QUEENSLAND COURTS

OFFICE OF THE STATE CORONER

NON-INQUEST FINDINGS

CITATION:	Investigation into the death of Michael John HAMILTON
TITLE OF COURT:	Coroners Court
JURISDICTION:	SOUTHPORT
FILE NO(s):	2013/4504
FINDINGS OF:	James McDougall Coroner
CATCHWORDS:	Abdominal pain, hospital admission and diagnosis, surgical management, post-operative care.

REPRESENTATION:

Counsel Assisting:	Ms Rhiannon Helsen, Office of the State Coroner

Michael Hamilton was 72 years of age at the time of his death. He had a notable medical history, which included a coronary artery bypass, the insertion of an artificial mitral valve, cholecystectomy, inguinal hernia repairs, lithotripsies, small abdominal aortic aneurysm, dyslipidaemia and benign prostatic hypertrophy.

At 7:59 pm on 4 September 2013, Mr Hamilton presented to the Logan Community Hospital ('LCH') after suffering from abdominal pain, nausea and vomiting. A week prior to his presentation, Mr Hamilton had undergone a resection of a bladder tumour and was taking the anticoagulant, Warfarin to prevent a blood clot following surgery.

Following an initial assessment conducted shortly after admission, Mr Hamilton underwent an erect chest x-ray at 9:26 pm, which showed that there was no free air under the diaphragm. However, it was thought that his presentation was suspicious for a duodenal perforation or gastric ulcer. A CT scan of Mr Hamilton's abdomen showed extraluminal free gas in the area of the duodenum. It was the opinion of the reporting Radiologist that this finding suggested perforation of the duodenum.

Mr Hamilton's case was discussed with Surgeon, Dr Mahi Ranasinghe, following which it was decided that a laparotomy was necessary. This procedure was subsequently carried out in the early hours of the morning on 5 September 2013. During the laparotomy, no duodenal perforation was found. There was, however, a dense adhesion of the anterior abdominal wall. After adhesiolysis, the site was said to be 'very oozy' and was managed with diathermy. The pancreas was also said to feel 'slightly hard' and the possibility of pancreatitis (chronic inflammation of the pancreas) was queried. An elevated serum lipase level (an enzyme elevated in pancreatitis) of 1080 U/L was measured. Unfortunately, whilst a lipase test was requested pre-operatively, it was not carried out as there was insufficient blood in the sample taken to conduct the test.

At 5:00 pm on 5 September 2013, Mr Hamilton was transferred to a general ward from the Intensive Care Unit ('ICU'). A Heparin infusion was continuing at this time.

On 6 September 2013, a MET Call was made by medical staff when Mr Hamilton began to experience an increase in respiratory rate and pulse rate, decrease in urine output and increasing abdominal distention. At 9:30 pm, he was transferred to the ICU. Shortly thereafter a CT scan was conducted. A large intra-peritoneal haematoma was detected, which required surgical treatment. Two litres of blood was found in the abdomen, which was managed with a VAC dressing (special dressing for an open abdominal wound where repeat abdominal entries are necessary).

In total, Mr Hamilton underwent six surgical procedures in 16 days for abdominal washout, sometimes with examination of the abdominal contents, and change of the VAC dressing, until closure of the surgical wound on 22 September 2013.

Unfortunately, Mr Hamilton suffered from a number of further complications, including the following:

- Renal failure, which required dialysis for 27 days;
- Type 1 respiratory failure necessitating ventilation;
- Coagulopathy and blood loss requiring a significant amount of blood and blood products;
- Low blood pressure;
- Abnormal liver chemistry;
- Cessation of bowel motility and function;
- Abnormal and dysfunctional heart rhythm;
- Recurrent infection in different sites with different organisms;
- Neutropenia;
- Erosive gastritis with blood in the naso-gastric aspirates;
- Possible neurological impairment;
- Incarcerated inguinal hernia; and
- Recurrent hypernatremia (elevated blood sodium).

For 98 days, Mr Hamilton consistently required respiratory support. At best, he was weaned off the ventilator on 30 October, however, required pressure support overnight.

On 13 October 2013, Mr Hamilton suffered another episode of sepsis. During this time, he also had a recurrent pseudomonas' infection, which was resistant to a large number of antibiotics. Mr Hamilton's poor prognosis was discussed with his family, and it was decided that further treatment should be palliative only.

Mr Hamilton died on 13 December 2013 at 9:30 pm.

Autopsy

On 23 December 2013, Forensic Pathologist, Dr Dianne Little conducted an external only post-mortem examination. A post-mortem CT scan and further toxicological testing were also undertaken.

Relevantly, the post-mortem CT scan showed the following:

- A large area of haemorrhage surrounded by marked oedema, which was thought to be suggestive of a haemorrhagic infarct in the right frontal lobe.

- Diffuse consolidation of the right lung.

- Patchy consolidation of the left lung.

- Extensive calcification of the coronary arteries and aorta.

- Fluid level in the large bowel.

- Hypodense areas around the spleen and liver, which would be consistent with haemorrhage.

Evidence of recent medical intervention was also observed, including multiple healed scars on the abdominal wall, a tracheostomy stoma in the root of the neck, and previous heart surgery. A pressure sore was found on the left side of the hip.

Having considered Mr Hamilton's medical records and the findings at autopsy, Dr Little concluded that Mr Hamilton died as a result of multiple organ failure, due to or as a consequence of, sepsis, due to or as a consequence of abdominal adhesions, which were due to or as a consequence of, previous surgery. Other significant conditions noted were coronary atherosclerosis and mitral valve disease.

Clinical Forensic Medicine Unit Review

At my request, Forensic Medical Officer, Dr Nelle van Buuren subsequently conducted a review of the circumstances surrounding Mr Hamilton's death. On 19 December 2013, I received her report, which confirmed that Mr Hamilton's death was reportable. Dr van Buuren also suggested that the opinion of a Surgeon be sought if the surgical management of Mr Hamilton at the LCH required further review.

Expert report by Dr Geoffrey Miller, Specialist Surgeon

An expert report was subsequently sought from Dr Geoffrey Miller, Specialist Surgeon. Dr Miller was asked to review the matter, and comment upon the care and treatment provided to Mr Hamilton at the LCH following his admission on 4 September 2013.

The specific issues Dr Miller was asked to address, and his answers, are as follows:

(a) *Was intervention necessary and appropriate when Mr Hamilton presented to the LCH on 4 September 2013?*

Dr Miller is of the view that the surgical procedure Mr Hamilton underwent on 5 September 2013, as performed by Dr Ranasinghe, was appropriate. Considering the findings of the radiologist, which suggested that Mr Hamilton was suffering from an acute perforation of the duodenum, such a serious condition, if left untreated, could have a higher morbidity and mortality. He is of the view that the indication for a laparotomy was compelling.

(b) *Your opinion as to the appropriateness of the treatment provided to Mr Hamilton by the treating team at the LCH.*

(i) Admission and diagnosis:

Dr Miller is of the view that Mr Hamilton's abdominal pain was as a result of pancreatitis. He notes that there were features of his admission, which pointed to a possible diagnosis of pancreatitis. In retrospect, Dr Miller opines that the CT scan of the abdomen was consistent with a diagnosis of pancreatitis, in that the free fluid surrounding the second and third parts of the duodenum and thickening of the duodenal wall was consistent with this condition.

Dr Miller notes that a lipase was not estimated until after his laparotomy. He is of the view that the elevated lipase that was detected after his operation was not due to palpation of the pancreas at the time of surgery.

(ii) Operative Findings:

Dr Miller notes that the surgical team found no evidence of a perforated hollow viscus. It was noted that the head of the pancreas felt slightly hard. A drain was inserted and Mr Hamilton was transferred to the ICU. Dr Miller is of the view that the care provided was appropriate.

(iii) Immediate post-operative management:

Dr Miller is of the view that Mr Hamilton should not have been discharged from the ICU to the ward on the first day post-operatively. A number of tests were conducted when he first arrived at the ICU, including lipase, calcium and a blood gas. In Dr Miller's opinion, these findings suggested a diagnosis of pancreatitis, with potential for possible further complications and mortality.

Given the results of the tests conducted, the increased bleeding experienced during the procedure, his medical history and the possible diagnosis of pancreatitis, Dr Miller is of the view that Mr Hamilton should not have been discharged from the ICU.

(iv) Post-operative anticoagulation:

Dr Miller is of the view that given the increased bleeding Mr Hamilton experienced during his procedure, which necessitated the insertion of a drain, the Heparin infusion should not have been recommenced the following morning. In his opinion, the issues associated with thrombosis as a result of Mr Hamilton's mechanical mitral valve were not as significant as those associated with the immediate post-operative issues.

(v) Management in the ward:

It is Dr Miller's opinion that given Mr Hamilton suffered from ongoing abdominal distention resulting in a MET Call on 6 September, he should have been immediately transferred to the ICU.

(vi) Further surgical intervention and intensive care management:

Dr Miller is of the view that the subsequent surgical procedures and

intensive care management provided to Mr Hamilton was appropriate.

(c)Any other issues you may wish to comment upon regarding the care provided to Mr Hamilton.

Dr Miller is of the view that it is likely Mr Hamilton presented to the LCH on 4 September 2013 suffering from pancreatitis. However, this condition was not diagnosed as a lipase was not taken. A subsequent error in interpreting the abdominal CT scan resulted in Mr Hamilton undergoing a laparotomy. However, Dr Miller does not believe that the laparotomy was the cause of Mr Hamilton's demise.

Dr Miller is of the opinion that Mr Hamilton's early discharge from the ICU, in the presence of red flags, which indicated a significant pancreatitis, was not prudent. He believes it is likely that the Heparin infusion subsequently conducted, likely exacerbated Mr Hamilton's intra-abdominal bleeding, thus causing abdominal distension. Dr Miller is of the view that had Mr Hamilton remained in the ICU, his deterioration would likely have been detected earlier, and corrective measures instituted earlier.

Dr Miller concludes that Mr Hamilton's initial management was not ideal. However, after the problems associated with this initial management settled, he underwent multi-organ failure. Dr Miller opines that had Mr Hamilton's management been ideal, he would likely have suffered a similar outcome, secondary to an attack of severe pancreatitis.

Response by the LCH to Dr Miller's report

Dr Miller's report was subsequently provided to the LCH for consideration and comment. Dr Hayden White, the Director of the Intensive Care Unit, and Dr Brian McGowan, the Director of Surgery at the LCH subsequently provided statements in response to the matters raised by Dr Miller. Neither Dr White or Dr McGowan were personally involved in Mr Hamilton's care, however, had a thorough understanding of the treatment provided at the LCH having carefully considered all of the relevant material, including Mr Hamilton's complete medical record, autopsy report and Dr van Buuren's report.

Response by Dr Hayden White

Dr White has been the Director of the Intensive Care Unit at the LCH since 2006. Having considered Dr Miller's report, he acknowledged the three main issues identified as follows:

(1) The patient's abdominal pain was due to pancreatitis, which was not diagnosed as a lipase was not taken prior to surgery;

(2) The patient should not have been discharged from the ICU on 5 September 2013; and

(3) The Heparin infusion re-commenced the morning after surgery, exacerbated Mr Hamilton's intra-abdominal bleeding.

Dr White's responses to those issues, were as follows:

(1) Diagnosis of pancreatitis

Dr White notes that Mr Hamilton's presentation was very acute, in that he reported epigastric pain half an hour after having some soup for dinner and being completely well until then. He described pain with vomiting, radiating posteriorly to his shoulder blades. The initial CT scan report suggested a duodenal perforation, which was in keeping with his clinical presentation. The abdominal scan was not suggestive of severe pancreatitis.

Dr White further states that although lipase is more specific than amylase for the diagnosis of pancreatitis, an elevated lipase result can have other causes. As such, the online medical database, Uptodate notes that between 11 to 12.5 % of patients admitted to hospital with non-pancreatic abdominal pain, have an elevated serum lipase. The other testing conducted, namely the LDH results and hypocalcaemia, are nonspecific findings and are often abnormal in patients in intensive care.

Dr White notes that the initial blood gas, which was venous not arterial, reflected a primary respiratory acidosis, with a relatively normal metabolic component. Following resuscitation, the patient did develop a mild normal anion gap metabolic acidosis, however, this was most likely the result of fluid resuscitation with sodium chloride. Dr White is of the view that this is not in keeping with severe pancreatitis as was suggested by Dr Miller, where a high anion gap metabolic acidosis would be expected. Furthermore, Mr Hamilton's blood gas normalised before he was discharged from the ICU. The follow up CT scans conducted, failed to demonstrate evidence of pancreatitis. Therefore, apart from the high lipase taken post-operatively, Dr White does not believe that there was strong evidence of pancreatitis.

(2) Premature discharge from the ICU

Dr White notes that Mr Hamilton was admitted to the ICU on 5 September at 3:31 am following laparotomy surgery. He was extubated later that morning. He developed some chest pain but had no ECG changes, and his condition quickly settled. A CT pulmonary angiogram was performed and did not find evidence of an acute pulmonary embolism. Mr Hamilton's Adult Deterioration Detection System Chart from 5 September indicated that his observations were normal from midday until 5:30 pm when he was discharged to the ward.

Relevantly, the Surgical Registrar made a note on Mr Hamilton's chart, which stated, "ICU has discussed with Dr Ranasinghe, Happy to start Heparin infusion – started at 16:45 and happy to discharge to the ward."

Dr White is of the view therefore that the reasons referred to by Dr Miller as to why Mr Hamilton should have remained in the ICU, were not in fact present. Relevantly, the CT scans and laboratory findings made the diagnosis of pancreatitis unlikely, and were not 'red flags' as referred to by Dr Miller, indicating significant pancreatitis. Furthermore, entries in Mr Hamilton's chart on the following day, indicate that he was stable, suffering from only minimal pain after the cessation of the patient controlled analgesia, and had been allowed clear fluids.

Later that day, Mr Hamilton's urinary output started to drop and the surgical team treated this with fluids. A MET Call was made at 7:30 pm as his oxygen saturation level had dropped below 90%, the cause of which was thought to be respiratory compromise due to worsening abdominal distention. When he was repositioned, his saturations improved to 97%. Mr Hamilton was subsequently reviewed by the ICU consultant, who ordered a CT scan and admission to the ICU. This was followed by intubation in the ICU for agitation and worsening hypoxia.

(3) Heparin infusion exacerbated intra-abdominal bleeding

In relation to anticoagulation, Dr White notes that Mr Hamilton had a mechanical mitral valve, which creates a high clotting risk in itself. Prior to commencing anticoagulation therapy, the ICU team discussed this with the surgeons, and as there was no major resection or evidence of ongoing bleeding, anticoagulation was commenced. Dr White notes that international guidelines are vague in relation to anticoagulation following surgery, and it largely depends on the clinician's assessment of bleeding considering the risk of clotting. In Mr Hamilton's case, it was determined that the latter was more of a risk, and anticoagulation therapy was commenced. Dr White notes that on 6 September, Mr Hamilton's activated partial thromboplastin time score was good and there was contemporaneous clinical evidence of effective coagulation, with respect to the drain site.

Response by Dr Brian McGowan

Dr McGowan has been the Director of the Department of Surgery at the LCH since January 2007. In relation to the three issues identified by Dr Miller, as outlined in Dr White's report, Dr McGowan provided the following responses:

(1) Diagnosis of Pancreatitis

Dr McGowan notes that although Mr Hamilton's lipase was raised post-operatively, it is not clear whether this was as a result of manipulation of the pancreas intra-operatively. A pre-operative lipase was requested on admission but not performed due to a lab error (that being that there was insufficient blood sample to test). The lipase was repeated a few hours later after the surgery. Furthermore, Dr McGowan highlights that Mr Hamilton's lipase was normal at 5:00 am on 8 September, 2 days later.

In any event, Dr McGowan is of the view that a pre-operative lipase probably would not have avoided the laparotomy, given the radiologist's report that suggested an acute perforation of the duodenum. Furthermore, an elevated lipase is not pathognomonic and also goes up with ischemia and perforation.

Dr McGowan subsequently reviewed the CT scans taken of Mr Hamilton's abdomen pre- and post-operatively, and noted that whilst there is some retroperitoneal fluid around the pancreas, the pancreas itself is not oedematous or inflamed. There are also other potential causes for retroperitoneal fluid, such as a duodenal diverticulus. In Dr McGowan's opinion, pancreatitis, whilst still a possibility, was far from definitive based upon the CT evidence.

Dr McGowan is also of the view that Dr Miller's interpretation of the pre-operative blood gas is incorrect. Firstly, the blood gas was venous, not arterial. Secondly, he notes that the academia is a result of high CO2 levels and the HCO3 is normal. This suggests a respiratory cause rather than metabolic. If pancreatitis was the cause, it would be expected that there would be a low HCO3 with high anion gap. Furthermore, the arterial blood gas taken on 5 September at 1:42 pm prior to discharge to the ward, was entirely normal.

Accordingly, Dr McGowan is of the view that the diagnosis of pancreatitis was far from clear.

(2) Premature discharge from the ICU

Dr McGowan is of the view that Mr Hamilton was not prematurely discharged from the ICU, given the clinical observations recorded. Whilst he concedes that this matter is best addressed by Dr White, he notes the following relevant matters in support of his view:

- Mr Hamilton was in the ICU for approximately 12 hours post-operatively. At no time during his stay in the ICU did he demonstrate any signs to indicate he was bleeding. In fact, Mr Hamilton's blood results improved during his stay in the ICU, and his arterial blood gas was completely normal pre-discharge. His observations, including urine output, blood pressure and heart rate were entirely normal pre-discharge.

- A CT scan of the chest was performed on the afternoon of 5 September, and although not focused on the abdomen, the upper abdomen was visualised. There were no signs of bleeding at this time.

- The ICU Registrar attended the MET call on 6 September at around 7 pm. The ICU Consultant subsequently reviewed Mr Hamilton's matter, who sent for an urgent CT scan and admitted

him directly to the ICU.

(3) Heparin infusion exacerbated intra-abdominal bleeding

Dr McGowan notes Dr Miller's comment that given Mr Hamilton had increased bleeding during his procedure, which necessitated the need for a drain placed into the abdomen, his Heparin infusion should not have been recommenced the following morning. Dr McGowan is of the view that it was not inappropriate to commence the infusion given Mr Hamilton was obviously not bleeding at the time of commencement (5 September at 4:45 pm), and had a mechanical mitral valve. Furthermore, the following morning, Mr Hamilton's activated partial thromboplastin time score had only slightly risen when his abdomen was already distended. He notes that they routinely start Heparin infusions in post-operative patients in the surgical wards, which he does not believe is a good reason for a patient to remain in the ICU.

Dr McGowan is of the view that the decision to anti-coagulate was sound considering Mr Hamilton had a prosthetic mitral valve, had recently undergone surgery and was suffering from pancreatitis, which are all prothrombotic states. He notes that generally, haemorrhage is a manageable complication, as opposed to a major stroke due to embolization from a prosthetic valve, which is an irreversible catastrophe for which failure to anticoagulate could, in some circumstances, be considered potentially culpable.

Conclusion

Michael Hamilton was 72 years of age when he died on the 13 December 2013. The cause of Mr Hamilton's death was multiple organ failure due to sepsis caused by abdominal adhesions inflicted during a previous surgical procedure. He had a significant medical history, having previously undergone coronary surgery and the insertion of an artificial mitral valve.

The central issue in this case is the adequacy of the initial assessment and treatment provided by the LCH, following Mr Hamilton's presentation on 4 September 2013. Dr Miller is critical of the initial management and assessment conducted, and suggests that in retrospect, a diagnosis of severe pancreatitis was quite compelling. He is also of the view that the decision to transfer Mr Hamilton from the ICU on the day of surgery, and to recommence the Heparin infusion, was inappropriate.

Whilst I agree that there may have been some missed opportunities during Mr Hamilton's initial assessment, such as the failure to conduct a lipase test, I accept that his clinical presentation was quite complex, and that more than one possible diagnoses was open, considering his symptoms and the results of the extensive testing conducted. Whilst pancreatitis was identified as a possible cause for Mr Hamilton's condition, a suspected perforated duodenum certainly fitted with the results of the initial tests conducted. I accept that the subsequent decision by treating staff to transfer Mr Hamilton from the ICU following the

laparotomy, and to recommence the Heparin infusion, was done so following careful consideration of his clinical presentation and pre-existing medical conditions. Regardless of the missed opportunities during Mr Hamilton's initial assessment, it is clear that such matters did not affect his eventual outcome, and the care and treatment he received at the LCH prior to his death, was appropriate.

The circumstances surrounding Mr Hamilton's death have been thoroughly investigated during the course of the coronial investigation. Having considered the material obtained, I am satisfied that I am able to make the requisite findings as required under the Coroners Act. Accordingly, I am of the view that there are no further lines of enquiry, which require consideration by way of an inquest.

James McDougall
Southeastern Coroner
Southport
8 July 2015