved a degree of clinical stability. This included monitoring her NEWS score, maintaining adequate cardiac output, and assessing her respiratory effort.

[143] The blood results demonstrated a markedly elevated CK level of 3386 as recorded by Dr Scott at 16.55 hours. In the context of hyperpyrexia and medication history, differential diagnoses at 16.55 hours were broadened to include severe sepsis, central nervous system infection. The intensive care team became involved in the deceased's care and around 17.30 hours, for the first time, differential diagnosis included medication-related syndromes - neuroleptic malignant syndrome or serotonin syndrome after a discussion about the deceased's prescribed medications. Dr O'Kane stated that serotonin syndrome was rare and can occur at initiation of a particular type of drug including antidepressant medications, at an increase of their dosing or on some occasions when other medications are taken in combination.

[144] A discussion with the intensive care team took place in relation to what else to prescribe in the case of serotonin syndrome. Neuroimaging was arranged, with CT brain scan showing no acute intracranial pathology.

[145] Dr O'Kane discussed the seriousness of the condition with the deceased's family, including her view that she was unsure whether intensive care was the best option for the deceased in the context of the deceased's overall condition and baseline function. The main issues were the very high temperature and her reduced consciousness.

[146] Dr O'Kane later handed over care to the oncoming medical team, with a diagnosis still uncertain but active management ongoing. It was her understanding from the notes and from subsequent discussion with the medical team was that the intensive care team advised the prescription of cyproheptadine, for the treatment of possible serotonin syndrome. However, this was not commenced prior to further deterioration.

[147] Dr O'Kane confirmed that cyproheptadine functions as an antihistamine with serotonin-blocking properties. She emphasised, however, that her personal clinical experience with this medication was extremely limited, stating that the deceased was the only patient in her practice since qualification in 1999 to whom cyproheptadine had been prescribed. She indicated that her understanding, informed by subsequent reading and case review, was that the drug does not have a strong or well-established evidence base.

[148] When asked whether earlier administration of cyproheptadine at around 14.30 hours might have altered the outcome, Dr O'Kane stated that she had considered this question herself but was unable to provide a definitive answer due to her limited experience. She noted that the deceased was critically unwell and that the drug can only be administered orally, requiring absorption and metabolism, which may limit its effectiveness in an acutely deteriorating patient. She further observed that available literature suggested potential benefit may be associated with repeated dosing over a number of days rather than a single early dose.

[149] Dr O'Kane explained that she had discussed the case in a morbidity and mortality meeting and in subsequent departmental discussions, including with consultant colleagues and an intensive care specialist. She reported that no clear consensus was reached and that colleagues had little or no experience of serotonin syndrome. The informal view relayed to her from an intensive care colleague was that a single dose of cyproheptadine would be unlikely to have made a material difference.

[150] Overall, Dr O'Kane expressed the view that cyproheptadine should not be considered a definitive or rapidly effective treatment, and she did not consider it to be a measure that would be expected to produce immediate or dramatic clinical improvement in the circumstances described.

[151] Dr O'Kane was asked whether, with the benefit of hindsight, features of serotonin syndrome could have been identified at an earlier stage. She stated that, in her opinion, the condition did not become apparent until it had "*declared itself*," by which she meant the point at which more definitive clinical features emerged. She identified a markedly elevated temperature, unresponsive to treatment, as a key development prompting consideration of the diagnosis.

[152] Dr O'Kane explained, however, that certain classical clinical signs associated with serotonin syndrome were absent at the relevant time. In particular, clonus and increased muscle tone were not observed, and Dr Devine had recorded the absence of clonus in the clinical notes. Dr O'Kane emphasised that, in the absence of these hallmark features, the clinical approach at the time was to continue evaluating and excluding other potential causes.

[153] Dr O'Kane described clonus as an involuntary rhythmic movement of a limb, typically elicited during examination by dorsiflexing the foot, and indicative of neurological dysfunction. She confirmed that she and Dr Devine actively examined the deceased for this sign but did not find it. She further noted that subsequent discussion of the case in morbidity and mortality meetings, involving multiple consultant colleagues, had not resulted in criticism or suggestions that an earlier diagnosis should have been made.

[154] Dr O'Kane had no further direct involvement prior to the deceased's death later that evening, at 21.05 hours. She subsequently initiated an internal incident review (IR1), noting a potential learning point regarding the transfer of an acutely

deteriorating patient (with a NEWS score of 15) from the Emergency Department to the MAU.

[155] When asked for her view on the appropriateness of the transfer, Dr O'Kane acknowledged that the NEWS score guidance provided clear escalation principles. She expressed the view that the involvement of a senior nurse during the transfer may have been appropriate, particularly given the potential need for resuscitation. However, she also considered that transfer out of a busy Emergency Department to a ward environment may have been beneficial in allowing more focused care.

[156] Dr O'Kane concluded that, while there had been a potential risk of harm associated with the transfer process, the deceased did not suffer harm as a result of the move itself. Any harm that occurred was attributable to her underlying condition rather than the act of transfer.

[157] Dr O'Kane outlined learning arising from the deceased's case in relation to the recognition and management of suspected serotonin syndrome. She stated that, in future, she would be more likely to consider the diagnosis earlier in the assessment and would include measurement of CK at an earlier stage where clinically indicated. She also emphasised the importance of raising awareness among ward staff of the potential for deterioration and of clearly identifying features that should prompt escalation, including early referral to intensive care for advice where necessary.

[158] Dr O'Kane confirmed that, while combinations of medications such as opioids (including tramadol) and antidepressants (including SSRIs and SNRIs) may increase suspicion, such combinations are common in clinical practice and do not routinely lead to serotonin syndrome. Accordingly, she indicated that clinicians do not ordinarily suspect serotonin syndrome in all such cases but rather consider it where the clinical presentation is unusual or evolving in a manner not explained by more common conditions.

[159] Dr O'Kane explained that standard practice in cases of overdose already includes monitoring for serotonin syndrome and checking CK levels in accordance with toxicology guidance. However, she noted that, in her experience, cases of serotonin syndrome are rare despite the frequent use of multiple serotonergic medications.

[160] In relation to the potential role of CK testing, she stated that elevated CK levels are non-specific and may arise in a variety of clinical situations, including prolonged immobility following a fall or seizure activity. She indicated that, in the clinical context of this patient, other diagnoses—such as sepsis or encephalitis—were more consistent with the presenting features, including gastrointestinal symptoms and electrolyte disturbance. She did not consider that the deceased's initial presentation would have prompted suspicion of serotonin syndrome or that earlier CK testing would necessarily have altered the diagnostic approach.

[161] Dr O'Kane concluded that, based on her review of the case, the deceased's presentation on admission did not align with a typical presentation of serotonin syndrome and did not give rise to suspicion of that diagnosis at that stage.

Dr Matthew Devine

[162] Dr Matthew Devine, then an ST6 in Anaesthesia and Intensive Care, gave evidence to the inquest, which was admitted into evidence by way of Rule 17. He became involved in the deceased's care on 10 June 2019 at approximately 17.30 hours when asked by the ICU team to assist with the deceased's care in the MAU and to co-ordinate her transfer to the radiology department for an urgent CT brain scan.

[163] On assessment, Dr Devine found the deceased to be very unwell, with significantly reduced consciousness (GCS 3), marked pyrexia (39.4°C), and elevated heart and respiratory rates. She had already been reviewed by senior medical and ICU staff and was receiving treatment for suspected sepsis and possible neuroleptic malignant syndrome.

[164] Dr Devine accompanied the deceased to radiology for CT imaging and back to the ward; the scan was reported as normal.

[165] Following discussion with the ICU consultant Dr Hendron, ICU Doctor and Dr Devine, it was agreed that admission to the Intensive Care Unit and multi-organ support was unlikely to be beneficial on the basis of the deceased's multiple co-morbidities, long term immunosuppression and poor baseline functional status.

[166] Dr Devine met with the deceased's family, explaining her critical condition, that she was receiving treatment for the possible diagnoses of sepsis and neuroleptic malignant syndrome and that if she did not respond to current treatment, ICU admission and multi organ support was unlikely to be beneficial. It was agreed that escalation to ICU would not be appropriate, and a DNAR order was completed.

Dr Katherine Patterson

[167] Dr Katherine Patterson, then an ST5 Medical Registrar, gave evidence to the inquest. She became involved in the deceased's care during the afternoon handover from the acute medical team on 10 June 2019, when she was informed of the deceased's initial presentation, subsequent deterioration and escalation of treatment.

[168] Later, she was informed by Dr Scott that the deceased had been transferred to the MAU. Upon arrival the deceased was judged to have deteriorated further, and a referral had been made to the intensive care team, and a CT brain scan was to be carried out.

[169] On review, Dr Patterson noted the CT brain scan showed no acute abnormality. She received handover from the ICU team regarding ongoing management, including the need for administration of medication via a nasogastric tube.

[170] Dr Patterson attempted insertion of a nasogastric tube, initially encountering difficulty with unsuccessful placement and transient hypoxia. A subsequent attempt was successful, with gastric aspirate obtained and imaging requested to confirm correct positioning. A nasopharyngeal airway was sited in the right nostril which triggered epistaxis (nose bleed). She then removed it as she was concerned about the deceased aspirating.

[171] Chest imaging demonstrated bilateral lung changes, raising concern for possible pulmonary oedema, infection or developing acute respiratory distress.

[172] On clinical reassessment, Dr Patterson formed the view that the deceased's condition had deteriorated further and that she was likely dying. She communicated this with the deceased's family, who agreed that no further intervention that could cause distress should be undertaken.

[173] Shortly thereafter, the deceased was found to have no signs of life, and Dr Patterson confirmed life extinct at 21.05 hours on 10 June 2019.

[174] Dr Patterson, now a Consultant Geriatrician, provided her opinion as to possible mechanisms by which elevated drug levels and toxicity may have arisen. She noted that tramadol carries particular risk in elderly patients due to variable metabolism and that such medications are often reviewed with a view to discontinuation in this cohort.

[175] She explained that, in clinical practice, there may be a difference between prescribed medication regimens and how patients actually take their medication at home. Patients may vary the timing, omit certain medications, or take them in a manner not strictly in accordance with prescribing instructions.

[176] Dr Patterson further noted that both tramadol and venlafaxine are commonly prescribed in sustained or modified-release formulations, designed to release medication gradually over time. She explained that alteration in the method of ingestion, such as chewing or crushing tablets, can disrupt this mechanism and lead to more rapid absorption, potentially resulting in toxicity. She raised the possibility that, if the deceased had difficulty swallowing and had chewed medication, this could have contributed to elevated drug levels, although she did not express a definitive opinion on this.

[177] Dr Patterson also raised the potential impact of the deceased's high temperature, noting that extreme fever may impair enzymatic function and thereby alter drug metabolism within the body.

[178] In relation to medication use more generally in elderly patients, she described the concept of "*medicines misadventure*," whereby adverse effects may arise from unintentional deviations in medication use, including missed or extra doses, rather than deliberate overdose.

[179] Dr Patterson further outlined that serotonin syndrome may develop along a spectrum from mild to severe, with non-specific early symptoms that may resemble

other conditions, particularly in elderly patients. She noted the possibility that mild symptoms could have been present in the community and progressed over time, although she emphasised that this remained a hypothesis.

[180] Overall, Dr Patterson identified a number of potential contributing mechanisms, including altered ingestion of sustained-release medication, variability in patient compliance, and the effects of severe illness on drug metabolism, but did not advance any single explanation as definitive.

Pathology evidence

Dr Christopher Johnson

[181] Dr Christopher Johnson, former Assistant State Pathologist for Northern Ireland, performed an autopsy on the deceased on 13 June 2019, and thereafter produced a report and gave evidence to the inquest.

[182] Dr Johnson observed that the autopsy revealed only minor bruising to the limbs, which he considered insignificant and not suggestive of serious trauma.

[183] Dr Johnson explained that no source of sepsis was identified at autopsy or on histology. However, he emphasised that in cases where antibiotics have been administered, it is not uncommon for no source to be found post-mortem, stating that this is "*a scenario that's fairly common*" in his experience. Accordingly, he opined that sepsis could not be confirmed on pathological findings alone, despite the clinical suspicion.

[184] Dr Johnson outlined that neuroleptic malignant syndrome is a life-threatening condition typically associated with antipsychotic medication, characterised by fever, cardiovascular instability, and complications such as rhabdomyolysis, hyperkalaemia and renal failure. He noted that it had also been described in association with venlafaxine. However, he emphasised that this is a clinical diagnosis and "*not a diagnosis that can be made with any confidence on autopsy findings alone.*"

[185] Dr Johnson opined that the deceased was in poor general health. He described marked cardiomegaly due to thickening and dilation of the cardiac chambers, most likely attributable to obesity (BMI of 44). He explained that such a condition can precipitate sudden death, either through heart failure or arrhythmia.

[186] He further noted the presence of liver cirrhosis, the cause of which was unclear. Dr Johnson explained that both cardiomegaly and cirrhosis would increase susceptibility to severe infection and the risk of death from sepsis.

[187] Turning to toxicology, Dr Johnson stated that concerns had been raised regarding possible medication issues. He explained that post-mortem blood analysis revealed elevated levels of tramadol, cyclizine, and venlafaxine. He noted that tramadol was present at a level associated in the literature with reported fatalities (3.9 mg/L of tramadol and 1.6 mg/L of desmethyltramadol), and significantly above

expected therapeutic levels. He described this as "*a significant… potentially very significant level*," adding that, in the absence of other history, he "*would readily say that she'd died from tramadol intoxication*."

[188] Dr Johnson further explained that cyclizine (1.4 mg/L) and venlafaxine (3.1 mg/L) were also present at levels exceeding their usual therapeutic ranges. He opined that both drugs could produce central nervous system depression and sedation, and that their combined effects could act "*synergistically*," thereby potentiating the impact of tramadol.

[189] Dr Johnson considered several possible scenarios. He explained that an excess of medication administered in hospital could account for the elevated drug levels and sudden deterioration. However, he noted that the clinical presentation—particularly pyrexia and rhabdomyolysis—was "*not entirely typical*" of drug overdose, as such cases would usually involve sedation and respiratory depression.

[190] He also considered whether pre-existing liver and renal impairment could have led to accumulation of the drugs. While acknowledging that such conditions can increase drug levels, he expressed doubt that they alone would account for the markedly elevated concentrations observed. He later accepted that, following review by Dr Philip Toner, this possibility had effectively been excluded.

[191] Dr Johnson stated that he considered it unlikely that an overdose had occurred prior to hospital admission. He explained that, on a "*common sense*" basis, one would have expected earlier features of tramadol toxicity, including sedation and respiratory depression, if a significant overdose had been taken many hours previously. He added, "*it would seem an unlikely scenario that a large overdose of these three substances, which all can cause respiratory depression, have all been taken 24 hours prior to her death in hospital and she hasn't exhibited low respiratory rate, sedation or anything else like that*."

[192] He noted that other detected substances, including amitriptyline and paracetamol, were present only at therapeutic levels and were not considered contributory to death.

[193] In summary, Dr Johnson explained that while the clinical history was not typical of drug-related death, the toxicology demonstrated significantly elevated levels of multiple medications capable of causing fatal intoxication.

[194] He therefore opined that further expert review by a specialist in toxicology or pharmacology was necessary. He subsequently confirmed that he considered Dr Toner's report to be "*very helpful*" and stated that he would defer to his expertise in relation to serotonin syndrome. He added that this is a clinical diagnosis and it is not a diagnosis as a pathologist that he would be able to make.

[195] Dr Johnson stated that he has never encountered a scenario, suggested by Dr Toner, in which prescribed capsules accumulated in the stomach during a period of gastrointestinal upset (such as ileus) and were then suddenly absorbed en masse

when bowel function resumed, leading to an unintended overdose. While he acknowledged that he cannot entirely exclude the possibility, he emphasised that, based on his experience of many hundreds of drug-related autopsies, this is not a recognised or familiar mechanism. He confirmed that such a scenario would not ordinarily have occurred to him as an explanation and that he has neither seen nor heard of it in practice. Dr Johnson accepted that such a mechanism could theoretically have occurred but emphasised that this falls more appropriately within the experience of a clinician treating living patients, such as a consultant physician. From a pathological perspective, he noted that his practice is confined to examining those who have died, and therefore this is not a phenomenon he would routinely encounter or assess in his work.

[196] Ultimately, Dr Johnson concluded that, although the deceased had significant natural disease capable of causing death, the abnormal toxicological findings were inconsistent with a purely natural cause. He explained that an autopsy represents "*a snapshot … at the time of death*" and, on that basis, determined that the cause of death should be recorded as unascertained at autopsy. Dr Johnson further noted that, having considered Dr Toner's report, he deferred to his expertise and regarded his proposed conclusion—namely that the cause of death was serotonin syndrome arising from drug toxicity—as reasonable.

Expert evidence

Dr Philip Toner

[197] Dr Philip Toner, Consultant in Clinical Therapeutics and Pharmacology & General Internal Medicine, prepared a report on my behalf and gave evidence to the inquest. Dr Toner opined that the most likely diagnosis at the time of admission to the Ulster Hospital on 9 June 2019 was serotonin syndrome as a result of tramadol, and cyclizine toxicity, together with concurrent use of venlafaxine. He told the inquest that serotonin syndrome is a life-threatening, relatively rare adverse drug reaction that can result from therapeutic drug use, toxic administration of drugs, or inadvertent drug-to-drug interactions. Classic symptoms include vomiting, diarrhoea, confusion, agitation, hyperthermia, involuntary movements and neurological disturbance.

[198] Dr Toner stated that even within the subgroup of individuals taking medications capable of precipitating the condition, its occurrence is uncommon, with an estimated incidence of approximately one case per 10,000 patients. This figure relates specifically to that higher-risk population rather than the general population and underscores the overall rarity with which clinicians would expect to encounter the condition in practice.

[199] Dr Toner explained that there is an overlap in symptoms between neuroleptic malignant syndrome and serotonin syndrome. The terms are sometimes used interchangeably and practically speaking, they are very similar. There are differences in presentation and in the drugs that precipitate them, but in terms of assessment and outcomes they are largely the same. He stated that both are ultimately clinical

diagnoses, but that several features in the deceased's case pointed towards serotonin syndrome rather than neuroleptic malignant syndrome. These included the use of venlafaxine, which is strongly associated with serotonin syndrome, particularly in combination with tramadol; the presence of bowel sounds and active gastrointestinal symptoms; normal muscle tone; and the time frame and pattern of deterioration.

[200] Dr Toner did not support the proposition advanced by Dr Patterson that chewing or biting modified-release medication would necessarily result in a sudden increase in drug absorption, noting that such formulations are designed in a way that limits any immediate "*rush*" of medication, even if physically disrupted.

[201] Dr Toner explained that a differential diagnosis included gastroenteritis, which is more common and could account for the pyrexia, gastrointestinal upset, and raised leucocyte count. He noted that the presence of hypokalaemia could have impaired gastrointestinal motility, resulting in delayed gastric emptying. In such circumstances, medication taken at normal intervals could accumulate in the stomach and subsequently be released in a sudden surge (described as a "*drug cake*"), providing a plausible independent mechanism by which serotonin syndrome might arise, separate from the initial illness.

[202] Dr Toner further explained that gastrointestinal upset associated with low potassium (2.6mmol/L on admission) could theoretically slow intestinal transit, leading to delayed absorption and a build-up of medication, with a larger quantity then absorbed at once, thereby mimicking an overdose. He emphasised that this mechanism was theoretical and that there was no evidence to confirm or refute it in this case. He also noted that the post-mortem examination excluded any cause of sepsis.

[203] Dr Toner noted that, although serotonin syndrome is a clinical diagnosis, the treatment approach adopted by the ICU team, including cyproheptadine, was consistent with that working diagnosis.

[204] Dr Toner addressed the potential use of cyproheptadine, explaining that while it has been used in some cases, it is not an established or evidence-based treatment for serotonin syndrome. He noted that there is insufficient clinical evidence to support its effectiveness, that it is not recommended in NICE guidance, and that any potential benefit is theoretical. He further advised that it require multiple doses administered over a number of days and is not comparable to an antidote such as naloxone.

[205] Dr Toner considered that the serotonin syndrome resulted from the combination of significantly raised levels of tramadol and cyclizine, together with the use of venlafaxine, as detected at post-mortem.

[206] Dr Toner opined that, having reviewed all of the available information, he would have made a diagnosis of serotonin syndrome from the outset. He explained that this opinion was based on the drug levels together with the clinical history,

which in his view demonstrated that the classical features of serotonin syndrome were present from the start.

[207] Dr Toner confirmed that, although with hindsight (including knowledge of toxicology results) the most likely diagnosis on admission was serotonin syndrome, he had no criticism of the Emergency Department's initial working diagnosis of gastroenteritis. He explained that, based on consultation with toxicology colleagues across the UK, the majority of patients presenting with serotonin syndrome in the absence of a known overdose are initially diagnosed with conditions such as gastroenteritis, delirium or encephalitis, with the correct diagnosis only becoming apparent following clinical deterioration and repeated assessment.

[208] Dr Toner examined whether pre-existing factors, including renal, hepatic or cardiac impairment, could account for the elevated drug levels identified on toxicology. He concluded that renal function was normal, liver impairment was mild such that the liver would have been capable of metabolising medication, and there was no clear evidence of significant cardiac dysfunction affecting drug distribution. He therefore considered that these factors did not explain the markedly elevated drug concentrations.

[209] In relation to individual drugs, Dr Toner considered that tramadol levels were significantly elevated beyond those expected with normal dosing, and cyclizine levels were also markedly above therapeutic ranges without evidence of hospital prescription. In relation to venlafaxine, Dr Toner told the inquest that recent developments in the interpretation of post-mortem venlafaxine levels mean that previously used reference ranges (which he referred to in his report) are no longer considered reliable or validated. In light of this updated research, he advised that the measured venlafaxine concentration in the deceased's case (3.1 mg/L) falls within the currently accepted (albeit broad) range and cannot be interpreted as indicative of venlafaxine toxicity or overdose.

[210] Dr Toner confirmed that post-mortem levels of tramadol and cyclizine were elevated and that venlafaxine, while used therapeutically, would have contributed to toxicity in combination.

[211] Finally, he considered the impact of medications administered in hospital, concluding that normal prescribed dose of tramadol would not account for the toxic level observed, although its administration, together with venlafaxine, may have compounded an already evolving toxic state.

[212] Dr Toner described this as a complex case. He opined that the cause of death, in his opinion, was the combined effect of excess tramadol and cyclizine, with the concurrent use of venlafaxine, causing serotonin syndrome. He considered that the preceding symptoms of confusion, nausea, gastrointestinal upset, diarrhoea and urinary frequency were consistent with tramadol toxicity. The combined excess use of tramadol and cyclizine, together with venlafaxine, may have precipitated serotonin syndrome, resulting in involuntary movements, pyrexia, confusion, rhabdomyolysis and sudden deterioration.

[213] Dr Toner was asked to consider an explanation for the elevated drug levels, namely excess ingestion of medication or delayed absorption. He stated that one possible mechanism for delayed absorption was gastroparesis secondary to hypokalaemia, leading to reduced gastric motility. He accepted that it was not possible to determine definitively which mechanism applied in the deceased's case. However, on the balance of probabilities, and in light of the available medication supply, he considered it more likely that medication had been taken as prescribed, but that impaired gastric emptying resulted in delayed absorption and a subsequent surge in drug levels, thereby mimicking an overdose. He further accepted that correction of potassium levels (they increased to 3.1mmol/L at 14.52 hours and 3.9mmol/L at 16.55 hours) could restore gastrointestinal motility and result in the release and absorption of accumulated medication. However, he emphasised that this mechanism was theoretical and could not be proven.

Conclusions on the evidence

[214] In reaching my conclusions, I have carefully considered all of the evidence adduced during the course of this inquest. The findings set out below are made on the civil standard of proof, namely the balance of probabilities.

Background and Presentation

[215] I find that the deceased was prescribed daily tramadol and venlafaxine and had taken cyclizine in the days prior to her admission to hospital.

[216] I accept the evidence of her daughters that she managed her medication in a careful and methodical manner, and I find that there is no evidence that she deliberately ingested an excessive quantity of medication.

[217] I find that, in the days preceding admission, she developed a gastrointestinal illness associated with electrolyte disturbance, including significant hypokalaemia.

Assessment and Clinical Management

[218] I accept the evidence of Dr Toner that the deceased's presenting features, including gastrointestinal disturbance, agitation and confusion, were capable of being consistent with both gastroenteritis and early serotonin toxicity.

[219] I find that the initial working diagnosis of gastroenteritis with associated dehydration and electrolyte disturbance was reasonable and supported by the clinical presentation at the time. Whilst some features were, with hindsight, capable of being consistent with evolving serotonin toxicity, those features were non-specific and equally explicable by gastroenteritis, dehydration and electrolyte disturbance. I am satisfied that serotonin syndrome was not an obvious diagnosis on admission.

[220] I find that the deceased was already experiencing a significant clinical deterioration prior to transfer from the Emergency Department to the Medical Assessment Unit. Whilst the transfer should have been subject to greater clinical

escalation and oversight, I find that the transfer occupied only a short period of time, did not interrupt treatment in any significant way and did not cause or make any material contribution to the deceased's death.

[221] I find that the deceased's condition deteriorated rapidly over the course of 10 June 2019, with features subsequently becoming consistent with serotonin syndrome.

[222] I find that features consistent with toxicity were present at the time of admission and that the deceased was already in an evolving toxic state prior to her arrival at hospital.

[223] I find that, once the severity of her condition became apparent, appropriate treatment and escalation were undertaken. Notwithstanding those interventions, her deterioration was rapid and ultimately irreversible.

[224] I accept the medical evidence and find that earlier recognition of serotonin syndrome would not, on the balance of probabilities, have altered the outcome.

[225] I find that the clinicians involved in the deceased's care responded to the clinical information available to them as her presentation evolved. The diagnosis of serotonin syndrome only became apparent as the clinical picture declared itself and alternative diagnoses, including gastroenteritis, sepsis and central nervous system infection, were reasonably considered during the course of her admission.

Toxicology and Mechanism

[226] I find that toxicological analysis demonstrated markedly elevated levels of tramadol and cyclizine, with venlafaxine present and capable of contributing to toxicity in combination.

[227] I find, on the evidence of Dr Johnson and Dr Toner, that the clinical course was not consistent with a conventional acute overdose, in that earlier sedation and respiratory depression would ordinarily have been expected.

[228] I find that the hospital medication records accurately reflect the medications prescribed and administered. I find no evidence that the deceased was inadvertently administered excessive quantities of tramadol or venlafaxine during her hospital admission and, on the balance of probabilities, I reject hospital medication error as the explanation for the toxicological findings.

[229] I further find that there is no direct evidence of excess ingestion prior to admission.

[230] I accept the evidence of Dr Toner and find that the most likely explanation for the elevated drug levels lies in altered pharmacokinetics rather than a straightforward overdose.

[231] In particular, I find that hypokalaemia may have impaired gastrointestinal motility, resulting in delayed gastric emptying and delayed absorption of medication.

[232] I find that this may have resulted in relative accumulation of medication within the gastrointestinal tract and that restoration of gastrointestinal motility following correction of the hypokalaemia may have been followed by increased systemic absorption over a relatively short period, sufficient to produce toxic effects.

[233] I accept that this mechanism, sometimes described in the evidence as the "*cake effect*", remains theoretical. However, I further find that it provides the most coherent explanation when the clinical history, toxicological findings and absence of evidence of excess ingestion are considered together.

[234] I am mindful that the precise mechanism cannot be determined with certainty; however, I am satisfied that this explanation best accords with the totality of the evidence.

[235] I find that such a process would not necessarily give rise to identifiable post-mortem findings.

Causation

[236] I find that the deceased was suffering from serotonin syndrome by the time of her admission to hospital and that the syndrome progressed rapidly during the course of 10 June 2019.

[237] I accept the evidence of Dr Toner and find that the combined toxic effects of tramadol and cyclizine, in the context of concurrent venlafaxine use, gave rise to serotonin syndrome. I prefer Dr Toner's evidence to the other possible explanations considered during the course of the evidence.

[238] I find that the deceased's underlying co-morbidities, including cardiomegaly, liver cirrhosis and obesity, increased her vulnerability to acute illness but do not adequately explain either the toxicological findings or the clinical course which followed.

[239] I further find that neither the timing of diagnosis nor the treatment provided in hospital materially contributed to the deceased's death and that, on the balance of probabilities, the outcome would not have been altered by earlier recognition of serotonin syndrome.

Conclusion

[240] I find that the cause of death, representing the most probable explanation for how the deceased came by her death, is:

1(a) Serotonin syndrome

1(b) Tramadol and cyclizine toxicity in the context of concurrent venlafaxine use.

Postscript

[241] The deceased was a 73-year-old woman of deep faith, quiet strength and dignity, who remained the heart and anchor of her family throughout her life. Despite periods of ill health in later years, she demonstrated resilience and meticulous care in all aspects of her daily life, including the conscientious management of her medication. She was a devoted mother to her two daughters and a cherished grandmother, whose love, wisdom and reassurance brought comfort and unity to those around her. Remembered as the matriarch of her family, her steady presence and unwavering belief sustained those she loved. She is deeply missed by her daughters and grandchildren, who mourn her and will continue to carry her memory and values forward.

[242] This inquest has highlighted the serious and potentially life-threatening condition of serotonin syndrome. The condition may arise from the use or interaction of serotonergic medications, including commonly prescribed antidepressants and certain analgesics, and may initially present with subtle or non-specific symptoms such as agitation, confusion, tremor, sweating and gastrointestinal disturbance before progressing to more severe manifestations, including hyperthermia and autonomic instability.

[243] The evidence in this case demonstrated that serotonin syndrome is an uncommon condition whose presentation may overlap significantly with a number of more common illnesses, particularly during its early stages. As this case illustrates, diagnosis may therefore present considerable clinical challenges. Whilst I have found that serotonin syndrome was not an obvious diagnosis on admission and that earlier recognition would not, on the balance of probabilities, have altered the outcome, this inquest nevertheless underscores the importance of maintaining awareness of medication-related toxicity and potential drug interactions when assessing patients with an evolving and unexplained clinical presentation.