HEALTH & DISABILITY COMMISSIONER
TE TOIHAU HAUORA, HAUĀTANGA

Inadequate monitoring and escalation of care

Executive summary

1. This report concerns the care provided to Mr A by Health New Zealand | Te Whatu Ora – Lakes (Health NZ) at Rotorua Hospital during a six-day period in May 2021. Mr A's daughter, Ms A, complained to the Health and Disability Commissioner (HDC) about the standard of care provided to her father while he was in Rotorua Hospital.

2. HDC's investigation focused on the management of Mr A's fluid status and electrolyte imbalances during his admission, the recognition of his fluctuating levels of confusion, and the escalation of his care in accordance with the Early Warning Score (EWS) system when his condition deteriorated rapidly on Day6 May 2021. Mr A passed away in Rotorua Hospital on Day6 May 2021, and I offer my condolences to his family for their loss.

3. I find Health NZ in breach of Right 4(1) of the Code of Health and Disability Services Consumers' Rights (the Code), which states that every consumer has the right to have services provided with reasonable care and skill, for failing to appropriately monitor and manage Mr A's fluid status and electrolyte imbalances and follow the EWS mandatory escalation pathway on Day6 May 2021. I also make an educational comment on the recognition of Mr A's confusion throughout his admission.

Changes made and recommendations

4. Health NZ has reflected on the care provided to Mr A and noted that his case highlighted areas for improvement regarding documentation, recognising acute deterioration, and escalating care according to the EWS mandatory escalation pathway. Health NZ has

- discussed with the director of nursing and clinical nurse managers the need to ensure that staff are completing fluid balance charts as required; the director of nursing and clinical nurse managers have planned to follow this up with their staff, with the support of Health NZ's professional development unit;
- discussed Mr A's case at a Mortality and Morbidity Committee meeting in September 2021, where a number of areas for improvement were identified; and
- implemented a framework to support the assessment and management of delirium in patients; the framework incorporates the 4AT, a widely used bedside tool to quickly screen patients for delirium and cognitive impairment.

5. I acknowledge and support these initiatives. In addition, I recommend that Health NZ:

    a. provides a written apology to Ms A and Mr A's family for the breaches of the Code identified in this report and that this apology be sent to HDC for forwarding within three weeks of the date of this report;

b. carries out an audit of 15 files of patients whose clinical records indicated they required their fluid status and electrolyte levels to be monitored to confirm that this was undertaken as required;

c. carries out an audit of 15 files of patients who had an EWS or vital sign recorded in the red or blue zones to confirm that the steps in the mandatory escalation pathway were taken;

d. sends a summary of the audit findings for (b) and (c) to HDC within nine months of the date of this report;

e. based on the findings of (b) and (c), provides tailored education to nursing staff and junior medical staff in the Rotorua Medical Unit on monitoring and managing a patient's fluid and electrolyte levels and on the use of the EWS system, including the mandatory escalation pathway and thresholds for escalating care; and

f. provides evidence of the education provided in the form of education materials and the percentage of staff who attended training sessions and updates HDC on the outcome of the training within nine months of the date of this report.

Background

6. Mr A was aged 78 at the time of the events and lived independently in his own home. He had no notable comorbidities, and both his general practitioner (GP) and his daughter noted that he was generally fit and healthy.

7. On 20 April 2021, Mr A visited his regular GP practice complaining of abdominal pain that had started 10 days earlier. When the GP examined Mr A's abdomen, it was tender, and he felt a large abdominal mass. Mr A's GP queried whether he had cancer and scheduled him for an urgent abdominal ultrasound.

8. The abdominal ultrasound performed on 21 April 2021 showed that Mr A had a large mass in his lower abdomen. A CT scan performed on 28 April 2021 confirmed a large retroperitoneal (the space behind the lining of the abdomen) mass involving his mesenteric lymph nodes (lymph nodes in the mesentery tissue that attaches the intestines to the abdominal wall) and encasing the renal and mesenteric vessels. The CT scan also showed evidence of peritoneal metastases.

9. Mr A experienced increasing abdominal distension and pain over this period and visited his GP on 27 and 30 April 2021 for help with pain management and again on 4 May 2021. At his appointment on 30 April 2021, Mr A's GP noted that he had developed oedema (swelling caused by excess fluid in the body's tissues) in his lower legs.

10. On 5 May 2021, Mr A saw a surgeon at Taupō Hospital, who noted the findings of the CT scan and the need for an urgent biopsy, which was scheduled to be done at Rotorua Hospital on Day5 May 2021. This timeframe for biopsy was in line with Ministry of Health guidelines at the time.

Presentation at Taupō Hospital

11. On Day1 May 2021 at 12.20pm, Mr A presented to Taupō Hospital's Emergency Department (ED) with increased abdominal distention, significant swelling in his lower legs that had increased over the preceding week, and nausea. The medical admission document notes that Mr A's pain, previously managed with Panadol and tramadol, had become unmanageable for him.

12. A point-of-care ultrasound in the ED showed that Mr A had mild ascites (excess fluid in the space between the peritoneum and abdominal organs) along with the abdominal mass previously identified.

13. Blood tests also showed that Mr A had low sodium/hyponatraemia (117mmol/L compared with a reference range of 135–146mmol/L) and high potassium/hyperkalaemia (5.9mmol/L compared with a reference range of 3.5–5.2mmol/L).

14. The ED doctor spoke to Mr A, and it was agreed that he would be transferred to Rotorua Hospital, where he would have his scheduled biopsy and the hospital's medical unit could manage his pain, fluid status, and electrolyte imbalances.

15. Mr A's discharge summary from Taupō Hospital notes mild ascites, mild hyperkalaemia likely secondary to decreased renal function, and hyponatraemia likely secondary to hypervolaemia/fluid overload due to ascites and oedema.

Transfer to Rotorua Hospital and subsequent care

16. Mr A was transferred by ambulance to Rotorua Hospital and arrived at 7.15pm on Day1 May 2021.

17. The action plan listed on Mr A's patient transfer form included daily weight monitoring, the completion of fluid balance charts, daily intravenous (IV) furosemide (a diuretic that helps the body remove excess water and sodium), daily blood tests at 8am, a chest X-ray, and regular pain relief.

18. On the morning of Day5 May 2021, Mr A had a biopsy of his right groin lymph node, and an ascitic drain was placed to drain the fluid from his abdomen (paracentesis). Over 2L of fluid was drained during the procedure, and a further 1.5L was drained over the remainder of Day5 May 2021. Following these procedures, Mr A was struggling with ongoing pain and was referred to Rotorua Hospital's Acute Pain Service. He was given a 12.5mcg fentanyl patch, regular paracetamol and tramadol, and an oxycodone elixir if required.

19. Throughout Mr A's admission, he continued to experience nausea and occasional vomiting and was given antiemetics to try to relieve this. He was also given laxatives to help with constipation.

20. Mr A was encouraged to take oral fluids to support fluid and electrolyte balance.

Management of electrolyte imbalances

21. Mr A had very low sodium levels and high potassium levels on arrival at Taupō Hospital, and his electrolyte imbalances required review and management on admission to Rotorua Hospital.

22. As high potassium can cause changes in the heart's electrical activity, an electrocardiogram (ECG) was done on Day2 May 2021, and this showed no signs of change.

23. Mr A was given a trial of spironolactone, a diuretic and hormone-blocking medication that helps the body remove excess water and sodium while conserving potassium. However, this was stopped because of his high potassium levels. Mr A had also been initially prescribed IV furosemide, but this was also stopped, possibly because of his low sodium levels.

24. Urine sodium, osmolality (a measure of the concentration of particles such as sodium and potassium in urine, indicating kidney function and hydration status), and cortisol tests were done on Day4 May 2021. Mr A's urine sodium levels were low, suggesting possible dehydration, and his urine osmolality was within the normal range. Mr A's cortisol levels were taken at 8.05am, according to standard practice. His level was 1,029nmol/L, which is well above the reference range (170–500nmol/L), suggesting that adrenal insufficiency was unlikely. A short synacthen test to assess Mr A's adrenal gland function was planned for Day5 May 2021 but does not appear to have been done.

25. Mr A's sodium and potassium levels remained relatively unchanged throughout his admission up until his death on Day6 May 2021, and regular blood tests were taken on Day3, Day4, and Day5 May 2021 to monitor this.

Fluid management

26. When Mr A was admitted to Rotorua Hospital, it was determined that he required strict fluid balance and monitoring of fluid input and output.

27. Health NZ's fluid balance monitoring guidelines state that staff will monitor patients with actual or potential fluid and electrolyte imbalances so that imbalances can be detected and corrective treatments can be evaluated in a timely manner.

28. The guidelines also state that if a fluid balance chart is required (as was the case for Mr A), the input and output must be documented as accurately as possible. For patients requiring a fluid balance chart for more than 24 hours, a cumulative balance must be calculated and documented at 6am each day, and each shift is responsible for totalling the patient's fluid input and output at 6am, 2pm, and 10pm.

29. A 24-hour fluid recording chart was completed on only Day2 and Day5 May 2021, despite Mr A's patient transfer form from Day2 May 2021 stating that he would have fluid balance charts completed during his admission. Mr A's total fluid input and output were not summed at the bottom of the forms as required, and there are relatively few entries in the forms from Day2 and Day5 May 2021, so it is unclear whether all instances of fluid input and output were recorded on these days.

30. Health NZ acknowledged that fluid balance charts for Mr A were not completed appropriately. It agreed this was significantly below the standard of care it aspires to and noted it was an area for improvement that had been discussed with the director of nursing and clinical nurse managers, who would follow up with their staff and seek support from the hospital's professional development unit.

Recognition and management of confusion

31. Mr A experienced fluctuating levels of confusion throughout his admission to Rotorua Hospital. Nursing notes first mention that Mr A appeared confused and required re-orientation on the evening of Day3 May 2021. On Day4 and Day5 May 2021, his confusion improved slightly, and he was alert and able to converse with staff and his family but did require some re-orientation to time, place, and the people around him. His confusion worsened again on the evening of Day5 May and the early hours of Day6 May 2021, when he pulled out his ascitic drain and was noted to seem delirious.

32. Clinical notes do not record any investigation into the causes of Mr A's confusion, and no assessment tools were used to identify delirium.

33. Health NZ acknowledged that documentation of Mr A's confusion could have been clearer and that staff were aware that he was experiencing confusion, although they may not have fully appreciated the extent of this. It added that the use of assessment tools to identify confusion in situations such as Mr A's is not common practice in hospitals.

Recognition of deterioration and escalation of care using the EWS system

34. Staff used the EWS observation chart to monitor Mr A's vital signs and identify deterioration requiring escalation of care.

35. Throughout Mr A's admission to Rotorua Hospital, his vital signs and EWS were typically recorded every one to four hours, which aligns with the guidance included in Health NZ's Adult EWS Observation Chart and Escalation Pathway policy. However, gaps of 6–11 hours between recordings on Mr A's EWS were sometimes apparent.

36. Mr A's EWS remained between 0 and 2[1] throughout his time in Rotorua Hospital but rose to 3 at 12.45am and 1.15am on Day6 May 2021.

37. Although Mr A's total EWS remained below 5 throughout his admission, his systolic blood pressure fell into the red zone (74/54mmHg) at 12.45am and into the blue zone (54/42mmHg) at 1.35am on Day6 May 2021.

[1] The EWS system ranges from 1 to 10, with higher scores indicating deterioration, critical illness, and a requirement for escalation of care. The EWS form also uses a colour coding system. Yellow corresponds with an EWS of 1–5, orange with an EWS of 6–7, red with an EWS of 8–9, and blue with an EWS of 10 or more. The EWS form uses these colours so staff can easily see when a vital sign falls within the red or blue zones, signalling a likelihood that the patient will deteriorate or is experiencing an immediately life-threatening critical illness. The EWS mandatory escalation pathway sets out the steps staff must follow depending on a patient's EWS.

38. When any vital signs fall into the red zone, a house officer or nurse in charge is required to review the patient within 20 minutes, with regular review every 30 minutes or less, and vital signs must be checked and recorded every 20 minutes.

39. The on-call house officer (OCHO) reviewed Mr A at 1.05am and noted his decreased blood pressure, confusion, and vomiting and that he had pulled out his ascitic drain. The OCHO asked nursing staff to check Mr A's vital signs in 30 minutes or sooner if he deteriorated clinically and to page him with the repeated vital signs at 1.30am as he had to attend to another patient in the interim. The OCHO also advised nursing staff to give Mr A an antiemetic and encourage him to have oral fluids. He ordered C-reactive protein and liver function tests to check for sepsis or infection.

40. After this assessment, the steps in the EWS mandatory escalation pathway and the OCHO's instructions do not appear to have been followed. A retrospective note written by the OCHO states that nursing staff did not page him at 1.30am as requested and that the OCHO instead called the ward for an update on Mr A's condition at 2am, more than 30 minutes after Mr A's last review. The retrospective note says Mr A's latest blood pressure measurement had improved to 93/54mmHg, which is outside the red zone.

41. The EWS mandatory escalation pathway says that a 777 call should be made in the case of acute deterioration. However, although Mr A's systolic blood pressure fell into the blue zone at 1.35am, and a 777 call was not made until 2.35am.

Acute deterioration and cardiac arrest on Day6 May 2021

42. At 2.35am, a retrospective nursing note recorded that Mr A became unresponsive as the nurse was putting a blood pressure cuff on him. The nurse pressed the emergency call button and began cardiopulmonary resuscitation (CPR).

43. The medical registrar attended at 2.40am. Over the course of the next 75 minutes, staff attempted to resuscitate Mr A. This included giving him IV adrenaline and fluids, treating him for hyperkalaemia, and using a defibrillator to shock his heart when appropriate (Mr A did not have a shockable rhythm for most of the 75 minutes).

44. At 4.11am, active treatment was stopped, as directed by the medical consultant, and Mr A was extubated. He was pronounced dead by the OCHO at 4.30am on Day6 May 2021.

45. The medical registrar phoned Mr A's wife and daughter to inform them of Mr A's death. Mr A's death was also referred to the Coroner.

46. No post-death debrief was conducted with the team involved in Mr A's care.

Coronial autopsy results

47. An autopsy was performed on Mr A at 10am the day following Day6 May 2021.

48. The autopsy found that Mr A's primary cause of death was a cardiac arrest (pulseless electrical activity, as per records from Rotorua Hospital) due to advanced abdominal lymphoma (diffuse large B-cell lymphoma).

49. The main findings of the autopsy were that Mr A had advanced malignant diffuse large B-cell lymphoma, coronary atherosclerotic disease (a build-up of plaque in the arteries, which narrows them), severe atherosclerosis of the abdominal aorta, and mild liver steatosis (fatty liver disease).

50. Health NZ addressed the autopsy findings of atherosclerosis in its response to HDC, noting that this finding was concerning for Mr A's family in light of his cardiac arrest. It noted that atherosclerosis is a common condition in older adults and that, given Mr A's condition, treatment of his asymptomatic atherosclerosis was not a clinical priority. It also noted there is no acute treatment for atherosclerosis. Health NZ is of the view that Mr A's atherosclerosis was not a key contributor to his cardiac arrest.

Responses to provisional opinion

51. Ms A was given an opportunity to comment on the 'background' section of the provisional opinion, and her comments have been incorporated into the report where relevant.

52. Ms A expressed disappointment with the way Health NZ addressed many of her concerns throughout the complaint and investigation process. She often felt that her concerns were dismissed or minimised and that Health NZ's responses to her concerns did not reflect the information in Mr A's clinical records.

53. Ms A also noted that Mr A's family were troubled by a lack of open and honest communication from Health NZ staff about Mr A's condition during his admission, which limited their ability to make informed decisions and spend precious time with Mr A before his death. Ms A said family were not informed of Mr A's increasing confusion and deterioration and were reassured by the medical team caring for him that his condition was stable enough for Ms A and Mr A's wife to return home to rest for a couple of days while he remained in hospital. Unfortunately, Mr A died after Ms A and Mr A's wife returned home.

54. Health NZ was also given an opportunity to comment on the provisional opinion, and its comments have been incorporated into the report where relevant.

Analysis

55. As a healthcare provider, Health NZ is responsible for providing services in accordance with the Code. In finding that Health NZ breached the Code, I have relied on advice from Dr Richard Shepherd, consultant general physician (see **Appendix A**).

56. Dr Shepherd acknowledged the complex nature of Mr A's case and noted that he was mindful of the impact of hindsight bias on his advice as he was able to view Mr A's full clinical course, including post mortem results, which was not possible for the team treating Mr A at the time.

57. I also acknowledge the serious and complex nature of Mr A's case. Although this report identifies failings in his care, a change in the management of his condition may or may not have changed the outcome given his aggressive and advanced cancer. Although Dr Shepherd's advice includes comments on contributing factors in Mr A's death, I note that

it is not HDC's role to comment on cause of death but to consider the standard of care provided based on the information available to clinicians at the time of the events.

Management of electrolyte imbalances

58. Dr Shepherd advised that the management of Mr A's hyponatraemia represented a moderate departure from the accepted standard of care, noting that this was a complex aspect of Mr A's care.

59. In his advice, Dr Shepherd referenced the UpToDate[2] clinical treatment guidelines for hyponatraemia, which set out a pathway for determining the cause of low sodium and appropriate treatment options. He noted that there were several potential causes of Mr A's hyponatraemia, some with opposing management strategies, and this necessitated a systematic approach and early investigation to identify the underlying cause and appropriate treatment pathway.

60. Mr A's hyponatraemia was initially assessed as hypervolaemic hyponatraemia due to the excess fluid in his body (abdominal ascites and oedema in both legs). Dr Shepherd noted that this was a reasonable initial diagnostic impression. However, this diagnosis does not appear to have been challenged over the course of Mr A's admission, and this, along with delays in ordering appropriate investigations, contributed to the most likely cause of his hyponatraemia not being identified and the appropriate treatment pathway not being followed.

61. Dr Shepherd was of the view that tests such as for urine osmolality, urine sodium, and cortisol levels should have been done much earlier in Mr A's admission to identify the cause of his hyponatraemia. Although Mr A was admitted to Rotorua Hospital on the evening of Day1 May 2021, these tests were not done until the afternoon of Day4 May 2021.

62. In Dr Shepherd's view, the most likely cause of Mr A's hyponatraemia was hypovolaemic hyponatraemia (caused by fluid loss from the body or fluid moving into body cavities or tissues (third-spacing)). This was supported by the results of Mr A's urine tests done on Day4 May 2021, which showed low urine sodium, as well as the cortisol test result that showed that adrenal insufficiency was very likely not the cause of Mr A's electrolyte imbalances.

63. Dr Shepherd also highlighted other aspects of Mr A's presentation that support a diagnosis of hypovolaemic, rather than hypervolaemic, hyponatraemia. This includes low documented fluid intake (noting that fluid balance charts were not adequately completed during Mr A's admission), recurrent vomiting, constipation, and progressive weight loss (Mr A's weight reduced from 88kg on Day1 May 2021 to 84kg on Day4 May 2021), suggesting dehydration.

64. The team treating Mr A do not appear to have updated their diagnosis or management plan after receiving the results of the urine and cortisol tests. This was evidenced by a short synacthen test being ordered for Day5 May 2021 after it was determined the test was not

[2] Richard H. Sterns. (2025). Overview of the treatment of hyponatremia in adults. *UpToDate*. https://www.uptodate.com/contents/overview-of-the-treatment-of-hyponatremia-in-adults

required. Mr A's condition continued to deteriorate as interventions to address his hypovolaemic hyponatraemia were not put in place.

65. Health NZ noted in its response to HDC that a range of differential diagnoses were considered in relation to Mr A's hyponatraemia and that the causes were likely to be complex and multiple.

66. I have taken note of Dr Shepherd's advice and accept the position he has reached. Although the initial diagnostic impression of Mr A's hyponatraemia was reasonable, failure to follow a systematic approach and order appropriate urine and cortisol tests early in his admission meant that this impression was not challenged. This meant the correct cause of his hyponatraemia was not identified and therefore appropriate treatment was not given. I note that Dr Shepherd recommended the use of a clinical guidance algorithm and a list with differential diagnoses and supporting results to aid clinical decision-making and diagnosis in cases of hyponatraemia. Health NZ has advised that its medical staff regularly use the UpToDate guidelines to manage hyponatraemia; however, it does not appear that the systematic approach set out in the guidelines was followed in Mr A's case. I encourage Health NZ to ensure staff are confident using the guidelines.

Fluid management

67. Dr Shepherd noted that management of Mr A's fluid status was another complex aspect of his care and was tied closely to his hyponatraemia and errors in the diagnosis of this.

68. As outlined above, Mr A required strict fluid balance and monitoring. However, 24-hour fluid recording charts were not consistently completed as required during Mr A's admission, and the steps outlined in the fluid balance monitoring guide were not followed. Overall, very little monitoring and recording of Mr A's fluid input and output appears to have been done. Dr Shepherd advised that this represented a moderate departure from the accepted standard of care.

69. Health NZ agreed that recording and monitoring of Mr A's fluid intake and output were not completed to an acceptable standard.

70. I accept Dr Shepherd's advice. I am critical that nursing staff did not complete fluid balance charts, an important aspect of Mr A's care, throughout his admission. I note that Health NZ has discussed this with its director of nursing and clinical nurse managers and plans to seek support from the hospital's professional development team to support staff training on this issue. I support this initiative.

Assessment and escalation of care when Mr A became hypotensive on Day6 May 2021

71. Dr Shepherd considered this aspect of Mr A's care to have been a moderate departure from the accepted standard.

72. As noted above, staff used the EWS system to monitor Mr A's vital signs and support recognition of deterioration.

73. The mandatory escalation pathway included on the EWS form states that an EWS of 1 to 5 requires staff to manage the patient's pain, fever, and distress; inform the nurse in charge

of the patient's EWS; and repeat vital signs within one hour and then four-hourly if the patient is stable. If indicated, a house officer may review the patient. This pathway appears to have been followed when Mr A's EWS was from 0 to 2 during his admission.

74. However, if any of the patient's vital signs fall within the red or blue zones, even if their overall EWS remains low, this requires escalation. As noted above, Mr A's systolic blood pressure fell into the red zone at 12.45am on Day6 May 2021. The OCHO reviewed Mr A at 1.05am. Dr Shepherd advised that this review and the advice to give Mr A an antiemetic and encourage him to have oral fluids was inadequate given his clinical deterioration, low blood pressure, and confusion after his procedures earlier in the day. Dr Shepherd advised that more senior input could have been sought at this point if the OCHO had recognised the seriousness of Mr A's condition.

75. Staff are required to call 777 in the case of acute deterioration and to review the treatment escalation plan, consider referral to the Intensive Care Unit or palliation as indicated, and seek on-site consultant review of the patient. This was not done when Mr A's systolic blood pressure fell into the blue zone at 1.35am; instead, a 777 call was made only when he lost consciousness and went into cardiac arrest at 2.35am.

76. Dr Shepherd was critical that the steps in the mandatory escalation pathway of the EWS form were not carried out when Mr A's blood pressure fell into the red zone and later the blue zone.

77. I accept Dr Shepherd's advice and am critical that staff failed to appropriately escalate Mr A's care and monitor him in accordance with the EWS system's mandatory escalation pathway, particularly when his blood pressure fell into the blue zone and he required urgent review and escalation of care.

Mitigating factors

78. Dr Shepherd noted in his advice that there appeared to be mitigating factors that influenced the care provided to Mr A. Both the nursing notes and the OCHO's clinical notes mention high workloads impacting on the ability of staff to complete full reviews. Nursing notes highlight negative workload, and the OCHO asked nursing staff to page him with Mr A's vital signs as he had to attend to another patient and was not able to review Mr A in the next half hour as required. Dr Shepherd recommended that Health NZ review resident medical officer workloads, escalation culture, and nursing staff ratios and workloads to determine whether this is an ongoing issue.

<u>Summary: Health NZ – breach</u>

79. I recognise that Mr A presented with an aggressive and advanced form of cancer that made his symptoms both complex and at times challenging for Health NZ to manage. I also accept that a change in Mr A's treatment may or may not have changed the outcome. In this context, this investigation has focused on the standard of care Health NZ provided to Mr A based on the information available to clinicians at the time of the events. In the circumstances, Mr A was entitled to a comfortable, dignified end to his life, and I am concerned that aspects of his care fell short in this regard.

80. For failing to adequately manage Mr A's fluid and electrolyte imbalances and escalate his care in accordance with the EWS system, I find that Health NZ did not provide services to Mr A with reasonable care and skill and breached Right 4(1) of the Code. These were important aspects of Mr A's care that required close attention and appropriate action and, although I note the impact of heavy workloads and the complex nature of Mr A's condition, I remain critical that the accepted standard of care was not met.

Management and investigation of confusion: Health NZ – educational comment

81. Dr Shepherd advised that the management and investigation of the confusion Mr A experienced while on the ward was not ideal and represented a mild departure from the accepted standard of care.

82. Health NZ acknowledged that staff did not fully appreciate the extent of Mr A's confusion but noted that it is not clear whether a change in the management of his confusion would have produced a different clinical outcome.

83. Dr Shepherd noted that, although the nursing staff and the consultant physician recorded Mr A's episodes of confusion during their ward round, a specific cause or evidence of additional investigations is not documented. Dr Shepherd acknowledged that, given Mr A's condition, his confusion was likely to be multifactorial and the team overseeing his care may have reasonably attributed his confusion to his pain, new medications (including opioids and other pain medications), and low sodium levels. However, he stated that staff did not consider other conditions, such as hypovolaemia, which in his view may have been an important contributor to Mr A's confusion.

84. Dr Shepherd also commented on the fact that nursing assessment tools were not used to assess Mr A's confusion, which in his view may have facilitated improved assessment and recognition of confusion as a potential indicator of clinical decline. In its response to HDC, Health NZ noted that it is not usual practice for nurses to use assessment tools to assess confusion. Dr Shepherd also stated in his advice that not using these tools would not in itself be a departure from accepted practice but that use of such a tool might have helped to focus the team caring for Mr A on confusion as an important vital sign that required review. Dr Shepherd suggested that the use of formal confusion assessment tools in routine practice could improve recognition of confusion and its role in indicating clinical deterioration.

85. I accept Dr Shepherd's advice. I note that, since these events, Health NZ has implemented a tool for assessing delirium and cognitive impairment, and I support this initiative.

Distribution and follow-up actions

86. A copy of this report with details identifying the parties removed, except Health New Zealand | Te Whatu Ora – Lakes and my expert advisor, will be placed on the HDC website, www.hdc.org.nz, for educational purposes.

Rose Wall
Deputy Health and Disability Commissioner

Appendix A: Independent clinical advice to the Commissioner

The following independent advice was obtained from Dr Richard Shepherd, consultant general physician.

Qualifications, training and experience relevant to the area of expertise involved:	My name is Dr Richard Shepherd. I am a Consultant General Physician employed by Te Whatu Ora Waikato. I graduated from Otago Medical School in 1997 with a Bachelor of Medicine and Surgery (MBChB). I have attained fellowships with the Royal New Zealand College of Urgent Care, The Division of Rural Hospital Medicine and the Australasian College of Physicians. I have subspecialty interests in acute care medicine, nephrology, emergency medicine and palliative care. I have completed the Auckland University Postgraduate Diploma of Community Emergency Medicine, the RACP Clinical Diploma in Palliative Medicine, the Otago University Certificate in Physician Performed Ultrasound and the Auckland University Postgraduate Certificate in Health Leadership, including quality and safety. I am also employed part time as an ACC medical advisor within the treatment injury portfolio. I have no conflicts of interest to declare in this case.
Documents provided by HDC:	1. Letter of complaint dated 8 August 2022 2. Health New Zealand | Te Whatu Ora – Lakes (Health NZ) response dated 24 January 2023 3. Clinical records from Health NZ covering the period 20 April 2021 to [Day6] May 2021.
Referral instructions from HDC	For Health NZ – Lakes, comment on: 1. The investigation and management of Mr [A]'s intermittent confusion/delirium. 2. The adequacy of monitoring and management of Mr [A]'s electrolyte abnormalities. 3. The timeliness of scheduling of Mr [A]'s abdominal mass biopsy and paracentesis following his admission to Rotorua Hospital on [Day1] May 2021. 4. The adequacy of assessment and escalation of care when Mr [A] was first noted to be hypotensive in the early hours of [Day6] May 2021.

	5. The adequacy of clinical documentation, noting the retrospective note completed by the OCHO following Mr [A]'s death. 6. Any other comments on Mr [A]'s care. In relation to each of the questions above, please advise: 1. What is the standard of care/accepted practice? 2. Has there been a departure from the standard of care or accepted practice? If so, please identify how the care departs from standards or accepted practice, and the extent of that departure (mild departure, moderate departure or severe departure) having regard to the scale below: • No departure – the care provided was consistent with standards and accepted practice in all respects. • Mild departure – while generally acceptable, the care provided did not meet some standards or accepted practice to a minor extent. • Moderate departure – the care provided did not meet a particular standard or accepted practice but there were relevant mitigating factors present and considered. • Severe departure – the care provided fell well below an acceptable standard in respect of an essential aspect of the consumer's care, and there was an absence of relevant mitigating factors. 3. How would the care be viewed by your peers?

Brief summary of clinical events:

The complainant has raised concerns about the consumer's rapid deterioration and unexpected death whilst under the care of Rotorua Hospital.

Provider Response:

Mr [A]'s case has highlighted areas of documentation and attention towards recognising acute deterioration and to escalating the care when signalled by the EWS system. We are

also working more proactively on ensuring family are both consulted and advised regarding care and treatment pathways.

Clinical Notes:

History:

- Mr [A] was under investigation for ongoing abdominal and back pain;
- He was seen by GP on 20th April 2021;
- On examination a palpable abdominal mass was found;
- Abdominal US 21/4 revealed retroperitoneal mass;
- Discussed with surgical registrar, and a surgical urgent referral was done and an Outpatient CT scan was booked for 28/4/21;
- CT scan 28/4/21 revealed: retroperitoneal/intraperitoneal mass and lymphadenopathy above and below the diaphragm. Findings are favoured to represent lymphoma;
- Mr [A] was seen by the Surgical Team in Outpatients on 05/5/21;
- A biopsy was planned for [Day5]/5/21.

Hospital presentation:

- Mr [A] presented to Taupo Hospital [Day1]/5/21 with increased abdominal distention/discomfort, lower extremity swelling and ongoing nausea;
- Obs. in Taupo: T 36.3, P 91 BP 166/85 R 26 O2 96% on RA;
- Bloods: hyponatraemia, acute kidney injury;
- Discussed with Surgical Team on call;
- Transferred to RPH [Day2]/5/21;
- On presenting to Rotorua Hospital, Mr [A]'s overall examination was normal except for a distended and generally tender abdomen, and swelling of both legs to the knees;
- Admitted under Medicine Services; Impression IVC obstruction secondary to retroperitoneal mass;
- Discussion regarding expediting biopsy +/- Ascitic drainage;
- Trial spironolactone;

- Hyponatraemia could be related to Addisons from infiltration of adrenals by tumour.

Issues while on Medical Unit:

- Mr [A] experienced new fluctuating confusion multifactorial: hyponatraemia/ analgesia/ lack of sleep/ zopiclone;
- Continued hyponatraemia/ hyperkalaemia;
- Ongoing pain.

[Day5]/5/21

- Mr [A] underwent an ultrasound-guided biopsy and ascetic drainage which resulted in >2L of fluid being drained;
- Acute pain referral and a pain relief fentanyl patch administered;
- Mr [A] then experienced drowsiness, confusion and unsteadiness all of which were noted in the PM shift clinical record;
- Mr [A] then suffered worsening confusion in early hours of [Day6]/5/21 with haemodynamic instability followed by cardiac arrest;
- Prolonged CPR;
- Mr [A] declared deceased;
- No debrief of teams involved was carried out.

Prior to responding to the specific questions posed by the HDC I would make the following comments:

It is acknowledged that Mr [A]'s case was a complex multifaceted presentation with a rapidly changing clinical course over a short timeframe on the background of a severe underlying pathology. In my view there were a number of failings over the 5 days of his care in Rotorua Hospital that ultimately combined and summed together leading to his cardiac arrest.

With the benefit of a post-mortem examination, no time constraints or competing patient priorities for review and the entirety of his clinical course laid out before me it is a considerably more simple matter to identify the underlying diagnoses and the points where alternative decisions in my view would likely have led to a different outcome and prevention of his arrest.

Whist his arrest was a sudden clinical event, the seeds had been sown much earlier in his care. I am however very mindful of the inherent potential retrospective case review bias in my below responses to the questions posed appreciating this is not how clinical bedside medicine occurs. At the bedside, pieces of the puzzle evolve over time, often in less than ideal timings and in a setting where the most experienced and senior clinician may not even be involved.

With the benefit of hindsight then in my view Mr [A]'s death was due to a PEA (Pulseless Electrical Activity) cardiac arrest. On balance I would attribute that arrest predominantly to his progressive hypovolaemia perhaps complicated by a degree of mild hyperkalaemia and inferior vena caval compression. Underlying that hypovolaemia was a complex mix of third spacing, **hypo**volaemic hyponatraemia with an acute kidney injury - and a precipitating procedure of ascites paracentesis (removal of free fluid from the abdomen) likely contributing to post-paracentesis circulatory dysfunction syndrome tipping his already compromised but compensating physiology one step too far - resulting in his PEA arrest. Underlying all of that was of course a significant burden of high grade (aggressive) diffuse large B-Cell lymphoma.

(Diffuse large B-cell lymphoma (DLBCL) is a type of aggressive, fast-growing non-Hodgkin lymphoma that originates in B cells, a type of white blood cell, and is the most common type of non-Hodgkin lymphoma.) In Mr [A]'s case his prior CT imaging (reported … before his admission) had identified **a very large** lymph node mass in his abdomen (10.7cm x14.5cm x 17cm), multiple enlarged mesenteric nodes as well as significant sized nodal mass in his chest 4.6cm x 3.5cm. At that stage a moderate amount of ascites (free fluid in the abdomen) was also noted to be present.

Question 1: The adequacy of investigation and management of Mr [A]'s intermittent confusion/delirium:

Advisor's opinion- In my view the investigation and management of Mr [A]'s intermittent confusion / delirium was not ideal in the clinical circumstances and likely fell below the expected standard of care.

His confusion and episodes of drowsiness were documented by the attending nursing staff and on the consultant physician ward round; however, no specific cause appears to have been documented in the notes available to me or any apparent consideration for additional investigations or targeted treatment.

I would acknowledge delirium will often have a multifactorial basis and in this case acknowledge the treating team may have reasonably attributed his confusion to his significantly low serum sodium (his hyponatremia of Na 117-121), his pain and his new medications (oxycodone, tramadol, pregabalin, zopiclone). In my view however the progressively important hypovolaemia as part of this delirium was not appreciated, adequately examined for and ultimately was not treated. In my view this was one missed opportunity for a systematic approach to identify a further part of the puzzle contributing to Mr [A]'s confusion.

What was the standard of care/accepted practice at the time of events? Please refer to relevant standards/material- Delirium/confusion is acknowledged to represent a broad clinical syndrome with a wide differential diagnosis. In general a systematic approach that addresses both the underlying cause and the acute symptoms would be expected to be followed. Recognition involves an appreciation of a rapid onset with a fluctuating course with inattention, disorganised thinking and altered consciousness. This can be assessed using formal bedside screening tools such as the CAM tool — (Confusion Assessment Method) or the 4AT tool — though often in real world clinical practice the nursing staff will simply comment that the patient appeared confused or more drowsy than expected for the circumstances. Once identified consideration for the appropriate investigations to determine the cause would be anticipated acknowledging the differential diagnosis is often

multifactorial with a significant degree of clinical judgement being applied to dictate investigation in the circumstances. Because the confusion can be intermittent, good communication between nursing and medical staff is important to facilitate recognition and review. Initial workup would be considered to involve: History (including medications, recent and current illness, medication, drug and alcohol use), physical and neurological exam and more detailed cognitive assessment. Initial investigations would be anticipated to include blood tests including FBC, U&Es, LFTs, calcium, CRP, glucose, thyroid function, B12/folate, Urinalysis and culture, Chest X-ray (rule out infection). ECG (electrolyte disturbances, ischemia) and a specific Medication review —especially anticholinergics, opioids, & benzodiazepines. Depending on the clinical course and results more advanced imaging may be indicated. Management would then be targeted to the identified cause often initiating treatment based on initial best clinical assessment pending results of investigations.

Was there a departure from the standard of care or accepted practice?

Yes, in my view there was a departure from the expected standard of care. I would regard that as a mild departure early on in his presentation increasing to a moderate departure as his confusion symptoms intensified and began to indicate significant clinical deterioration was occurring.

No specific delirium assessment tool appears to have been utilised in this case, though I would not regard that in of itself as a departure, though its use may have had the effect of focusing the team more specifically on confusion as an important vital sign (cerebral function) needing active review. Nursing staff did however first document the presence of confusion over the evening of [Day2]/5-[Day3]/5 with the overnight on call house officer requested to review but note made *"was busy with another patient then left ward".* The following morning ward round does not record any overnight confusion issues or consider additional investigations. The following night [Day3]/05 a further episode of confusion was again documented in the nursing records with urinating on the bedside floor described. A bell mat was placed with documentation *"For close watching"* (Bell Mat -a mat device placed on the floor which will alarm and alert nursing staff when a patient climbs out of bed and stands on it — used to manage confused patients where concerns of falls or wandering etc exist). I note a nursing note entry made which states *"Due to negative workload all A&D not complete though patient stable physically"*. The following morning registrar ward round notes the confusion and urination though does not comment on the significance or potential causes. A number of additional investigations were requested including an ECG, and urinalysis (for Na, osmolality, cortisol). Ongoing confusion was again documented on the consultant ward round the following day on [Day5]/05 though again specifics of clinical thinking / cause or any documented review of fluid status (fluid balance, patients weight change, JVP, Cr, Ur) is not present. Recognition of Mr [A]'s poorly controlled pain was made with a pain team referral initiated. Mr [A] was again documented to be drowsy, wandering, confused and talking to himself on the evening of [Day5]/05. A house officer review was requested at 2300hrs but was documented as *"House officer was informed but patient was asleep when house officer went to see him"* There are no medical notes indicating a review occurred at that stage. Mr [A] pulled out his ascitic drain at approximately 0100hrs with ongoing confusion – *"appears delirious"* was documented in the nursing notes. A House officer review was requested and performed at approximately 0105hrs - though a retrospective note was not made until 0420hrs (after his death). In my view that review fell well below the expected standard. The details of that medical review is addressed specifically in question 4 below.

How would the care provided be viewed by your peers? Please reference the views of any peers who were consulted.

It is acknowledged that Mr [A]'s case was a complex one with a severe underlying pathology, multiple evolving symptoms, a less usual cause of hyponatraemia (due to initial 3rd spacing due to the lymphoma) and a number of clinically distracting circumstances — including the need for a biopsy for a definitive diagnosis, and the presumed cause of his hyponatraemia. This appears to have been further complicated by his more acute deterioration overnight in a provincial hospital staffed at night by a resident medical officer (junior doctor) under what appears to have been perhaps a less than ideal clinical workload setting - perhaps without time for an adequate review or escalation for advice to a senior supervising doctor. The details of this are however unknown to me and have not been specifically commented on in the provider response or by those involved in the care. I would also acknowledge the nursing staff comments around an adverse workload. It is acknowledged then that a number of mitigating circumstances were perhaps at play. That said on balance the standard of care relating to the adequacy of investigation and management of Mr [A]'s intermittent confusion/delirium would be similarly viewed by my peers as falling below the expected standard though an understanding of the mitigating factors are acknowledged and I would anticipate lead to differing views on the severity of the departure.

Please outline any factors that may limit your assessment of the events

Circumstances around the clinical workload for nursing staff and junior medical staff are unknown though assumed from the limited comments in the notes to have been challenging.

Recommendations for improvement that may help to prevent a similar occurrence in future

Use of formal confusion assessment tools in routine practice may facilitate improved assessment, recognition and communication to the treating team of confusion recognising this as an important vital sign potentially indicating clinical deterioration. The Medical review of confusion particularly overnight for on call medical staff would ideally be included in House Officer orientation and education programs.

Question 2: The adequacy of monitoring and management of Mr [A]'s electrolyte abnormalities (and fluid status)

List any sources of information reviewed other than the documents provided by HDC: UpToDate Clinical Treatment Guidelines for Hyponatraemia.

Advisor's opinion: In my view this aspect of Mr [A]'s care was not managed adequately. I would regard that as a moderately severe departure from the expected standard of care. I acknowledge this is a complex aspect of general medical care in this case. In my view however the standard of assessment for fluid status, consideration of early appropriate investigation for hyponatremia and review of those investigation results to guide appropriate treatment, fell well below that expected of a Specialist General Physician and ultimately contributed to Mr [A]'s death.

What was the standard of care/accepted practice at the time of the events? Please refer to relevant standards/material:

Mr [A] was identified as having Hyponatraemia (a low serum Sodium level (Na)). Investigation and treatment guidelines and the expected clinical practice is well summarised and available in resources such as UpToDate. A clinical guidance algorithm as to the potential causes and suggested investigations for Hyponatraemia is available on UpToDate within that topic (Determining the Cause of Hyponatraemia in Adults - attached as an appendix[1]). I would accept that algorithm as a reasonable summary of the expected investigation pathway for hyponatraemia. In the circumstances of this case I would recognise a wide differential diagnosis for the cause of hyponatraemia existed with opposing management strategies making the need for a systematic approach and appropriate early investigations even more relevant to guide the correct treatment.

Was there a departure from the accepted standard of care or accepted practice?

Yes in my view there was a significant departure from the standard of care for the diagnosis and then management of hyponatraemia. I would regard that as at least a moderate departure recognising perhaps some mitigating factors. In my view the diagnosis of the cause of Mr [A]'s hyponatraemia was not correctly identified. An early systematic approach to the diagnosis does not appear to have been followed with the initial diagnosis being made as **Hyper**volaemic hyponatraemia. This does not appear to have been reconsidered or rechallenged over the course of his admission with the correct treatment not having been initiated. With reference to the Hyponatraemia algorithm —particularly in complex situations - measurement of the urinary sodium, osmolality and cortisol should in my view have occurred very early on in Mr [A]'s journey to inform the correct diagnosis in the setting of a potentially complex clinical picture. He was admitted to Rotorua Hospital on [Day1]/05/21 at 12:20pm with the appropriate investigations having not been performed until [Day4]/05 with the results not available until 6:55pm to 7:25pm (some 3 days later). The time the results were acknowledged is not recorded on the laboratory reports provided to me - only that they were accepted by the treating physician. The available clinical notes do not mention the results, their significance or any resulting alteration to the diagnosis or importantly treatment. It is unknown to me whether these were seen and signed off before or after Mr [A]'s death. I do note the consultant ward round of [Day5]/05/21 at 1005am does not note the results of the recently available urine Na, osmolality or serum cortisol or update the working diagnosis or alter management. No fluid balance assessment is documented. I also would note that specifically a short synacthen test was requested for the day at that ward round (a blood test following the injection of a synthetic form of adrenocorticotropic hormone (called synacthen) which checks how well the patients adrenal glands produce cortisol - a vital hormone involved in sodium, potassium and fluid balance) The morning 8am cortisol result from the previous day [Day4]/05 had already been resulted however (very rapidly) at 0945hrs on [Day4]/05 —it was significantly high at 1025 indicating that adrenal insufficiency was not the likely cause of the low sodium and high potassium — and so making a short synacthen test unnecessary and inappropriate (and so I would surmise indicating those results were not reviewed on that ward round). (The synacthen was however never actually given despite its charting as I assume it would

[1]the appendix to the advice has been omitted from this report (Appendix: UpToDate : Determining the cause of Hyponatraemia in Adults)?

have been most appropriate to have been administered at 8am the following day ([Day6]/05) with Mr [A's] arrest unfortunately occurring prior to that).

I would also note hydrocortisone 200mg IV was given on [Day3]/05 at 1145am for what was felt to be a potential cause of Mr [A]'s low sodium and high potassium due to potentially adrenal infiltration by his lymphoma (causing adrenal insufficiency and so low cortisol levels). No cortisol level was however taken prior to this. This again appears in the registrar ward round notes on [Day4]/05 incorrectly stating "?Addison's disease secondary to tumour invasion" with cortisol 1025. In my view the summative effect of not identifying the correct diagnosis of progressive fluid 3rd spacing due to the lymphoma leading to **Hypo**volaemic hyponatraemia meant the inappropriate use of diuretics (frusemide and spironolactone) no documented attention to clinical fluid balance and the lack of use of fluid replacement to manage the progressive intravascular volume depletion. In my view the delayed cortisol testing, use of presumptive hydrocortisone, and a very late inappropriate synacthen test further indicated the wrong diagnostic pathway due to a lack of following a systematic approach to the assessment and diagnosis of hyponatremia. In my view this then set the stage for a rapid clinical decompensation following the drainage of his ascitic fluid. (Initially 2200mls of ascitic fluid on the morning of [Day5]/05 to a total of 3550mls prior to his arrest.) The removal of that volume of fluid in a man with likely already compromised intravascular volume and with a large tumour burden to rapidly produce 3rd spacing (movement of fluid from the vascular compartments into the free abdominal space) in my view was causative to his PEA arrest. His available history documented poor oral intake over the preceding 5 days, recurrent vomiting, constipation, progressive weight loss likely indicating dehydration (88kg on [Day1]/05 to 84kg on [Day4]/05) (weight not done on the [Day5]/05 despite being indicated on the chart as required and no fluid balance records being completed by nursing staff) - and a high urine osmolality (667) with very low urinary sodium (7) when testing was done late on [Day4]/05 (3 days after admission with a very low Na). In my view these all argued strongly for a diagnosis of **hypo**volaemic hyponatraemia (with acute kidney injury eGFR 31 - previously near normal) due to 3rd spacing secondary to his large lymphoma tumour burden. With the benefit of hindsight his peripheral oedema was likely contributed to by his lymphatic and venous compromise due to his tumour burden rather than being due to volume overload (**hyper**volaemic hyponatraemia). In my view following the hyponatraemia guideline or applying the similar correct General Medical Critical thinking would have identified the correct diagnosis at an early stage in Mr [A]'s illness and led to a very different treatment pathway and outcome.

How would the care provided be viewed by your peers? Please reference the views of any peers who were consulted

I would regard my peers as reaching similar conclusions. I would acknowledge this was a complex clinical case with less usual pathophysiology underlying Mr [A]'s Hyponatraemia. A reasonable initial diagnostic impression of things being due to fluid overload and ascites could be accepted - however the delay to appropriate diagnostic confirmatory testing, the absence of apparent consideration of those results and the lack of challenging the original clinical impression however meant the expected standard of care was not ultimately delivered. The poor attention to fluid balance and fluid status assessment in what must be a vital part of the fluid management and treatment of hyponatraemia in the circumstances of this case is also difficult to overlook.

Please outline any factors that may limit your assessment of the events

The laboratory result form provided does not allow an assessment of the **time** of acknowledgement of the requested results. It is unclear if this was before or after Mr [A]'s death.

Recommendations for improvement that may help prevent a similar occurrence in future

It is unclear to me why the results were not actioned or considered in the available clinical record. It is also unclear if there was a communication breakdown between the original requesting registrar and the consultant in charge around the ordering of the tests. A robust ongoing problem list with differential diagnosis and supporting results may have assisted to clarify critical thinking.

Question 3: The timeliness of scheduling of Mr [A]'s abdominal mass biopsy and paracentesis following his admission to Rotorua Hospital.

List any sources of information reviewed other than the documents provided by HDC:
NZ Ministry of Health targets

Advisor's opinion: In my view this aspect of Mr [A]'s care was reasonable and met the expected standard of care - particularly without the benefit of hindsight of knowing his ultimate histology diagnosis (a high grade aggressive lymphoma). I would not regard this aspect as being critically causative in his death. His initial Ultrasound was performed on 21/04/21 due to his development of symptoms, followed by his CT on 28/04, and his first specialist appointment on 05/05/21. He was admitted to Rotorua Hospital on [Day1]/05/2021 — […]. He underwent his biopsy on [Day5]/05 some 4 days later.

What was the standard of care/accepted practice at the time of events? Please refer to relevant standards/material.

The New Zealand government sets standards for time to assessment and care. In 2021, New Zealand guidelines aimed for patients with a high suspicion of cancer to receive a specialist assessment within two weeks of referral and their first cancer treatment within 62 days of the hospital receiving the referral, with treatment commencing within 31 days of the decision to treat.

Was there a departure from the standard of care or accepted practice?

In my view a departure from the standard of care for time to treatment did not occur.

How would the care be viewed by your peers? Please reference the views of any peers who were consulted.

I would regard my peers as holding similar views.

Please outline any factors that may limit your assessment of the events.

Nil.

Recommendations for improvement that may help to prevent a similar occurrence in future

Nil.

Question 4: The adequacy of assessment and escalation of care when Mr [A] was first noted to be hypotensive in the early hours of [Day6] May 2021.

Advisor's opinion:

I my view this aspect of Mr [A]'s care fell substantially below the expected standard of care. I would regard that as a moderate departure from the expected standard of care though acknowledge competing house officer clinical priorities and workload may perhaps have been mitigating factors from a severe departure.

What was the standard of care/accepted practice at the time of events? Please refer to relevant standards/material.

The use of the Early Warning Score (EWS) system is a National framework supported by bedside documentation and recording charts to aid in recognition of clinical deterioration and to guide staff in seeking appropriate clinical review and ongoing monitoring. Its use is now well established in clinical practice in NZ hospitals. Once informed of a patient's deterioration it also guides attending medical staff regarding the prioritisation of review and when to consider escalation to any senior colleagues.

Was there a departure from the standard of care or accepted practice?

Yes, in the circumstances of this case a number of significant and severe failings can be identified. I note the provider response regarding this aspect of care which I have reproduced below:

"Nursing staff monitored Mr [A] by assessing vital signs and using the Early Warning Score (EWS) system.

From admission [Day2]/05/21 until 2015 hours on [Day5]/5/21 the staff took 3 sets of observations consistent with Mr [A]'s presentation. For this period his baseline observations remained stable and did not score higher than 3. In the early hours of [Day6] May 2021 (00:45) Mr [A] scored an EWS of 3 (previously he scored 0). Vital signs were repeated within one hour, and when he still scored an EWS of 3 the house officer was called to review.

At approximately. 0105 Mr [A] pulled out his abdominal drain. The nurse contacted the night house officer for a review which occurred. A retrospective note was written at 0420hrs noting BP 74/54 post vomit. Ascitic drainage since insertion at 0825hrs that morning of 3550mls was recorded. An incomplete fluid balance chart was also noted. Patient was recorded as walking back to bed having been redirected due to confusion. Repeat BP post back in bed 92/60. Instructions given to give antiemetic for nausea and encourage oral fluids (at least 2 cups) and recheck obs again in 30mins or sooner if any deterioration clinically. Requested to be paged again at 0130hrs as had to leave to review other patients in hospital. Was not paged at 0130hrs and rang back ward at 0200hrs. Informed BP 93/54 and was drinking fluids. Requested ongoing Q30min obs. Cardiac arrest call initiated at 02:35. Baseline BPs over admission been 120-150/60-80."

There is a retrospective clinical note from the house officer of the patient review written at 0420 hours following the 777 event and after Mr [A] deceased. This note details the request for the nurse to continue 30 minute observations and encourage fluids.

The EWS chart does show the 30 minute observations taken by the nurse on shift.

These observations have not been scored but initially would have rated as 6 (Medical Officer review within 20mins). A recording at 0135hr was documented as BP 54/42- as per the EWS guideline this fell within the blue zone indicating "immediately life-threatening critical illness" recommending *"Call 777 if acute deterioration, review treatment escalation plan consider ICU referral /palliation as indicated and On site consultant review".* This did not appear to occur. The next documented set of obs appears to be 0205hrs where the BP was 93/54.

As the nurse is taking the final set of observations at 0240 Mr [A] becomes unresponsive and the 777 emergency process is activated followed by active resuscitation for approximately 75 minutes.

In my view the EWS system was recurrently not followed and appropriate escalation did not occur – from House Officer to more senior input. Further urgent reviews should have occurred following the House Officers initial (and only) review at 0105hrs. In my view multiple opportunities for intervention were then missed over the next some 1.5hrs before his arrest. Further escalation should and did not occur as per the guideline. In my view the House Officer review and treatment at 0105hrs likely fell below the expected standard of care. In the setting of significant clinical deterioration, low BP, and confusion post a procedure earlier in the day the advice to encourage oral fluids and give an antiemetic was not ideal and in my view fell below the standard expected of a resident medical officer on night shift. In my view the gravity of Mr [A]'s clinical circumstances was not recognised at that stage by the attending doctor and hence appropriate senior advice was not sought. It is my reflection that such circumstances involving overnight junior medical staff not recognising what they do not know — and so not seeking senior input overnight — can be a common theme in HDC case reviews when things unravel overnight. When standards and guidelines are not followed (such as the escalation to consultant review guidance for blue zone observations) regrettably such fail safes are circumvented.

How would the care provided be viewed by your peers? Please reference the views of any peers who were consulted.

I would regard my peers as reaching similar views. I would acknowledge the retrospective nature of the clinical entry and the apparent competing clinical responsibilities of the night House Officer may have been mitigating factors as to the adequacy of the clinical review and the ability to provide the standard of care expected. The exact circumstance around this are however unknown.

Please outline any factors that may limit your assessment of the events

As above.

Recommendations for improvement that may help to prevent a similar occurrence in future.

Review of overnight Resident Medical Officer workloads and escalation culture + nursing staffing ratios and workload. No reports from the available staff are available to me but I note a number of red flags in the clinical documentation suggesting high workload as being contributory. It is unknown if this is a recurring culture or a "perfect storm" situation.

Question 5: The adequacy of clinical documentation noting the retrospective note completed by the OCHO following Mr [A]'s death.

Advisor's opinion:

Overall the clinical documentation was not ideal with a number of aspects falling below the expected standard. I would regard those aspects as perhaps a mild to moderate departure acknowledging the reasons for the lack of documentation relate more to the clinical management rather than simply being due to not documenting what was known.

What was the standard of care/accepted practice at the time of events? Please refer to relevant standards/material

EWS National Observation Chart, Fluid Balance Charts

Was there a departure from the standard of care or accepted practice?

Yes. Most obviously there was poor attention to fluid balance and fluid status assessment and recording. This has been acknowledged in the provider's response noting further follow-up, education and review via nursing pathways should occur.

Whilst retrospective clinical note entries are not ideal I would not regard that as a major failing in documentation as clinical care must be prioritized ahead of note writing with night shifts traditionally being a busy, high stress, low resource circumstance for House Officers.

The failure of documentation and use of the EWS documentation is discussed above. The failure to review, document and act on the investigation results is discussed above also.

I would also note the providers comments: "Multidisciplinary input is very important in complex patients. While ward rounds cannot be done at the same time, any recommendations/suggestions by one team should be documented and passed to the House Officer on the ward. That House Officer could then a point of contact for the second team to receive information and recommendations. Generalised overall approach to the patient with adequate management of pain is highly recommended."

How would the care provided be viewed by your peers? Please reference the views of any peers who were consulted.

I would anticipate similar views.

Please outline any factors that may limit your assessment of the events

Nil.

Recommendations for improvement that may help to prevent a similar occurrence in future

Issues discussed more specifically above as the less than ideal documentation in many ways reflected the underlying root causes.

Question 6: Any other comments on Mr [A]'s care

Advisor's opinion: It is unclear from the 777 call documentation at what stage of the remarkably prolonged (75 minute) resuscitation IV fluids (volume replacement) was started in the setting of a recurrent PEA arrest. (A type of cardiac arrest where the heart is still functioning but there is often insufficient blood in the heart for it to pump ie not enough volume present in the body's blood vessel circulation. (Causes for this are popularly referred to in Resuscitation Medicine as the "H"s & "T"s — one "H" being **Hypo**volaemia). Blood tests taken the afternoon before his arrest indicate a stable potassium level similar to that throughout his admission (6.1) whilst gases taken during the arrest indicate an initial further elevation to 6.5. 1L of N/Saline is documented as having been started at 0359 with an indication it is the 2nd bag. The first bag is not recorded anywhere nor is the start time. That second bag would be noted to have been started some 1hr and 20mins after the start of the arrest. Appreciating the challenges of the arrest situation in the early hours of the morning with limited onsite senior staff in a provincial hospital an awareness of the need for significant volume replacement early in Mr [A]'s PEA arrest and in the unique circumstances of his case with recent large volume paracentesis was perhaps not ideal. The adequacy of training of the overnight junior staff resuscitation teams is unknown**.**

By signing this report, I agree to HDC correcting any formatting, spelling, or grammar issues on the proviso that the substance of the report and any quoted material remains unchanged.
Signature: 
Name: Dr Richard Shepherd
Date of Advice: 11 April 2025

In April 2026, Dr Shepherd provided additional independent advice to this Office in response to further information received from Health NZ.

Independent clinical advice to Health and Disability Commissioner

Complaint:	Click or tap here to enter text.[Mr A] / Te Whatu Ora - Lakes - Rotorua Hospital
Our ref:	**CAS-14609-C4B6F2**
Independent advisor:	Richard Shepherd

I have been asked to provide clinical advice to HDC on case number CAS-14609-C4B6F2. I have read and agree to follow HDC's Guidelines for Independent Advisors.

I am not aware of any personal or professional conflicts of interest with any of the parties involved in this complaint.

I am aware that my report should use simple and clear language and explain complex or technical medical terms.

Documents provided by HDC:	1. Letter of complaint 2. Notification response letter from Te Whatu Ora -Lakes-Rotorua Hospital's dated 25 September 2025 3. Copy of previous expert advice dated 15 April 2025 4. Coronial autopsy report dated […] May 2021
Referral instructions from HDC:	Te Whatu Ora-Lakes-Rotorua Hospital 1. Does the information provided in the response from Te Whatu Ora-Lakes-Rotorua Hospital/Dr […] change any of your initial advice? If yes, please provide your updated advice. 2. Any other comments you wish to make

Question 1: Does the information provided in the response from Dr […] change any of your initial advice? If yes, please provide your updated advice.	
List any sources of information reviewed other than the documents provided by HDC:	Nil additional.
Advisor's opinion:	No there is no additional information provided in the response from Dr […] that would alter my initial advice. In response to a number of specific areas Dr […] raises I would make the following comments:

	1/ Cortisol Results and Testing: I am not able to agree with Dr […] comments. From the records available to me Mr [A]'s Cortisol test was performed at 8:05am – the standard screening test time for cortisol testing (not a random sample done near midday as stated by Dr […]). The result was high at 1029nmol/L (ref range 170-500) (not normal or slightly raised as stated by Dr […]). General guidelines would indicate an 8am Cortisol level <100-140nmol/L strongly suggests adrenal insufficiency, levels 140-500nmol/L indeterminate and potentially requiring a synacthen test to confirm, levels >500 would be regarded as making adrenal insufficiency unlikely, while a level >1000 would essentially exclude adrenal insufficiency- with a very high sensitivity for ruling out adrenal insufficiency and an extremely high negative predictive value. At this level synacthen testing would not normally be indicated unless there was concern for rare central causes with preserved basal cortisol but impaired stress response (not the situation here). Whilst the CT scan and post mortem findings did indicate the presence of lymphoma in and around the adrenal glands in my opinion the cortisol result >1000 was strong clinical evidence of no functional deficiency of cortisol at such a high tested level and a synacthen test was not relevant and detracted from the relevant underlying correct causes and application of the hyponatraemia diagnostic algorithm (or similar diagnostic reasoning). From the provided laboratory date and time for clinician sign off logs this result was reviewed and signed off on [Day4] May 2021 at 10:03am (blood sample taken [Day4] May 2021 at 8:05am, result available at 9:45am). 2/Volume depletion and hypovolaemia hyponatraemia. I am not able to agree with Dr […] comments. In practice BP and pulse rate would be considered late and non specific markers for hypovolaemia in this setting (not acute blood loss / hypovolaemia shock) and would have very low diagnostic valve. They are useful for severity and resuscitation decisions but poor discriminators for progressive hypovolaemia over time where cardiovascular compensation is able to occur

	(the circumstances of this case). Over the course of Mr [A's] admission a down trending in BP values is visible (ranges 140-170/70-90 at the beginning of his admission to 120-130/60 in the last two days of his admission. I also note no observations are recorded over the 5.5 hour period from 2015hrs [Day5]/05/21 to 0145hrs [Day6]/05/21 when his BP went from 125/65 to 74/54). Even during the extended pre PEA arrest period of BPs <80 Mr [A']s pulse rate did not go above 100 indicating specifically in Mr [A]'s situation the poor non specific validity of pulse rate. Osmolality testing, urine sodium and a clinical evaluation of volume status (weight comparisons, JVP, creatinine/Ur changes, oedema/third spacing, postural BP response, response to fluid challenge) offer considerably higher diagnostic validity and for this reason are fundamentals in the diagnostic algorithm for evaluation of hyponatraemia - included in the appendix of my original advice. Mr [A]'s urine sodium test was 7mmol/L – i.e. very low and a very strong indicator of renal conservation of sodium (urine osmolality also 667 (very high)– i.e. significant hypovolaemia (not simply "*a degree"* of dehydration. From the records available to me Mr [A] showed a significant pre renal acute kidney injury (eGFR 29-31 – (previously normal eGFR 82 on 20/04/2021) – this represents an approximately 65% decline in his renal function – a severe reduction) – further compelling evidence of hypovolaemic hyponatraemia. His weight was also progressively rapidly declining adding to the weight of evidence (fluid balance records were not complete). I am then unable to agree with Dr […] comment that "no significant change in his renal function" had occurred. Mr [A] had an acute pre renal kidney injury over his course of his care from Taupo Hospital to Rotorua Hospital. The weight of medical evidence available to me including the post mortem result indicates a pre renal hypovolaemia basis to this. In my view this was not adequately examined for, considered, or recognised from the results available and was therefore not specifically treated and was treated incorrectly (as **hyperv**olaemia -with diuretics (frusemide & spironolactone) and fluid restriction).

	3/ SIADH – I am not able to agree with Dr […] comments. In my opinion the diagnosis of, or consideration of SIADH (Syndrome of Inappropriate Antidiuretic Hormone Secretion), would not be consistent with the clinical picture in the circumstances of this case. If following the hyponatreamia diagnostic algorithm (or similar clinical physiological reasoning) the urine sodium of 7 would essentially exclude that diagnosis. Administration of IV fluids (N/Saline) would be expected to elevate that urine sodium level (hence the algorithm recommendation to infuse 1L and remeasure if considering SIADH with potentially other causes as being present). Any N/Saline given would be expected to elevate that urine sodium level and so not cause any confusion when the level returned so low (at 7). I am not however able to reference any considerations in the clinical records available to me nor any references to the results of the relevant investigations (osmolality, urine sodium, cortisol) or to the use of N/Saline if this was actively being considered at the time. In my view I am unable to provide a clear contemporaneous chain of appropriate clinical reasoning and action from the records available to me. 4/&5/ Additional potential causes of Hyponatraemia. I am not able to agree with Dr […] comments. Paraneoplastic hyponatraemia has very rarely been associated with lymphoma via the SIADH phenotype (cytokine mediated ADH release or ectopic ADH production or CNS lymphoma involvement). Again with application of the hyponatraemia algorithm and the results of the available tests (fluid status, urine sodium, osmolarity, cortisol) this would not be relevant in the circumstances of Mr [A]'s case and is not supported. Similarly opiate and antiemetic induced hyponatraemia, also very rare, would also be expected to occur via the AHD secretion SIADH phenotype physiological pathway. Again this is simply not supported by the results and clinical circumstances contained in Mr [A]'s records - as per the SIADH discussion above.

	6/ Cause of Death. I am not able to agree with Dr […] comments. Dr […] consideration of the cause of death includes potentially being IVC occlusion, partial occlusion of the aorta, and / or pulmonary embolism. In my opinion I would agree with the post mortem report and findings as to the cause of death. This is stated as: *"**SUMMARY:*** *Death was due to cardiac arrest (pulseless electrical activity as per hospital records) due to advanced intra-abdominal lymphoma (diffuse large B-cell lymphoma).* ***PRINCIPAL PATHOLOGICAL FINDINGS:*** ***1.** Advanced malignant lymphoma (diffuse large B-cell lymphoma).* ***2.** Coronary atherosclerotic disease.* ***3.** Abdominal aorta, severe atherosclerosis.* ***4.** Liver, mild steatosis.* ***CAUSE OF DEATH:*** ***Part I a.** Cardiac arrest.* ***b.** Advanced malignant lymphoma."* No thrombus (pulmonary emboli) is noted within the respiratory system post mortem report and is specifically commented on in that report. The body of the report does reference "*There is a large lobulated solid white tumour mass measuring 230 x 140 mm involving the superior mesenteric and para-aortic nodes throughout the length of the abdominal aorta that surrounds and compresses the inferior vena cava and aorta."* In my view this would not be consistent with "occlusion" to those vessels as a pathological cause of death. I am unable to rationalise the clinical records, results and circumstances to provide a cohesive pathophysiological mechanism for

	occlusion as the cause of death. No acute thrombus (occlusive) cause was noted in the IVC or aorta on post mortem. A progressive tumour related external compression to the IVC (IVC obstruction syndrome) (or aorta) to the point of causing vessel obstructive shock and PEA arrest in my opinion is not supported by the available clinical records or post mortem findings. These findings might be expected to include progressively worsening bilateral pedal oedema, rapidly worsening ascites, peripheral shutdown, and post mortem findings consistent with IVC / aortic obstruction such as congestive hepatomegaly, renal venous congestion, acute tubular necrosis, dilated distal venous system, bowel ischaemia and thrombus obstruction in the vessel(s). On balance of probabilities then in the circumstances and clinical evidence described I would not regard the cause of death as being due to obstructive shock (IVC or aortic compression). A component of external IVC c*ompression* by the lymphoma was certainly present and contributing to Mr [A]'s symptoms and clinical presentation however I am unable to provide a cohesive clinical narrative that I am able to fit to the available clinical data to postulate an *obstruction* causation as the likely cause of death. I would then agree with the post mortem causes of death conclusions above. 7/ The arrest Rhythm PEA (Pulseless electrical Activity – a present beating heart rhythm but insufficient blood output) vs VF and the potential causes. I am not able to agree with Dr […] comments. From the arrest records available to me Mr [A]'s initial arrest rhythm 5 minutes into his CPR was not shockable (PEA). A shock was delivered at 0248hrs (before any adrenaline was ever given) though the rhythm is not described or available to view. (Note emergency call initiated at 0235hrs and CPR commenced at 0240hrs and first adrenaline given at 0250hrs) – in keeping then with the hypotensive periarrest observations on file over 50 minutes which then progressed to no BP, and unconsciousness at 0235hrs - but a sustained cardiac rhythm i.e PEA arrest. Multiple rhythm checks over the following 28 minutes

	from 0245hrs to 0313hrs were all PEA non shockable rhythms over which time severe metabolic acidosis occurred and further degeneration of Mr [A]'s cardiac electrical function to VF arrest would be expected (and did) occur. A further cluster of shocks was then given to treat this at 0316hrs, at 0318hrs and at 0337hrs. In my opinion this arrest pattern indicates PEA as the primary disturbance issue with the VF pattern occurring late and due to the advancing severe metabolic disturbance from ongoing accumulative tissue hypoxia, and extensive (indicated) adrenaline use (11 doses in total). This pattern strongly suggests circulatory failure initially without primary electrical instability (VF). In the circumstances hypovolaemia and obstructive causes (PE, Thrombus, IVC obstruction) would be most likely to be considered (returning to the "H"s and "T"s of resuscitation*) (* A structured way to identify and treat reversible causes of cardiac arrest "H"s = Hypovolaemia, Hypoxia, Hydrogen ions (acidosis) Hypo/Hyperkalaemia, Hypothermia and the "T"s = Thrombosis (Pulmonary embolus, myocardial infarction (heart attack), Cardiac Tamponade) Tension pneumothorax, Toxins (poisoning, Overdose). I note no thrombus was found on post mortem and the clinical picture and post mortem does not fit with a rapidly progressive tumour causing progressive external compression and then functional obstruction to the IVC or aorta). Mr [A] had however recently undergone a significant volume paracentesis procedure without post volume replacement on top of an already compromised hypovolaemic circulatory system (evidenced by his lymphoma third spacing (ascites), his acute kidney injury, his low plasma and urine sodium, high urine osmolality, dropping BP and declining weight) and in the presence of a degree of IVC external compression. In my view this post-paracetesis circulatory dysfunction syndrome was the accumulating final straw. The initial hypotensive peri-arrest from 0145hrs, the initial PEA rhythm at 0235hrs and the prolonged PEA phase of the very lengthy resuscitation indicates an ongoing failure to restore effective circulation and suggests a persistent underlying cause not corrected during resuscitation (the hypovolaemia). The PEA in my view was the first and dominant pathology driving Mr [A]'s arrest and is

	supported by the available weight of clinical evidence discussed above. I am not then able to agree with Dr […] comment and so to provide a clinical narrative that fits with the available clinical evidence and post mortem findings to support his comment: ie "*the documents surrounding the arrest suggest there was actually a shockable rhythm, shocked twice and the pulseless rhythm followed this as it often does prior to death"* and that *"this recorded arrest is not consistent with the reviewer opinion…..that there was NOT an unrecognised chronic dehydration process".*
What was the standard of care/accepted practice at the time of events? Please refer to relevant standards/material.	No change to previously provided opinion.
Was there a departure from the standard of care or accepted practice? • No departure; • Mild departure; • Moderate departure; or • Severe departure.	No change to previously provided opinion.
How would the care provided be viewed by your peers? Please reference the views of any peers who were consulted.	No change to previously provided opinion.
Please outline any factors that may limit your assessment of the events.	No change to previously provided opinion.
Recommendations for improvement that may help to prevent a similar occurrence in future.	No change to previously provided opinion.

Question 2: Any other comments you wish to make	
List any sources of information reviewed other than the documents provided by HDC:	

Advisor's opinion:	From an overall comments perspective I am not able to agree that Mr [A]'s death was a sudden and catastrophic progression of his terminal illness. This may have been noticed suddenly but in my view from the medical information available to me it did not occur suddenly. Mr [A] did have a very serious underlying lymphoma with a significant disease burden and a deteriorating clinical status. Whether he would have been well enough to have been offered chemotherapy treatment in time, or have tolerated the significant potential toxicity of that treatment to have allowed an improvement in his prognosis is speculative and ultimately unknown. In my opinion it was likely skewed significantly towards the poor prognosis side with time not being on Mr [A]'s side. In my view however his PEA cardiac arrest and unsuccessful resuscitation was not an inevitable progression of his illness when it occurred on the date and time that it did. Mr [A]'s case was a medically complex one with potentially multifactorial causes for his hyponatraemia, his delirium and his deteriorating clinical condition. In that setting a systematic reasoned clinical approach to the differential diagnosis was vital. Attention to the detail of timely and adequate investigation, reviewing of those results and so informing his treatment to offer the most ideal circumstances to slow his deterioration was necessary to buy the time needed for his tissue diagnosis to be made and then any chemotherapy treatment decisions – or palliation decisions – to be made. I am not able to provide an adequate clinical narrative with cohesive supporting clinical and post mortem evidence that expected standard occurred in this case. I would acknowledge even the most ideal of care may not have altered the ultimate terminal outcome for Mr [A] due to the severity of his illness and his age - but it may have altered the timing and circumstances for that terminal event for both Mr [A] and his family. In my view there are learning opportunities identified for the involved team.

By signing this report, I agree to HDC correcting any formatting, spelling, or grammar issues on the proviso that the substance of the report and any quoted material remains unchanged.
Electronically Signed. Signature:
Name: Richard Shepherd
Date of Advice: 21 April 2026