Penrose Inquiry

Report on Mrs O'Hara by K Robertson 24th February 2011

I have attempted to ascertain what aspects of Mrs O'Hara's care are of interest to the Inquiry but to date neither NHS CLO or the Inquiry team have made these plain. For this reason I have provided a summary of her management whilst under my care. This is constructed from her case records as I do not recall the case in detail.

I treated Mrs O'Hara at Stobhill Hospital in 2003 in my capacity as a consultant surgeon. I hold the qualifications of MB, ChB, FRCS and MD.

Mrs O'Hara was an emergency admission under the care of Mr McMahon, also a consultant surgeon at Stobhill Hospital. She was admitted on the 26th March 2003 with abdominal/ back pain and vomiting. Her blood amylase was 1194 U/l. In conjunction with the clinical picture this was considered diagnostic of acute pancreatitis. She had, in fact, seen her GP the week preceding admission with similar symptoms and was noted at that time to have an elevated amylase (p226-7).

An ultrasound scan demonstrated gallstones in the gallbladder and also a fluid collection adjacent the head of pancreas (p244). This, in conjunction with a minimal alcohol intake (p239), made gallstones the most likely aetiology.

Routine supportive care with analgesia, fluids etc. had been instituted at the time of her admission. She developed pyrexia on the night of admission and liver function tests (LFT's) at this time were slightly deranged. This raised the possibility of cholangitis (infection in the biliary tree). With gallstones in this structure as the most likely cause of her pancreatitis, this fitted the clinical picture.

A further spike in temperature occurred on the 28th March (p245).

Mr McMahon asked if I would assume her care as I have an interest in managing this disorder. I did so on 28th March. My note records that I felt she should have ERC+ES (endoscopic retrogrde cholangiography and endoscopic sphincterotomy) or LC (laparoscopic cholecystectomy) prior to discharge. This is in line with both British and World pancreatic guidelines.

Mrs O'Hara had a complex past medical history and was taking multiple medications. I have not listed these here but they are documented in her records – page 237. She is described by her GP practice as 'well known to the hospital'. Her records indicate regular medical reviews (Diabetic, Cardiology and Gastroenterology) over a protracted period. Her medical problems made general anaesthetic (GA) for LC, with the risk keyhole surgery would need converted to open surgery, unattractive. I felt she would be best managed by ERC+ES.

CT (31st March, p607) scanning was arranged and conservative treatment was continued. A plan was made for ERC on 3rd April. During this time leg swelling and chest crepitations were noted and medical review was sought (p249-50). Input from the nutritional team was also obtained.

On the 1st April Mrs O'Hara's warfarin medication (anti-coagulant or blood thinning drug), required for her prosthetic heart valve, was stopped as a prelude to her ERC+ES as this procedure involves cutting a muscle at the bottom of her bile duct and bleeding is a risk. It was substituted by Heparin (also an anticoagulant) as this drug's effect can be better controlled allowing it to be stopped for a short period at the time of her ERC. Unfortunately ERC had to be postponed because Mrs O'Hara's anti-coagulation had not reduced adequately by the 4th April. It was re-arranged for the 7th April.

Meanwhile she had been responding to the supportive measures in place but remained unwell. ERC on 7th April failed. This was largely due to distorted anatomy consistent with her diagnosis of acute pancreatitis (p6). Following this procedure Mrs O'Hara became more unwell; spiked temperature and low blood pressure. She was transferred to the High Dependency Unit and Anaesthetic review was obtained (p262). Clinically she was felt to be septic and have pulmonary oedema (fluid in the lungs – most often associated with cardiac failure). She was commenced on Tazocin (a broad spectrum antibiotic) and adrenaline (a drug that can increase blood pressure through an effect on heart, blood vessels and kidneys).

Mrs O'Hara made a gradual improvement but continued to spike her temperature. On the 10th April ERC+ES was re-attempted (p5). Again this proved difficult and was complicated by bleeding at the sphincterotomy. This required endoscopic treatment which was given at the time. It had been anticipated that this was a high risk intervention and a consultant anaesthetist attended the procedure (It had also been discussed with Mrs O'Hara and her daughter) (p273). The procedure was cut short due to this bleeding and concerns over Mrs O'Hara's airway. Further anaesthetic input was sought following the procedure (p272).

Mrs O'Hara did drop her haemoglobin and had some heavily blood stained bowel movements in the days thereafter but she remained haemodynamically (Blood pressure and heart rate) stable (p274-277). She did however receive a blood transfusion of 4 units. The situation with respect to bleeding was particularly difficult as she required further anti-coagulation after ERC because of her prosthetic valve.

Over the next few days she improved. By 14th April she was described as 'Doing well. No complaints of pain. Obs stable'(p280).

The possibility of SBE (subacute bacterial endocarditis – an infection of the heart valves of which Mrs O'Hara was at greater risk because of her replacement valve) was raised (p272). Cardiology review was requested to address this concern and request help with her cardiac/ fluid management. She was seen on 15th April (p283-285).

CT was repeated on 16th April. This suggested that the pancreas was not necrotic (parts can infarct in acute pancreatitis) and was not oedematous (swollen). By this point CRP (a marker for inflammation/ infection) was 25 – this is elevated (normally less than 20) but much less than the 135 recorded on the 9th April (page 46). Along with her improved clinical condition her acute pancreatitis seemed to be resolving.

On the 18th April a final ERC+ES was performed at which sphincterotomy was completed. This was an essentially uneventful procedure.

Over the next few days she seemed to make progress. There were difficulties with her diabetic control and her re-warfarinisation (obtaining the correct level of anti-coagulation) but advice was sought from the diabetic service and haematology. We were encouraging mobilisation and beginning to think about possible discharge.

On the 23rd April it was noted that her leg swelling was worse as was her abdominal distension. An ultrasound confirmed ascites (fluid in the peritoneum) and liver changes consistent with cirrhosis and portal hypertension (increased pressure in the veins draining the gut caused by cirrhosis) (p298). Gastroenterology review was sought and obtained on 24th April (p300). They advised that a sample was taken and this was done but did not prove diagnostic. They also modified her medication.

On the 26th April she managed to attend her son's wedding.

By this stage I was of the opinion that her problems no longer related directly to her pancreatitis but it was unclear what precisely was the underlying cause. She clearly had complex cardiac and hepatic pathologies and it seemed likely that decompensation of these was the cause of her ascites and leg swelling/ oedema.

She continued under gastroenterology review (p303 and 307). On the 1st May coliforms (a gut bacteria) were cultured from a swab of her now ulcerated swollen legs. Antibiotics were prescribed as advised by bacteriology colleagues. Further Gastro/ Cardiac review was requested as her ascites/ leg oedema was unimproved. Advice was also sought from tissue viability and dermatology services.

Further medical review was obtained on 2nd May. Several alterations to her medical management were made and TOE (trans-oesophageal echo – an ultrasound method of assessing the heart and its valves was suggested). Early medical review in event of general deterioration was advised.

On the 3rd she did deteriorate with increasing confusion and shortness of breath. Renal function had also deteriorated. Further medical review was obtained (p327-30). ITU review was also sought (p331-2) but ITU admission was thought inappropriate. That evening she was transferred to CCU (Coronary Care Unit) under the care of the cardiologists/ physicians. I was minimally involved thereafter.

I do not propose to make comment on her care in CCU; her carers there are in a better position to do so. I note that she died on the evening of 7th May 2003.

The correspondence I have from NHS CLO (and indirectly from the Inquiry team) has asked for my comments on:
1. If her Hepatitis C might have contributed to her death. I think this is difficult for me to answer. The management of Hepatitis C and its complications is outwith my knowledge and experience.
2. To comment on the completion of her death certificate. I did not issue it. At the time of her death I was no longer her principal carer. It is usual practice for the consultant team providing care at the time of death to issue the certificate.

3. To comment on my discharge letter. No specific issue has been raised. I provided a discharge letter for this lady as she was under my care for a protracted period with complex problems. Of her carers, I felt I was best placed to speak to the management of her pancreatitis and the period preceding her transfer to CCU. As she died there, following a number of medical interventions, I had thought a letter might also be sent from that unit. I have not found one in the notes I have.