CITATION: *Inquest into the death of John Roberts* [2017] NTLC 024

TITLE OF COURT:	Coroners Court
JURISDICTION:	Darwin
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FINDING OF:	Judge Greg Cavanagh
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REPRESENTATION:

Counsel:

Assisting:	Jodi Truman
Family	Peter Bellach
Top End Health Service	Stephanie Williams

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IN THE CORONERS COURT
AT DARWIN IN THE
NORTHERN TERRITORY
OF AUSTRALIA

No. D0029/2016

In the matter of an Inquest into the death of
JOHN ROBERTS
ON 20 FEBRUARY 2016
AT DARWIN CORRECTIONAL PRECINCT, HOLTZE

FINDINGS

Judge Greg Cavanagh

Introduction

1. John Roberts ("the deceased") was born at Milikapiti (also known as Snake Bay) in the Northern Territory on 19 December 1978. Out of respect for the family and the cultural practice of avoiding use of the Christian name of an Aboriginal person who has passed away, I will hereafter refer to the deceased as Mr Roberts (or the deceased), with the exception of the formal findings.

2. The deceased's father is Tracy Puruntatameri and his mother is Stephanie Roberts. His uncle, Claver Warlamerapui, and his sister, Rita Roberts, were both in attendance at the inquest to represent the family and I thank them for the respect they showed to the coronial process.

3. The deceased died at the Darwin Correctional Precinct ("the prison") on 20 February 2016. He was 37 years of age at the time of his death. The deceased had been on remand at the prison following his arrest on 29 October 2015. He was remanded in custody without bail from that day and was transferred to the prison on 30 October 2015. He remained on remand up until his death.

4. Following his remand, the deceased underwent the usual checks and searches that occur for any person who is received at the prison. These

included a medical assessment. At the time of his medical assessment it was noted that the deceased was on medication being Olanzapine and Zuclopenthixol, both are anti-psychotic medications. As a result, the deceased was referred to the Forensic Mental Health Team ("FMHT").

5. I received evidence that the deceased had in fact been diagnosed as suffering from schizophrenia since 1995. He had a long history of mental health issues and was well known to mental health services including numerous admissions to the psychiatric unit at the Royal Darwin Hospital ("RDH"). He had been prescribed anti-psychotic medication for many years. It also appears however that there were times when the deceased was not compliant with his medications. He also had a history of alcohol and drug abuse, mostly cannabis, which had a negative effect upon his mental health.

6. It appears that the deceased was initially prescribed Flupenthixol, but this was changed to Olanzapine in June 2002. He had therefore been receiving Olanzapine for over a decade. In September 2002 he was also prescribed Zuclopenthixol as a "depot" medication. This combination of prescribed medication was therefore a longstanding one; other than his lengthy periods of non-compliance.

7. Upon his remand to prison, and following his assessment and review by the FMHT, this medication regime was continued. Unfortunately the deceased's psychotic symptoms continued and appeared to worsen. As a result on 11 December 2015 his Olanzapine dose was increased to 15mgs twice per day, i.e. in the morning and at night. Reviews by the FMHT continued over the coming months, however the deceased continued to exhibit psychotic symptoms despite his high dose of Olanzapine and despite being on two (2) anti-psychotics.

8. In relation to the deceased's worsening psychotic state, I received evidence from Consultant Psychiatrist; Dr Ranjit Kini, who was involved in the psychiatric care of the deceased. Dr Kini gave evidence that it was his

opinion that the reason the deceased's psychiatric symptoms were not improving is that the deceased was unfortunately suffering from treatment resistant schizophrenia:

> "Unfortunately for a small proportion of patients who have what is termed treatment resistant schizophrenia it is well known that despite being on antipsychotic medication they can continue to have residual psychotic symptoms. So even in the case of the deceased in the community, for example, when he was known to be compliant (with) the antipsychotic medication; there were residual psychotic symptoms for a long time. So it is known that people, despite being compliant with medications, can continue to present with residual psychotic symptoms".

9. The deceased's incarceration continued and so too did his medication regime. On 20 February 2016 however, the deceased was seen by fellow prisoners to have a seizure whilst sitting cross legged on the floor outside of cell 7 in Block 5. He was immediately attended to by other prisoners and then medical staff. Despite attempts by medical staff to revive him, he was pronounced deceased at 4.52pm that day, only 21 minutes after his seizure was noted to have taken place.

10. As a result of the deceased being incarcerated at the time of his death, this inquest was mandatory. The investigation into Mr Roberts' death was undertaken by Detective Sergeant Isobel Cummins of the Major Crime Squad; a very experienced Major Crime Detective. I thank her for her investigation and her detailed brief of evidence. Having considered that brief and the oral evidence given before me, I now outline what I consider to be the relevant issues in this inquest.

ISSUES:

Findings at autopsy

11. The body of the deceased was examined by Forensic Pathologist, Dr John Rutherford on 23 February 2016. Dr Rutherford provided a detailed report setting out the various examinations he conducted upon the body during the course of his autopsy. Following his detailed examination both externally

and internally of the deceased's body, Dr Rutherford found that "(t)here was no demonstrable anatomical cause of death":

> "That means that in terms of structure of the body there was no abnormality of sufficient severity to account for death. For example, there was no coronary artery disease of any severity, there was no obvious natural heart disease involving the muscle, there was no pulmonary embolism, there was no brain haemorrhage. There was nothing that would cause a sudden death which was otherwise unexplained".

12. As a result Dr Rutherford turned to consider the results of a toxicological investigation that had been done of the post mortem bloods he had sent for analysis. In this regard I received into evidence a toxicology report from Heather Lindsay, Senior Forensic Scientist of Forensic Science SA, dated 22 July 2016.

13. The toxicology report of Ms Lindsay detailed her comprehensive screening and analysis of the post mortem iliac blood sent to her. Ms Lindsay reported that she had detected in the blood approximately 0.40mg of Olanzapine per litre of blood. This was in addition to therapeutic concentrations of Zuclopenthixol also detected in the blood.

14. Importantly in relation to the concentration of Olanzapine found, Ms Lindsay noted as follows:

> "1. Modification of this interpretation of results may be required depending on autopsy findings, previous drug history and potential post-mortem drug concentration changes. Care should be taken when comparing post-mortem blood concentrations to clinically derived data, particularly if the blood is sampled from the central regions of the body (e.g. heart of abdominal cavity).
>
> 2. Interpretation of individual drug concentrations above does not take into account the potential of combined drug interaction".

15. Taking into account the care that Ms Lindsay noted needed to be taken with the toxicology results; of significance to this death is the evidence provided by Ms Lindsay that the concentration of Olanzapine found in the deceased's blood was at a level that had been suggested by some authors to "cause toxic

effects". Further Ms Lindsay noted that there have been "reported cases of death attributed to olanzapine at these levels".

16. Ms Lindsay further noted within her report however that a complication of interpreting such results was that "olanzapine may undergo post-mortem redistribution" and that a further complication was that "olanzapine may be subject to degradation *in vitro*". Ms Lindsay noted that these factors also needed to "be considered when comparing case results to literature data".

17. In relation to these issues of redistribution and degradation Ms Lindsay provided further information to the inquest by way of an email where she stated as follows:

> "…I can explain that post-mortem redistribution is basically used to describe the movement of drugs within the body after death, including diffusion from organs that have higher concentrations. So this affect could increase the level measured in the blood post-mortem relative to what would have been there at the time of death. The biggest affect from redistribution by diffusion will be in the more central blood closer to the organs. The blood in this case was said to be "iliac" blood, which is regarded as peripheral blood. Redistribution does not happen to all drugs to the same extent but as stated in the report, olanzapine may undergo post-mortem redistribution. It is not possible to say from our results on the blood received whether this has occurred.
>
> Another factor is degradation on storage, which it has been reported may occur for olanzapine. Once we receive the blood it is stored at -20°C except when being analysed. However, there are reports that olanzapine degradation can occur even at -20°C in preserved blood. If degradation had occurred it would have the effect of decreasing the concentration in the blood but we are not able to determine whether degradation of olanzapine has occurred between taking the sample and our analysis.
>
> I am unable to say whether either or both redistribution or degradation have occurred in this case.
>
> The other thing in the report to take note of is the blood to plasma ratio, which is reported to be 0.6 for olanzapine, as stated in both reports. As much of the ante-mortem literature data quoted has been

> measured in plasma, but our result is reported in blood, the ratio needs to be taken into account when comparing them."

18. In relation to these results, Dr Rutherford considered carefully his findings and undertook what he referred to as a "process of exclusion":

> "In other words this is a process of exclusion. If we don't find anything that explains death in the body the next phase is to go to the toxicology and the histology and in the toxicology we have this level of Olanzapine which was capable of causing death."

19. In his initial autopsy report Dr Rutherford expressed his opinion that taking into account the "absence of any other definable pathological explanation for demise, it would be reasonable to attribute death to olanzapine toxicity". Dr Rutherford did state however that he was prepared to revise this opinion if further information became available.

20. What occurred thereafter is a report was obtained from Professor Jason White, Professor of Pharmacology and Head of the School of Pharmacy and Medical Sciences at the University of South Australia. Professor White was asked to consider the concentration of olanzapine found in the deceased's blood samples and its relevance to cause of death. Professor White also noted "the possible changes in drug concentration that (occur) after death". However he noted that there was "now a considerable body of data relating to the post-mortem concentrations involving olanzapine and other antipsychotic drugs".

21. Professor White referred to systematic data from analysis of deaths occurring in Sweden since the 1990s and noted:

> "The results for olanzapine include 473 cases. The average concentration in cases where olanzapine alone was the cause of death was 0.55mg/L, while in cases where other drugs contributed to the death, the average concentration was 0.40mg/L".

22. Professor White noted that:

> "These results indicate that the concentration in the post-mortem blood sample from Mr Roberts is consistent with the concentrations found in cases of fatal overdose due to olanzapine. It is the same as

> the average concentration in cases where another drug or drugs were involved and a little below (but still within the range) for cases where olanzapine alone has been responsible for death".

23. In terms of possible explanations for the concentration of olanzapine found, Professor Whist opined that there were "several possible explanations". The first being that "the post-mortem concentration may not accurately reflect the concentration prior to death". This is consistent with the evidence of Ms Lindsay to be cautious when considering the toxicology results. The second possible explanation was that the deceased "took a larger dose than normal prior to death leading to a high concentration and toxic drug effects". The third possibility related to the "rate at which olanzapine is eliminated from the body" which was "influenced by genetic factors". Professor White stated that persons whose metabolism was low would "tend to achieve higher concentrations of olanzapine for a given dose than those whose level of enzyme activity is closer to the average".

24. As a result it was Professor White's opinion that:

> "… the concentration is higher than would be expected due to the dose prescribed, possibly due to a larger than normal dose or genetic factors".

25. I pause to note here that in terms of the third possibility of genetic factors, it was ultimately the evidence of Professor White that in the circumstances of this case and particularly the potential involvement of hyponatremia[1] (of which he was unaware of when he made that suggestion); genetic testing would not have assisted me in determining the issue of cause of death and his reference to genetic factors was in fact speculative.

26. At the same time that these reports were being obtained, the Primary Health Care Team ("PHCT") at the prison led by Dr Brian Cluney was also independently undertaking a review of the circumstances leading to this death. I received evidence from Dr Christine Connors that this review had taken place after the FMHT conducted its own review and referred the case

[1] See definition in paragraph 27 hereof.

to the PHCT for further investigation of matters relating to the death. As a result of that review a report was prepared and this was tendered into evidence before me.

27. That report raised in issue the potential for hyponatremia to have been relevant to cause of death. I received evidence that hyponatremia is a condition that occurs when the level of sodium in a person's blood is abnormally low. This is often caused by a person drinking too much water and therefore lowering the sodium level in their system. The report detailed the circumstances relating to the potential for hyponatremia to have been potentially relevant as follows:

> "27.1 Large quantities of "fluid" seen around the deceased when attended to by first responders;
>
> 27.2 The deceased observed to be having a "fit" prior to his collapse;
>
> 27.3 The deceased self-reporting to have been drinking large quantities of water in the weeks preceding and complaining that he was constantly thirsty and/or hungry."

28. As a result of this potential contributor, a copy of this review report was sent to Dr Rutherford and to Professor White. Upon receipt of that report, Dr Rutherford did (as he indicated he would) review his opinion and prepared a Supplementary Report. That report was also tendered into evidence and Dr Rutherford gave evidence addressing matters contained within that report.

29. Dr Rutherford noted that as a result of the issue of "water intoxication" having been raised; he had considered the results of his autopsy further. I pause to note that the term water intoxication is a short hand reference to hyponatremia. Dr Rutherford noted that he had sought the biochemistry results from the original vitreous humor submission. I heard evidence that this is a reference to the fluid around a person's eye. Dr Rutherford noted in his report that:

"Two of the parameters (sodium = 99mmol per litre and osmolality = 278 mOsm per kilogram) indicated haemodilution reflecting water overload.

The water overload may have been caused by excess ingestion of water by mouth.

Water overload has also been reported as a consequence of olanzapine therapy".

30. After considering these issues, including the complications of interpreting the olanzapine concentrations found in the post-mortem blood, issues associated with the high dose that the deceased was receiving and how that would have been metabolised by him and that he was in a state of water overload at the time of his death, Dr Rutherford relevantly concluded in his report as follows:

- "After reviewing the case and the relevant literature I am of the opinion that it is rather difficult to clearly separate the various components involved in this man's death. Water intoxication, olanzapine toxicity and the underlying condition requiring drug treatment (schizophrenia) are all likely to have played their roles. …

- Interpretation is compromised by the variability of toxic/fatal dose levels as recorded in the literature and the meaning of any single measured level given the propensity of the drug olanzapine to have unpredictable post-mortem diffusion characteristics over different body compartments.

- Given the foregoing, death is probably best recorded summarily as being due to **water intoxication and olanzapine toxicity in association with schizophrenia**".

31. In his oral evidence before me, Dr Rutherford stated as follows:

"Well, the additional material - much of which I was not aware of at the time of the autopsy, indicated - I am talking particularly of the Riskman Review, that water intoxication might be a cause or component of the cause of death, so I went back and sought some results which I hadn't received at the time of the original report which confirmed that he was, in fact, diluted, as it were. He had taken for some reason or other, excessive amounts of water on board and that his whole system was diluted - and we call that 'water intoxication'. So there's an element of water intoxication to this

death and the level was sufficient that it could have accounted for death on its own.

However, the issue becomes a little more complicated because not really that much is known about Olanzapine toxicity and the way in which it causes death. We know there are some deaths which are attributable to Olanzapine at therapeutic levels which are toxic effects on the heart but leaving that aside because there is no way that anyone can prove that, there have been cases reported where just taking Olanzapine actually causes the person to drink more water. So the drinking of the excess water may be directly related to the Olanzapine or it may be related to his primary psychiatric condition or the fact that he was in a prison environment, but we don't know which of those it is.

So it's difficult to separate out the threads of what is going on here but I am of the view that Olanzapine toxicity was a significant component. I am of the view that the water toxicity, now that I have reviewed the case, was a component but that component might have been related to the Olanzapine or it might not. So whichever way we look at it we still come back to Olanzapine toxicity and ….”

32. With respect to this issue of the potential involvement of hyponatremia and cause of death Professor White also gave evidence before me. In relation to his earlier report concerning Olanzapine toxicity and cause of death; Professor White agreed that olanzapine was known to cause an abnormal cardiac rhythm which can sometimes lead to death. Professor White stated that this potential could not be excluded by the fact that there was a pulse still able to be felt by medical staff very shortly after the deceased initially collapsed. Professor White stated:

> “No, it’s still possible there was an abnormality in the rhythm and it may have resulted in him collapsing. But the death didn’t occur immediately, there was some delay before death occurred.”

33. Professor White noted that when he provided his earlier report and opinion concerning the impact of Olanzapine upon the deceased’s death, he had not been aware of the potential for hyponatremia. Professor White confirmed that he had since considered the review report and the supplementary report provided by Dr Rutherford and with respect to the issues raised he stated as follows:

> "Well certainly the excess water consumption can be a cause of death, as was noted. And I would also add that there is a possible additive effect in the sense that excess water consumption can result in risk of seizures. There is also an increased risk of seizures associated with use of Olanzapine and with use of Zuclopenthixol. So there is some potential for, if you like, a combined effect of those medications taken by the deceased and the excess water consumption that may have occurred."

34. Professor White was asked if he thought it was possible to separate these various potential causes at all and stated:

> "No, it's not."

35. As part of the evidence relied upon by Territory Health Services, Dr Christine Connors (General Manager of Darwin Region and Strategic Primary Health Care) also provided a statement within which she too noted the report of the "fit" prior to the deceased collapsing and the difficulties with resuscitation because of the large amounts of clear fluid in his airway. Dr Connors noted these factors pointed to excessive water intake prior to death; further indicative of hyponatremia as the potential cause of death.

36. In her closing submissions to me, counsel for Territory Health Services Ms Williams urged me to find:

> "…that the primary cause of death was the acute water intoxication against a background of the other factors, because of the evidence of the significant amounts of fluid, the seizure that the deceased had, which is consistent with water toxicity and that perhaps the Olanzapine concentration levels may also be a red herring, because there is no precise explanation for why they occur in the post-mortem blood samples at that concentration."

37. With respect to this submission, I note that it is not in accordance with the opinion expressed by Dr Rutherford as to cause of death which was stated to be "best recorded as due to water intoxication and olanzapine toxicity in association with schizophrenia". I also note that Professor White agreed with this opinion.

38. Having considered the evidence very closely, I find that at the end of the day it remains highly speculative as to which scenario is the more likely than not based upon all of the evidence. However, in either scenario of Olanzapine toxicity or hyponatremia as the primary cause, it appears this death was unexpected and its cause rare in either case.

39. After careful consideration of the evidence relating to cause of death; I find the cause of death to be water intoxication and olanzapine toxicity in association with schizophrenia. I also find however that it is simply not possible to separate these potential causes from one another.

40. In my view, the deceased died suddenly with and from a rare and undiagnosed condition that was reasonably unforeseen.

Care, Supervision and Treatment

41. Section 26(1)(a) of the *Coroner's Act* requires that I must investigate and report on the care, supervision and treatment of the deceased while he was being held in custody.

42. The care, supervision and treatment of the deceased is also obviously important in this death given the issues relating to the concentration levels of olanzapine found in his post-mortem bloods and hyponatremia, but also the issues confronted by staff at the time they attempted to resuscitate the deceased. I will deal with each of these issues separately.

<u>The treatment of the deceased's psychiatric illness</u>

43. As earlier noted, the deceased had been diagnosed as suffering from schizophrenia for many, many years. He had also been prescribed olanzapine and zuclopenthixol for many years. I accept the expert evidence of Dr Kini that unfortunately the deceased suffered from treatment resistant schizophrenia and this made attempting to deal with his condition all the more difficult.

44. It was clearly apparent from the evidence however that attempting to deal with the deceased's illness and to improve his circumstances was an important matter to the FMHT and also the PHCT at the prison. Some criticism was attempted to be made by counsel on behalf of the family about the regularity (or otherwise) of the reviews conducted of the deceased during his incarceration. It was submitted on behalf of the family that the deceased "should have been monitored more frequently".

45. Whilst I empathise with the family in relation to the grief they feel at the passing of their loved one, I do not consider that there were inadequacies in the frequency of the monitoring of the deceased. I find that the deceased was appropriately monitored taking into account the relevant factors relating to his apparent drug compliance, his engagement with the FMHT, his lack of aggression or violence and his frequent indications of his willingness to continue receiving the medication that he was being provided.

46. I accept that the deceased did identify to the FMHT and the PHCT that he was hungry, thirsty and putting on weight in the weeks leading up to his death, and that he had a known history of consuming large quantities of water. I also accept that the weight gain and thirstiness are factors which would now be considered potential risk factors for Psychogenic Polydipsia, i.e. excessive water drinking in the absence of a physiological stimulus to drink. However these risk factors also need to be put into context in terms of the clear evidence that at the time of the deceased's death Psychogenic Polydipsia was not a well-known condition. In addition, it certainly was not a well-known condition within a prison setting nationally.

47. Further, there was some correlation of the "known" side effects of olanzapine compared to those now "known" to be relating to Psychogenic Polydipsia. Dr Kini gave evidence that "thirst" was not a side effect of olanzapine, but that "hunger" was. In terms of weight gain, Dr Kini gave evidence that this was a more common side effect of olanzapine rather than Psychogenic Polydipsia.

48. The deceased's electrolyte results on 4 February 2016 of 135mmol per litre were also highlighted on behalf of the family as a potential red flag that should have been more carefully considered. In relation to this issue I disagree. I find that this result was in fact carefully considered and I note that Dr Kini gave evidence that this level was "within normal range". In addition, Dr Connors gave evidence that such a level was "at the lower limit of normal" in terms of indicating a risk of hyponatremia.

49. I do not find that those involved in providing the deceased with medical care in prison were not providing appropriate care, supervision and treatment. In fact I find that the care, supervision and treatment was appropriate and I consider that as and when issues arose with respect to the deceased's illness, his circumstances were carefully considered by the medical team and decisions made in accordance with the circumstances as best they knew and understood at that time.

50. I acknowledge that the Top End Health Service conducted its own review and found aspects of the care provided to prisoners should be changed in light of the circumstances surrounding the deceased's death. However, that does not and should not mean that simply because there have been learnings from a death and improvements made that there has been a failure in the care, supervision and treatment provided. I commend the Top End Health Service for their proactive approach in reviewing this death prior to this inquest and putting in place changes in the health care services provided to prisoners. However I do not consider that the areas which were identified as requiring improvement were causative of the deceased's death.

Issues relating to difficulties resuscitating the deceased

51. As earlier noted, there were difficulties with resuscitation of the deceased because of the large amounts of clear fluid in his airway. I received evidence that despite several attempts there was never an open and clear airway able to be established due to the amount of fluid that the deceased appears to have consumed. This was despite the use of several different types of airways

and the use of a suction device to try and suction away the fluid and clear the airway.

52. In relation to the suction device I heard evidence that the manual device failed and as a result there was some delay in obtaining the automatic suction device as that was not then held in the equipment bags possessed by first responders. I received evidence that this has now changed as a direct result of the death of the deceased and that automatic suction devices are now part of the emergency response kit, rather than a manual suction device. Again, I do not consider this issue to have been causative of the deceased's death however it is an important change.

53. In relation to those who first arrived on scene after the deceased collapsed including the paramedic and ambulance staff that arrived shortly thereafter, I do not consider there can be any criticism made of the efforts they went to in an attempt to revive the deceased.

54. I note that ultimately no criticism was made of any action taken by Corrections with respect to the deceased and this was appropriate according to the evidence.

Formal Findings

55. Pursuant to section 34 of the *Coroner's Act*, I find as follows:

(i) The identity of the deceased was John Roberts born 19 December 1978, in Milikapiti, Northern Territory.

(ii) The time of death was 4.52pm, 20 February 2016. The place of death was Block 5E2, Darwin Correctional Precinct, Holtze, Northern Territory.

(iii) The cause of death was water intoxication and olanzapine toxicity in association with schizophrenia.

(iv) The particulars required to register the death:

1. The deceased was John Roberts.

2. The deceased was of Aboriginal descent.

3. The deceased was a prisoner and not employed at the time of his death.

4. The death was reported to the Coroner by the Darwin Correctional Precinct staff.

5. The cause of death was confirmed by Forensic Pathologist, Dr John Rutherford.

6. The deceased's mother is Stephanie Roberts and his father is Tracy Puruntatameri.

Recommendation

56. I have no recommendations to make arising from this inquest.

Dated this 13th day of October 2017.

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GREG CAVANAGH
TERRITORY CORONER