Record of Investigation into Death (Without Inquest)

Coroners Act 1995  
Coroners Rules 2006  
Rule 11

I, Rod Chandler, Coroner, having investigated the death of Kenneth Charles Richardson

Find that:

(a) The identity of the deceased is Kenneth Charles Richardson;

(b) Mr Richardson was born in Sorell on 6 September 1921 and was aged 93 years;

(c) Mr Richardson died at the Royal Hobart Hospital (RHH) in Hobart on 23 September 2014;

(d) The cause of Mr Richardson’s death was digoxin toxicity complicating acute on chronic renal failure. Significant contributing factors were valvular heart disease (mitral regurgitation) and atrial fibrillation with congestive cardiac failure and advanced chronic renal disease.

Background

Mr Richardson resided with his wife, Barbara, at 115 Marys Hope Road, Rosetta. He was retired. His medical history included osteoarthritis, mitral incompetence, atrial fibrillation, ischaemic heart disease, congestive cardiac failure, gout, gastro-oesophageal reflux disease and hypertension.

In April 2010 Mr Richardson was admitted to the RHH for worsening of his congestive cardiac failure. At this time he was taking a 0.0625 mg dose of digoxin each day for treatment of his systolic cardiac failure. The blood level for digoxin was recorded at 1.13 nmol/L. Whilst in hospital this daily dose was increased to 0.125 mg. A subsequent blood test done in December 2010 showed the digoxin level to be 1.8 nmol/L which was within the normal range of 1.0-2.6 nmol/L.

Circumstances Surrounding the Death

In the evening of 19 September 2014, Mr Richardson had a fall at home whilst getting ready for bed. It was witnessed by his wife. An ambulance was called and Mr Richardson was taken to the RHH. In the Emergency Department Mr Richardson was noted to be alert and pain free. His pulse was 30 bpm and his blood pressure was 100/60. Blood tests showed multiple irregularities suggestive of acute on chronic renal impairment and digoxin toxicity. The
digoxin level was recorded at 3.68 nmol/L, well outside the normal range. Treatments initiated included the administration of 'Digibind', an antidote for digoxin.

The following morning Mr Richardson was reviewed by the admitting team. Provisional diagnoses were made of digoxin toxicity and bradycardia (slowing of the heart rate). It was noted that family members reported that Mr Richardson had been unwell the previous few days and had had a loss of appetite over the past year. He was admitted to a ward.

Over the following days Mr Richardson rested in bed and had no complaints of pain or shortness of breath. However, in the evening of 22 September his oxygen saturations dropped and he became agitated. Thereafter, he continued to deteriorate. He was declared deceased at 6.00pm on 23 September 2014.

Post-Mortem Examination

This was carried out by forensic pathologist, Dr Donald Ritchey. In his opinion the cause of Mr Richardson’s death was digoxin toxicity complicating acute on chronic renal failure. Significant contributing factors were valvular heart disease (mitral regurgitation) and atrial fibrillation with congestive cardiac failure and advanced chronic renal disease.

In his report Dr Ritchey includes this helpful information:

“Digoxin and warfarin are two commonly prescribed medications that each has a narrow therapeutic index (IT). That is, the blood concentration needed to be efficacious overlaps with the concentration at which toxicity can develop. It is a challenge to carefully titrate the doses of these medications in people whose clinical condition changes with time. Because these medicines are partly cleared from the body by the kidneys, renal failure may cause increased blood concentrations of these drugs even though no increased dose is given. When chronic (long standing) kidney failure is complicated by acute failure, the blood concentration of many drugs, especially digoxin, increases rapidly causing unintended toxicity.”

I accept Dr Ritchey’s opinion upon the cause of death.

Investigation

The investigation has been informed by:

- A review of Mr Richardson’s records at the RHH undertaken by Clinical Nurse, Ms L K Newman.
- A report provided by Dr Dimitrios Klonaris, the general practitioner for Mr Richardson.
• A review of therapeutic drug monitoring of patients taking digoxin undertaken by Dr Ritchey.

• A report upon Mr Richardson’s medical care provided by Dr A J Bell as medical adviser to the coroner.

In his report Dr Klonaris advises that:

1. Mr Richardson was not tested for the level of digoxin after 10 December 2010 when the level was 1.8 nmol/L (as I’ve recorded above).

2. No changes were made to Mr Richardson’s digoxin dosage in the two years prior to his death.

Dr Bell, in his helpful report, advises me that:

• Mr Richardson required digoxin to manage his systolic heart failure. The dosage being taken in April 2010 was appropriate. Because of his atrial fibrillation it was also necessary for him to take warfarin as it reduced the risk of stroke.

• The usual clinical course for elderly patients is a slow and steady deterioration of renal function. On 30 April 2010 Mr Richardson’s creatinine and urea levels were respectively recorded at 106 nmol/L and 8.7 mmol/L. Testing over the following years showed a gradual increase in both levels confirming a decline in his renal function. When hospitalised on 20 September 2014 the creatinine level was 231 nmol/L and the urea level was 41.5 mmol/L. These readings suggest severely impaired renal function and hence a reduced capacity for the kidneys to clear the body of digoxin.

• Monitoring of the digoxin concentration is particularly important when digoxin is used for the treatment of heart failure with systolic dysfunction. Mr Richardson was in this category. Appropriate monitoring requires the serum digoxin level to be measured 7 to 10 days after commencing digoxin or changing the dose and repeat testing should occur every 3 to 6 months thereafter, or upon a change in the patient’s clinical status.

• In Mr Richardson’s circumstances, it is likely that blood testing would have shown a progressive elevation in his digoxin level requiring a commensurate reduction in his dosage to avoid toxicity.

• Mr Richardson’s presentation to the RHH on 19 September 2014 was typical of digoxin poisoning.

• Mr Richardson’s management in the RHH was appropriate.
Findings, Comments and Recommendations

It is clear that Mr Richardson was seriously ill when he presented at the RHH on 19 September 2014 with a failing heart and digoxin toxicity. The latter condition was appropriately and successfully treated with an antidote. However, Mr Richardson was unable to survive the effects of his underlying heart conditions and sadly died.

The evidence shows that Mr Richardson’s digoxin toxicity was a consequence of him taking digoxin in a dosage which exceeded the capacity of his impaired kidneys to process. I am satisfied that this situation evolved because of a failure to monitor Mr Richardson’s digoxin levels on a regular basis and in particular during the two years prior to his death. This leads me to adopt the suggestion of Dr Bell and recommend that for elderly patients taking digoxin for systolic heart failure, their treating general practitioners ensure that the levels of digoxin are monitored by repeat blood testing undertaken every 3 to 6 months.

I have decided not to hold a public inquest into this death because my investigation has sufficiently disclosed the identity of the deceased, the date, place, cause of death, relevant circumstances concerning how his death occurred, and the particulars needed to register his death under the Births, Deaths and Marriages Registration Act 1999. I do not consider that the holding of a public inquest would elicit any significant information further to that disclosed by the investigation conducted by me.

I convey my sincere condolences to Mr Richardson’s family and loved ones.

Dated: 10 November 2016 at Hobart in the State of Tasmania.

Rod Chandler
Coroner