IN THE CORONERS COURT
OF VICTORIA
AT MELBOURNE

Court Reference: COR 2015 5713

FINDING INTO DEATH WITHOUT INQUEST

Form 38 Rule 60(2)
*Section 67 of the **Coroners Act 2008***

I, IAIN TRELOAR WEST, Deputy State Coroner having investigated the death of Tara Sue LOVE

without holding an inquest:

find that the identity of the deceased was Tara Sue LOVE

born on 15 July 1993

and the death occurred on 9 November 2015

at 5/28 Wheatsheaf Road Glenroy, 3046 Victoria

from:

1 (a) SUDDEN ARRHYTHMIC DEATH SYNDROME

Pursuant to section 67(1) of the **Coroners Act 2008**, I make findings with respect to **the following circumstances**:

1. Tara Love was a 22-year-old woman who resided in Glenroy with her partner, Mr Michael Fox.
2. Ms Love had a history of depression and anxiety. She also had a lengthy history of marijuana, amphetamine and stimulant drug use.
3. On 17 November 2014, Ms Love attended the Summit Medical Group where it was ascertained she was pregnant. On 8 January 2015 at 18 weeks pregnant, she was transferred to the Royal Women's Hospital (RWH) by Ambulance Victoria (AV) after feeling light headed. On admission to the emergency department (ED), Ms Love stated that she had protracted nausea and vomiting which had increased in the preceding week. She also stated that in the previous week she had ceased all alcohol, ICE and cannabis usage. An electrocardiogram (ECG) was performed and she was admitted in the setting of acute alcohol withdrawal, complicated by hypokalaemia and hypochloraemia. She received rehydration and electrolyte replacement.
4. On the 9 January 2015, Anaesthetist Dr Cave was asked to see Ms Love regarding her hypokalaemia and ECG abnormalities. He documented in the progress notes at 1am the ST depression, flattened T wave on the ECG. His impression was "*hypokalaemia and hypochloraemia secondary to vomiting with ECG change.*" He again reviewed her at 4.45am and stated in the notes, "*ECG improvement.*" Due to this improvement and her hypokalaemia being treated, a decision was made to admit Ms Love to the ward rather than to the Complex Care Unit (CCU).

5. On 12 January 2015, Ms Love was discharged with no further withdrawal symptoms. She was tolerating good oral intake and was linked to the Women's Alcohol and Drug Service (WADS) clinic with an appointment for 16 January 2015.
6. On 6 April 2015 at 31 weeks, Ms Love was admitted to the RWH due to nausea and vomiting. She was treated for hypokalaemia, hypomagnesia and mild liver function (LFT) derangement. ECGs were obtained on 6 and 7 April 2015 and abnormalities were documented.
7. An abdominal ultrasound on 10 April 2015 was performed which concluded that Ms Love's liver was of normal size and contour, with no lesions. The pancreas was normal and there was no evidence of cholelithiasis. Observations indicated Ms Love's blood pressure was slightly elevated 151/88 mmHg. She was treated for dehydration with intravenous fluids and electrolyte replacement. On assessment by a resident medical officer, Ms Love's blood pressure was 120/60 mmHg. She was subsequently discharged.
8. On 7 May 2015, Ms Love attended the ED of the RWH with oedema to her legs, hands and face. Bloods were obtained and were normal with unchanged LFTs. Ms Love's blood pressure was normal and she was discharged with a follow up scan planned for the following day.
9. On 8 May 2015 at 36 weeks, Ms Love's ultrasound indicated that her foetus had a breech presentation. A scan on 22 May 2015 indicated the foetus was no longer breech but her placenta was 20% calcified. A discussion occurred between the reviewing Hospital Medical Officer (HMO) and Obstetrician Dr Rebecca Szabo, to determine if Ms Love's baby could be delivered at term. Ms Love was to be reviewed weekly and was planned for induction of labour. Her blood pressure at the consultation was noted as 125/75 mmHg.
10. On 29 May 2015, Ms Love was seen in the Pregnancy Day Care Centre (PDCC), and whilst at PDCC a deceleration was seen on the cardiotocographic machine with slow recovery. Given Ms Love was 39 weeks pregnant and her placenta was calcified, a decision was made for an induction of labour and she was transferred to the birth suite for an emergency caesarean section.

Post-partum to November 2015:

11. Ms Love gave birth to her daughter at 4.40am on 30 May 2015.
12. In post-acute care at 5am, Ms Love was noted to be tachycardic and hypertensive. An ECG was performed, and a 500mm fluid bolus of Hartmann's was intravenously administered. At 8am, she was asymptomatic but had ongoing elevated blood pressure readings. She was reviewed by the HMO who then discussed her ongoing elevated blood pressure with the Senior Registrar. Labetalol medication was commenced. Her blood pressure remained stable with continuing medication and she was planned for discharge. WADS reviewed Ms Love pre-discharge and documented in the progress notes "*For home visit Thursday. Healthy mums, healthy babies, midwife aware.*"
13. On 2 June 2015, Ms Love was discharged. Medical notes indicate that she should continue with her Labetalol medication and be checked by her General Practitioner (GP) in a week. Further that the WADS midwife check her blood pressure during the home visit. On the RWH discharge letter, antenatal complications were documented as oligohydramnios and hypokalaemia. No postpartum complications were noted. There was no documentation however of Ms Love's perinatal ECG changes, her hypertension or prescription of Labetalol.

14. Ms Love was reviewed on 4 June 2015 by a Post Care in the Home (PCITH) midwife. Her blood pressure was documented as 125/85 mmHg and she was continuing with the Labetalol medication. The midwife discussed that she was to attend the GP in one week for blood pressure check and a six week follow up appointment as a routine post-partum check.

15. Ms Love was again reviewed by PCITH on 6 June 2015. Although she was feeling physically well, she stated that she was feeling emotional and that breast feeding was difficult. A weight drop was noted in the baby's weight and education and information on how to increase breast feeds and signs to encourage the baby to feed were provided. No blood pressure was documented and there was also no documentation regarding Labetalol.

16. On 8 June 2015, PCITH reviewed Ms Love again. Due to a further drop in the baby's weight, Ms Love and the baby were re-admitted to the RWH. She was subsequently discharged on 10 June 2015 with the Department of Human Services to conduct an assessment at home. A paediatric and psychiatric review appointment was booked for 26 June 2015 but Ms Love did not attend. On 24 July 2015, Ms Love attended the RWH for a post-natal check but no observations were obtained and there was no documentation related either to her cardiac history or Labetalol.

17. On 8 November 2015, Ms Love felt unwell and developed diarrhoea and vomiting. She considered this may have been due to food poisoning as she had been out for lunch that day. At 11pm, Ms Love and Mr Fox went to bed. At 1am on 9 November 2015, Mr Fox turned over and realised that Ms Love was not breathing. He contacted emergency services. Paramedics arrived and attempted resuscitation for 30 minutes but Ms Love could not be resuscitated.

18. Senior Forensic Pathologist Dr Malcolm Dodd from the Victorian Institute of Forensic Medicine performed an autopsy on Ms Love and provided a written report of his findings. He stated that the immediate cause of death appeared to be of cardiac arrest in the setting of documented QT prolongation. QT prolongation can occur in cardiac arrhythmic syndromes such as Long QT Syndrome (LQTS). Acquired QT prolongation cannot be entirely excluded as Ms Love had long term hypokalaemia which was first detected during her pregnancy.

19. Dr Dodd requested an expert cardiologist opinion on Ms Love's ECGs for his report. This was done by Associate Professor (A/P) Neil Strathmore who considered Ms Love's ECG's from 8 January 2015, four ECG's from 9 January 2015, four ECGs from 6 and 7 April 2015, and one undated ECG. A/P Strathmore was of the opinion that the '*wave patterns were diagnostic of LQTS.*' LQTS is a heritable electrophysiological disorder which may lead to arrhythmia and sudden death. The apparent acute illness (of unknown aetiology) may have exacerbated the syndrome, leading to lethal arrhythmia.

20. Biochemical analysis disclosed elevated sodium and chloride levels which Dr Dodd stated may be explained by mild dehydration. Renal function tests were within normal parameters. On the basis of the possibility of food poisoning and infection, various biological samples were submitted for testing. The blood culture returned a mixed growth of *Streptococcus pyogenes* (Group A) and also *Streptococcus salivarius/vestibularis* and *Gemella morbillorum.* The combination of bacteria isolated almost certainly indicates contamination. A similar mixed result was returned from culture of heart tissue (including beta haemolytic Streptococcus A+). Dr Dodd stated that the significance of the Group A Streptococcus was unclear. There were no obvious macroscopic or histological septic foci. He concluded that

there appeared to be no bacteriological/virological aetiology for the sudden death in this instance.

21. Toxicological analysis revealed the presence of delta-9-tetrahydrocannabinol (cannabis). No alcohol or any other common drugs or poisons were present. Dr Dodd concluded that there was no evidence to suggest that Ms Love's death was due to anything other than natural causes.

Family Concerns:

22. On 24 March 2016, the Court received a letter of concern from Mr Fox querying the following;

    1. What follow up care was provided to Ms Love regarding her heart condition?
    2. Why was there no referral for her heart condition made after the baby's birth?

2. As a result of these concerns, I referred the matter to the Coroners Court Health and Medical Investigation Team (HMIT)[1] for review. They reviewed all available materials and also requested further statements from;

    1. Associate Professor Mark Peter Umstad, Clinical Director of Maternity Services at the RWH
    2. Ms Karen Cusack, Corporate Counsel for the RWH

    The HMIT subsequently provided a written report of their findings.

3. With respect to the first issue raised by Mr Fox, Ms Love had ECGs performed on 8 and 9 January 2015 whilst in the Emergency Department of the RWH. These were due to her episodes of hypokalaemia which were attributed to her hyperemesis. Her ECGs were reviewed by Anaesthetist Dr Cave and a discussion was held as to whether she should be admitted to the CCU. Ms Love was treated with appropriate medication at this time and changes to her ECG were identified on review; with the primary consideration and cause attributed to her electrolyte levels. There is no documented concern in relation to LQTS and no referral was made.

4. Ms Love had two further ECGs obtained during her April 2015 admission in the CCU. The ECGs were reviewed and written on by two medical staff.[2] Dr Huang noted ECG changes, however there is no documentation in the progress notes regarding any concerns and there was no escalation for further review or investigation.

5. Statement questions were sent by the HMIT to the RWH regarding Ms Love's ECG changes and who is tasked to review ECG's. A/P Umstad stated that the policy for ECGs recorded in the Women's Emergency Centre is as follows;

    '*ECGs are to be read by an Australian College of Emergency Medicine trainee or Fellowship of the Australian College of Emergency Medicine. If one is not*

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[1] The role of the Health and Medical Investigation Team (HMIT) is to assist the Coroner's investigation into the nature and extent of deaths which occurred during the provision of healthcare, and identify potential system factors in healthcare related deaths. HMIT personnel comprise of practising Physicians and Clinical Research Nurses who draw on their medical, nursing and research experiences, skills and knowledge to independently evaluate clinical evidence for the investigation of reportable healthcare deaths and to assist in identifying remediable factors that may assist in prevention and risk management in health services settings.

[2] Three ECGs were obtained between 6 and 7 April 2015. One was signed by Dr Huang. No signature is on the second (although the handwriting is similar to the RMOs in the progress notes) and the third has been reviewed but is unsigned.

immediately available, they are to be placed in the ECG drawer and these are to be checked regularly by emergency doctors. Any immediate concerns regarding the interpretation of an ECG when an emergency doctor is not present should be telephoned and then faxed for a discussion with the Royal Melbourne Emergency Physicians.'

6. The process of review in the Complex Care Unit is different. A/P Umstad stated;

> '*[...] Medical staff review the 12 lead ECGs which are recorded on the patient's admission to CCU and then as required. [...] As the Anaesthetic Department are closely involved in every patient admitted to CCU, it is the Anaesthetic registrar or Anaesthetic consultant who review the patients on a daily basis and review the 12 lead ECGs [...] If there is a concern about the interpretation of the ECG it will be escalated to RMH Cardiology Department for their interpretation.*'

7. The HMIT asked the RWH to provide the Royal Women's Hospital ECG policy, which was current at the time when Ms Love's ECGs were obtained. The RWH's response in a letter dated 8 March 2017 stated;

> '*We advise that the Women's is unable to provide you with ECG policy (or policies) as the Women's does not have a specific ECG policy (or policies).*'

8. Ms Love also had a further two ECGs obtained prior to her caesarean section in May 2015. A/P Umstad stated:

> '*The Director of our Women's Emergency Centre had advised me that hypokalaemia can be a cause of prolonged QT interval and has advised me that they generally require a QT >480 msec before its considered clinically significant. I note that subsequent ECGs, after the admission for hypokalaemia showed a QT of 480 msec on 6 April 2015 and 464 msec on 30 May 2015. It would appear that, since the subsequent QT lengths were not more than 480 msec, that no further evaluation was undertaken.*'

Thus, there was no follow up for Ms Love's heart condition as it was considered to be related to her hypokalaemia, which had resolved.

9. With respect to the second concern in Mr Fox's letter, Ms Love was discharged on 2 June 2015. There was no documentation on the discharge summary in relation to her hypertension or ECG concerns. She was prescribed Labetalol and told to see her GP for a check-up in one week. WADS was to check her blood pressure during the home visit.

10. Ms Love was reviewed by WADS on 4, 6 and 8 June 2015. Her blood pressure was checked on 4 June but no further documentation is apparent in relation to this on subsequent visits. Ms Love also failed to attend her GP for a check-up despite being told to do so both by the RWH and WADS. Notes from the Summit Health Care Group show that her last attendance was on 20 February 2015. It is not clear when Ms Love ceased her Labetalol medication in the postnatal period.

11. On 8-10 June, Ms Love was readmitted to the RWH. However, the HMIT could not locate any documentation in relation to her admission and sought clarification from the RWH. A statement provided by Ms Cusack indicated this to be correct. She stated;

> '*There does not appear to be any medical notes from the admission and no discharge letter held by the Women's. However, the postnatal observations*

chart notes that blood pressure was recorded on 9 June 2015 at 05.00 hrs. This appears to be the only record noted during the admission.'

12. Ms Love subsequently attended an appointment at RWH on 24 July 2015 for her six week check-up. Neither her peripartum hypertension nor medication with Labetalol is documented as being discussed.

HMIT ECG Review and Assessment:

LQTS and pregnancy:

13. LQTS is a disorder of ventricular myocardial repolarization characterized by a prolonged QT interval on the ECG that can lead to symptomatic ventricular arrhythmias and can lead to an increased risk of sudden death. It may be either genetic or acquired.

14. Acquired LQTS can result from many causes or a variety of medications as well as hypokalaemia or hypomagnesia. As the QT interval varies inversely with heart rate, therefore the QT measurement is adjusted for the heart rate, resulting in the correct QT interval or QTc. In adult females the normal range is 0.37 to 0.47 seconds. The position statement for clinical practice on familial Long QT syndrome by the Cardiac Society of Australia and New Zealand was updated in 2016. It noted the "prevalence is approximately 1 in 2,500." It identified the "*ECG typically reveals a heart rate corrected QT interval repeatedly greater than 470ms in women*" also; '*High risk individuals are identified by QTc>500ms and/or syncope in previous two years. Boys and post pubertal females (especially in the nine post-partum months) are high risk.*'

15. LQTS seen in pregnancy may serve to shorten the QT interval and therefore be partially protective.[3] The risk of VT is especially high in the post-partum period;[4] '*Increased risk of VT during the postpartum period may be related to a decrease in the heart rate and an associated increase in the QT interval. The physiologic stress and altered sleep patterns associated with caring for a newborn infant may also contribute to an increase in adrenergically mediated cardiac events.*'[5]

16. Ms Love's ECGs were reviewed by a Consultant Emergency Department Physician and a Consultant Intensivist within the HMIT. They provided the following commentary:

> '*A prolonged QT predisposes the patient to ventricular arrhythmias and sudden death. There are many causes of a long QT including genetic conditions.*
>
> *As well as the long QT interval the ECGs also show abnormalities of the ST segments. The changes are dynamic (they change) but particularly on the ECG dated 6 April 2015. There are marked biphasic T waves in V3 and widespread T wave inversion in leads II, III, aVF, V4, V5 and V6. These changes had been noted on one of the 6 April 2015, ECG. The changes are present on the ECGs in January but not as marked.*
>
> *In my opinion these changes on an ECG in a 22 year old female would certainly warrant further investigation with at least an echocardiogram and*

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[3] Louise Harris et al, Ventricular arrhythmias during pregnancy, Accessed 8 March 2017.

[4] Ibid.

[5] Ibid.

referral to a cardiologist. Also it is presumptuous to assume the prolonged QT is due to hypokalaemia as there are many other causes.'

17. A/P Strathmore's review of Ms Love's ECG's noted the following;

    'The abnormality in all the ECGs is prolongation of the QT interval. The QT interval is corrected for the heart rate and the corrected QT (QTc) interval ranges between 480 and 534ms, all of which are prolonged. In addition, there is ST and T wave change in the anterior and inferior leads. This is most marked in the ECGs from 6 and 7 April but is also present in the ECGs of January 2015.

    Only one of the ECGs is reported with a signature and that does not mention the QT prolongation, though that is the ECG with the shortest QTc which is 480ms. These ECGs indicate that Ms Love had long QT syndrome.'

18. The HMIT stated that in addition to Ms Love's ECG changes, she suffered from episodes of pre-syncope. In light of this, the HMIT concluded that further review of Ms Love's ECGs was warranted.

19. I am satisfied the ECGs should have been referred to the Royal Melbourne Hospital's Cardiology Department with a cardiology review sought and further cardiology investigations performed.

20. I find that the cause of death of Tara Love was sudden arrhythmic death syndrome.

RECOMMENDATIONS:

21. Pursuant to section 72(2) of the *Coroners Act 2008* (Vic), I make the following recommendations in relations to the death. I am aided by the HMIT in respect of these matters.

    1. That the RWH improve its documentation follow up and discharge documentation. This is due to the fact that there was no medical documentation or discharge information for Ms Love's presentation on 8 June 2015.
    2. That the RWH clearly document non-obstetric medical issues on discharge documentation. Further, that non-obstetric medical issues have a clear follow up discharge plan.
    3. That the RWH develop and implement a hospital wide ECG policy. This should include a specific section related to ECG review and reporting as well as the following;
        i. All ECGs be reviewed and signed, including date and time of review by a medical officer.
        ii. In the Women's Emergency Centre, ECGs are the responsibility of the Doctor managing the patient at that time.
        iii. If a patient is admitted into the RWH, the ECGs are the responsibility of the treating team.
        iv. All ECGs are to be formally reported by a cardiology registrar or above.
        v. Review of the formal cardiology report is the responsibility of either the Doctor or the medical team (as noted in points i and ii)

vi. Referral for further investigations for any detected ECG abnormalities.

4. That the RWH widely promote and distribute the ECG policy and change in practice to all staff members.

I direct that a copy of this finding be provided to the following:

Mr Michael Fox

Ms Karen Cusack, Corporate Counsel, The Royal Women's Hospital

The Consultative Council on Obstetric and Paediatric Mortality and Morbidity

Signature:

IAIN WEST
DEPUTY STATE CORONER
Date: **28 August 2017**