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Endoscopic procedures (3 cases)

Beware Aspiration in Elderly Patient with Oesophageal Motility Disorder

Summary

An 84 year old woman presented with a 4 year history of difficulty swallowing, worsening in the preceding 6 months and unable to swallow water in the final week. She was transferred for endoscopic review, with a view to PEG insertion. Upper GI endoscopy 4 months earlier demonstrated distal oesophageal ulcer, and the oesophagus was said to be difficult to distend. A barium swallow 15 months earlier was said to show corkscrew oesophagus.

Upper endoscopy was performed on the evening of admission, under light conscious sedation. The oesophagus was found to be dilated, with "food ++, ? stricture distal oesophagus". Oesophageal dilatation was performed with a 20mm Savary dilator without immediate event. Post-procedure, the patient was noted to have moist cough with frothy sputum, persisting overnight, and was helped with suction. She was reviewed by the surgeon in the morning after the procedure. CXR and CT of neck and chest were organised, which demonstrated grossly distended oesophagus filled with food throughout its length, no mass, moderate-sized right pleural effusion, small left effusion and bilateral basal atelectasis. She was also referred for speech pathology review. On the whole, the patient seemed to be as well as she was prior to the procedure.

Two days post-procedure, she was noted to have a significant fall in O_2 saturation (88% with nasal prongs), tachypnoea and right lung crackles. This was clinically assessed as aspiration pneumonia, physiotherapy and appropriate IV antibiotics were started, and the family notified. CXR next morning confirmed severe pneumonia with confluent opacification of bilateral mid and lower zones. The patient's health deteriorated until death on the 4th day post-procedure.

Comments

Although not documented well, this was clearly a patient with a severe, undiagnosed oesophageal motility disorder, superimposed on advanced Parkinson's disease, dementia, marked frailty and physical incapacity. Any procedure on such a patient is "high risk" and some record should be made that this risk was conveyed to patient/family/carer. The decision to postpone the PEG insertion at endoscopy was well judged.

Areas of consideration:

A more thorough assessment of the underlying motility disorder may have led to more appropriate and effective therapy. For instance, "? stricture distal oesophagus" as reported, without compressive obstruction, is suggestive of lower oesophageal sphincter hypertension (achalasia), best treated, in this case, with botulinum toxin injection. It is unusual for other motility disorders to cause such food hold-up. If indeed achalasia was the problem and the patient responded to Botox therapy, she may not have suffered the terminal aspiration events, but her prognosis would remain guarded due to her progressive debility and poor oropharyngeal function. I assume an Eder-Puestow wire was used to guide the dilator, although the fact was not recorded. Some dilators are used without guidewires, but to do so with a Savary dilator is incorrect. Anaesthetic supervision is recommended when there is risk of aspiration, although there is reluctance in automatically requesting assistance in the public sector, related to limited resources. In this case, the surgeon appeared aware of the risk and used an appropriately cautious dose of sedative agents.

Area of concern:

Dilating a full oesophagus is contraindicated given the high risk of aspiration and mediastinal soiling if perforation occurs. The risk of perforation is also higher if there are areas of stercoral ulceration/pressure necrosis (which are often obscured by food residue). Attempts should be made to clear the full oesophagus to substantially improve visibility, irrespective of whether dilatation is to be subsequently performed. However, it is wrong to assume that the terminal aspiration pneumonia was due to the endoscopic procedure. The

patient was stable post-procedure for 2 days, and she was already at high risk of aspiration at any time, given the state of her oropharyngeal function and obstructed oesophagus.

Endoscopic Perforation of Duodenum

Summary

An elderly woman with significant medical co-morbidities including cerebro-vascular disease, polymyalgia rhuematica, hypertension, ischaemic heart disease and non-insulin dependent diabetes, underwent upper gastrointestinal endoscopy at a regional hospital. The endoscopy was said to have demonstrated a Schatzki ring and a biopsy was taken. Following this procedure, her condition deteriorated characterised by abdominal pain, vomiting and, subsequently, both hypotension and tachycardia. The possibility of gastrointestinal perforation was considered, broad spectrum intravenous antibiotics commenced, a nasogastric tube inserted and the patient transferred to a teaching hospital for further surgical management and appropriate supportive care. The patient was taken directly to theatre, surgery commencing less than two hours after her assessment in the emergency department of the teaching hospital.

Findings at surgery were those of a retroperitoneal perforation of the first part of the duodenum. According to the operative notes the duodenum was found to be friable, and the omentum was mobilised and placed behind the first part of the duodenum in an attempt to seal the perforation. A 19 gauge Blake's suction drain was placed in the right upper quadrant. The patient was returned to the Intensive Care Unit for ongoing supportive management.

Over the subsequent 72 hours, the patient appeared to make some progress although investigations confirmed that she had suffered an anterior myocardial infarction. She subsequently developed atrial fibrillation and, on the 5th post-operative day, features suggestive of progressive sepsis. Fluid taken from the right upper quadrant drain revealed high levels of amylase indicative of ongoing leakage from the duodenum but there was consensus amongst both managing medical staff and the patient's family that further surgical intervention was inappropriate. Accordingly, and with the full agreement of the patient's family, active management was withdrawn on the 6th post-operative day and the patient rapidly succumbed.

Comments

It is clear that this elderly woman with significant medical co-morbidities and, most importantly, suffering a myocardial infarction in the immediate post-operative period, was highly likely to succumb to this duodenal perforation. It is not at all clear why the duodenum perforated and the hospital case notes do not contain a formal record of the original upper gastrointestinal endoscopy. Just the same, the findings at operation appear to indicate that the patient had suffered perforation of a large duodenal ulcer and one that was clearly not ideally suited to treatment by omental patch. A more usual surgical approach would have been to consider some form of gastrectomy with either direct closure of the duodenal stump or, at the very least, closure of the duodenum over a drain. The case notes indicate that the consultant surgeon was present in the operating theatre. It is certainly inappropriate for junior surgical staff to be called upon to deal with complex duodenal perforation on their own.

Although subsequent duodenal leakage produced sepsis which contributed to the patient's demise, the likelihood is that the myocardial infarction suffered in the immediate post-operative period (which had nothing at all to do with the choice of operative procedure) was an equally significant contributor. Even had the duodenal perforation been dealt with more definitively, a fatal outcome would appear to have been likely.

Mortality from PEG Leak

Summary

The patient in her early 80s was re-admitted from a nursing home when she was sent back the same day after replacement of leaking PEG tube. It was stated that when she returned to the nursing home after the procedure she was agitated and flushed. Comment was also made that she had a "seizure" and was "cyanosed". She was given oxygen and her condition apparently improved before she was returned to the hospital. This lady also had replacement of PEG tube on two other occasions for leakage of feed around the tube. The indication for insertion of her PEG tube was dysphagia from cerebellar degeneration. Other relevant past history noted was dementia, hypertension, NIDDM, osteoporosis, and chronic lymphocytic leukemia. Her medication also included prednisolone 2.5mg daily.

Examination at the hospital revealed she was not in any distress. She was afebrile and haemodynamically stable. A chest X-ray revealed free air under the diaphragm. A CT scan the following day reported free gas under the diaphragm. There was difficulty visualising the gastric wall surrounding the PEG tube. Since she was febrile she was commenced on IV antibiotics. She was also commenced on water via the PEG tube. On the fourth day after admission she was reported to have hematemesis (approximately 500ml of dark red blood). Her abdomen was also commented to be tender with guarding. The PEG feed was ceased, and she was given IV infusion of omeprazole. At this stage it was recorded that she was seen by her consultant and referred for endoscopy. Endoscopy was carried out and revealed severe erosive oesophagitis and a large volume of altered blood in the stomach. "There was an area of mucosal irregularity on the anterior wall of the stomach." The tip of the PEG tube was not seen within the stomach. She was given three units of blood, and the PEG tube was removed. Two days after the removal of the PEG tube the patient developed tachycardia and was referred to the cardiology unit and a surgical consultant. She was seen by the surgical registrar who informed the consultant surgeon of the finding of abdominal tenderness and guarding. Laparotomy was carried out by the registrar the same day after anaesthetic assessment and correction of her electrolytes. She had also reverted to sinus rhythm.

During the operation she was found to have "free perforation from external PEG hole into peritoneal cavity" with evidence "of inflamed tissue with free intra-peritoneal fluid". Peritoneal lavage and open gastrostomy was carried out. Post-operative course was complicated by oliguria which responded to fluid challenge. She also required sliding scale insulin for control of her blood sugar. It was recorded that she was "going well" and the abdomen was soft and non-tender the day before she was found to be cold and unresponsive and did not respond to CPR. She was declared deceased.

Comments

- 1. It is difficult to ascertain from the notes why the PEG tube leaked. The procedure was carried out under three different consultants. The final dislodgment of the PEG tube may be due to the patient accidentally pulling at her tube due to her disorientation.
- 2. Why was there a delay of five days in referral to a surgeon when it was noted on admission that she had free gas under the diaphragm? As pneumoperitoneum may be normally present after insertion of a PEG tube and there was no evidence of peritonitis at that stage, she was not referred immediately for surgical review. Until there was signs of peritonitis, which appeared to be apparent after removal of the dislodged PEG tube, she was then referred for a surgical consult.
- 3. On reviewing her notes there was hardly any documentation of the patient being seen by the consultant whom she was admitted under.
- 4. Post-op care also lacked any documentation of visits by the consultant surgeon although there appeared to be continued care by the team doctor which was appropriate.

- 5. It is also interesting that she was reported to be "going well" just a day prior to her sudden death. As there was no post-mortem carried out I am not certain of the cause of death. I would suggest that acute myocardial infarct is most probably the cause of her death, rather than sepsis.
- 6. This case demonstrated mortality associated with a PEG tube. When a CT scan on the second day could not confirm the position of the PEG tube to be in the stomach, I feel contrast medium should have been injected into the tube to confirm its position. This may have initiated an earlier surgical consult and perhaps a different outcome.

Fluid Management (4 cases)

Problems of Fluid Balance in the Very Elderly

Summary

A frail 100 year old Vietnamese lady with a past history of pulmonary oedema combined with pleural effusion two months earlier, had fallen a week before presenting to a peripheral hospital. X-ray revealed a displaced sub-capital fracture of the hip for which transfer was arranged to a central hospital. Testing on admission showed a potassium of 2.6 with urea and creatinine within normal limits. Haemoglobin was 12.0 g/L and a white cell count 10,206.

By next morning it was noted that her urine output was very low, and her pulse was 122. Blood tests at this stage showed a CK level of 483 U/L (normal 20-160) and a troponin T level of 0.15 mcg/L (normal < 0.04). This was considered to show myocardial damage. Repeat haematology at this stage showed a haemoglobin of 10.7 g/L and a white cell count of 12,600. At this stage she was seen by a medical registrar who found there was no obvious cause for the poor urine output except possibly dehydration. Intravenous fluids were initiated on admission and therefore continued. Later that day she was seen by an anaesthetic senior registrar for consideration of anaesthesia for planned hemi-arthroplasty of the hip. He requested further investigations including coagulation studies, an echogram and a search for the cardiac enzyme results to be found as they were not with the notes.

By next morning she was noted to be in a positive fluid balance of over 3 litres. Further diuretics were given. On Day 5 an echogram was carried out with the report that there was severe impairment of left ventricular systolic function with multiple wall motion abnormalities consistent with underlying ischaemic disease, and moderate aortic and mitral regurgitation. There was elevation of right heart pressure. On Day 5 she was again reviewed by an anaesthetist who noted the echo report and felt that this lady was an over 80% mortality risk with anaesthesia. He did not feel that she could be improved any further and felt that surgery should be carried out the following day. He spoke to the son so that the family understood the risks. Surgery was cancelled the following day and took place on Day 7, with an orthopaedic consultant assisting with the insertion of an uncemented Austin Moore Prosthesis. Post-operatively her condition never really improved in that her renal function continued to deteriorate. Her Urea on Day 19 was 18.9 mmol/L. She died on Day 21. No post mortem was carried out.

Comments

Whilst this case does reflect the very high mortality risk of fractured neck of femur in the frail and elderly patient, I do feel there are two areas here which could be looked at. Firstly, the amount of fluid that she was given intravenously without significant response from diuretics. In the first 8 hours she had one litre of fluid. Over the next three days, she had 6 litres (mostly normal saline), 4 litres and 2 litres of fluid, respectively. Post-operatively, she had a total of 13 litres of fluid in 13 days. This lady weighed 40kg. It is a little difficult to be sure about these volumes because the intravenous fluid chart is slightly unclear. I could not find any urinary output chart in the notes. It should be borne in mind that this lady had evidence of cardiac ischaemia and an abnormal cardiac echo report. An opinion from a renal physician or a geriatrician may help clarify this area. I note, however, that her cause of death was given as congestive cardiac failure.

Secondly, there would also appear to have been some delays because the surgery did not take place until day 7 of her admission. One cause of some of these delays would appear to be that the echocardiogram was requested on day 2, but only carried out three days later. Subsequently, her surgery was cancelled on one day, so was postponed for 24 hours. These types of delays are always inevitable with our congested hospital services and result in deterioration in the condition of this type of patient. The overall care given to this elderly non-English speaking woman by nursing and medical staff appears to have been otherwise excellent. Given that she did not present until a week following her fall, and it was then another six days before surgery could be carried out and, with her age and history of cardiac ischaemia, I feel that she would have been very fortunate to have survived this chain of events.

Central Venous Pressure Monitoring Might Have Helped to Manage Fluid Intake

Summary

A 90 year old patient with a previous history of chronic renal failure, dementia and myelodysplastic syndrome was admitted to ED after a fall in the bath of the nursing home. The patient was diagnosed with a fractured neck of femur of his left hip. After primary assessment in the ED and by the orthopaedic team, he was brought to theatre five hours later and a Thompson's hemi-arthroplasty was performed. A single dose of frusemide 40mg was given during surgery. Within the first 24 hours of admission and operation the patient received three litres of CSL, 1½ litres of saline and one unit of blood. The haemoglobin at the time of admission was 77, on the first post-operative day it was 69, and on the third post op day it was 76. On the second post-operative day he received two units of blood, and three days post-operatively another two units of blood. The urinary output was recorded as adequate on the day of operation as well as on the first post-operative day that the patient had a low urinary output and pulmonary oedema developed. The haemoglobin at that time was 76, indicating some hemodilution. Intravenous frusemide was administered. In the following days the patient deteriorated and passed away on the fifth post-operative day. The cause of death was listed as pulmonary oedema and post-operative ischaemic heart attack.

Comment

In the first-line assessment, the question arose whether five litres of fluid in the first 24 hours and one unit of blood could have caused some pulmonary oedema in a patient with known chronic renal failure. Unfortunately, it was not possible for me to track the urinary output of this patient, as according to the nurses the charts with the urinary output are not kept in the file. It also looks as if there was no central venous pressure monitored over this time period. As these elderly patients with neck of femur fractures generally arrive dry in the ED and mostly suffer from ischaemic cardiac disease, careful re-hydration is important. This can only be ensured with the monitoring of input, output and central venous pressure. Therefore, to avoid an overload and pulmonary oedema it would be recommended that patients with pre-operative anaemia who need transfusion and re-hydration could benefit from a central venous line and careful monitoring of urinary output.

Nevertheless, in the case of a 90 year old patient, a fracture of the neck of femur is a major event and the mortality rate is reported to be extremely high.

Importance of Peri-Operative Assessment

Summary

The patient was an 84 year old man who was admitted electively for a right hemicolectomy to manage a polyp in his caecal area. His significant past history included having previously undergone a resection for a cancer of his left colon. He also suffered from ischaemic heart disease and may possibly have had an acute

myocardial infarct in the past, and suffered from atrial fibrillation. The patient was also in mild renal failure on admission, with a creatinine of 158 and a urea of 8.8.

He underwent the procedure, which took approximately 1.75 hours to perform and was sent back to the ward. For the first few hours after being on the ward, the patient was noticed to be restless and his saturations tended to be dropping to the low 90s, despite being on oxygen. He underwent a repeat ECG, which showed no significant new changes on the pre-operative ECG, which was not available for me to review. It was also noted at this stage that the patient was in atrial fibrillation. His urinary output remained low for the rest of the day and he received some moderate boluses of IV fluids to try and improve his situation.

The day following the operation, the patient was noted by nursing staff to have moist respirations and to be generally sweating and to continue to be a little bit restless. His respiratory rate was sitting around about 30 and in the early morning his saturations had dropped down to 82%, despite being on oxygen. He was given IV Lasix 20mg to manage what I presume to be some heart failure and also due to the fact that his urine output continued to be oliguric sitting at below 50ml now.

In the afternoon the patient collapsed and had a cardiac arrest and was not able to be resuscitated.

Comments

Perusing the notes, it is difficult to get a really accurate picture on how unwell this patient was prior to the operation and what his ASA class was. It is also difficult to know if he underwent any reviews by the cardiologists prior to his surgery. It is also interesting to comment that I cannot find any evidence that the patient had any DVT prophylaxis prior to his surgery.

My overall impression of this case is that this is an elderly gentleman who represented a significant risk for major surgery and I feel it would have been more appropriate for him to have had his surgery at a major tertiary centre, with both coronary care and intensive care unit backup.

Although there is really only level III evidence to support the use of High Dependency Units and Intensive Care Units in managing elderly unwell patients having major surgery, I feel that it may have benefited the patient in this case, with more careful attention to maintaining his saturation levels over 90% and then preserving an adequate urine output for him, especially in view of the fact that he already had some mild renal failure. It is difficult to know whether his death was really preventable, but his chances may have been improved with this more intensive post-operative care.

The only confusing point to this case is that there is an ECG in the notes, which shows evidence of a lateral and inferior myocardial infarct, but without any clear evidence at the top of the chart as to when this EGG was performed and whether any action was taken with regards to that information.

Time For Review of Post-Op IV Fluid Regimes

Summary

An 82 year old man presented with an obstructing carcinoma of the rectosigmoid, the diagnosis being established on limited contrast enema. He was a permanent resident of a nursing home having suffered a cerebrovascular accident with left hemiparesis in the past. He was a long-term smoker and an active smoker at the time of his admission.

Within 24 hours of admission he underwent resection of his obstructing rectosigmoid malignancy with primary re-anastomosis and construction of a temporary diverting loop ileostomy.

In the post-operative period, he encountered a persistently low urine output with high nasogastric aspirates and a post-operative haemoglobin of 7.7. This was managed by blood transfusion and colloid and crystalloid resuscitation.

On the second post-operative day he became increasingly short of breath, agitated and overtly hypoxic. A diagnosis of respiratory failure secondary to a presumptive diagnosis of bilateral chest infection and pulmonary oedema was made and he was transferred to the Intensive Care Unit for respiratory and other support.

His course over the next week was one of steady deterioration characterised by increasing respiratory failure and subsequent multi-organ failure. In view of his age and significant co-morbidities, his overall prognosis was deemed poor and his condition steadily deteriorated until his death on the ninth post-operative day.

Comments

From the time of admission until the start of his surgery he received 4 litres of intravenous normal saline; during the three hours of his operation he received 3 litres of Hartmann's solution and 1 litre of Gelofusine. From the completion of surgery the same afternoon to his transfer to the Intensive Care Unit about 48 hours later, he received 3 litres of Hartmann's solution, 4 litres of normal saline, 2 units of packed red blood cells and a 250m1 bolus of Haemaccel. Therefore, in a period of 72 hours this patient received over 15 litres of salt-based intravenous fluids as well as 2 units of blood. No dextrose-containing fluid was administered.

In this patient, clinical deterioration on the second post-operative day was predominantly due to respiratory distress. The history of this patient having smoked for in excess of 50 years clearly indicated that respiratory complications were likely. Patients with established obstructed airways disease are even more vulnerable to pulmonary oedema in the event of salt and fluid overload than are individuals with normal airway function. There seems little doubt that the respiratory function of this clearly unwell and elderly individual was aggravated by indiscriminate transfusion of salt-containing fluids. Post-operative oliguria need not always imply dehydration and the requirement for increased fluid replacement. The total volume of fluid transfusion in this individual may also have been excessive.

It is important to emphasise that no formal written record of the hourly urine output for this individual, for this admission, could be located within the hospital case notes. This is a notable omission and serves to obscure the clinical circumstances in which decisions about intravenous fluid replacement were made.

Notwithstanding concerns about the volume and nature of fluid transfused, this elderly man with significant medical co-morbidities presented with a genuine surgical emergency for which an attempt at surgical correction was always likely to be dangerous. Although post-operative fluid management might arguably have been better, it is difficult to be sure that such improvement would have altered the ultimately fatal outcome. However, it should serve as an opportunity for the medical staff to review their post-operative intravenous fluid protocols since there is increasing awareness of our tendency to over-transfuse both fluid and salt in patients recovering from major abdominal surgery.

Urology (2 cases)

Beware the "troublesome" SPC

Summary

The patient, a non-English speaking Vietnamese lady in her late 70s, presented to ED because there was no urine output after her first SPC change.

This lady suffered episodes of urinary retention in the months subsequent to a left CVA and ultimately a long-term IDC was inserted. A decision to convert this to an SPC was made because of multiple problems, and hence one was sited using an Add-a-cath introducer as a day-case procedure a month prior to this admission. She was discharged once good return from the catheter was established. Her medical history included hypertension, asthma and NIDDM.

Two weeks after her SPC was sited she presented to ED with fever, purulent discharge and urinary leakage around her SPC site and per urethrally. A *Pseudomonas* UTI was confirmed on a prior specimen and she was discharged home with an appropriate antibiotic. The family were expecting a follow up with the specialty unit, but had not heard back to confirm.

At her first SPC change by Silver chain two weeks later, lack of urine output and suprapubic pain triggered an ED presentation. At ED she was noted to be tachycardic and tachypnoeic with suprapubic pain. ED staff changed her catheter and discharge was planned (after consultation with the specialty unit), but because her relatives had left the department, she was admitted overnight.

Overnight she complained of bladder fullness and eventually in the morning after team review, an IDC was passed and further investigations sought. A cystogram via the SPC confirmed it was lying within a small bowel loop.

A laparotomy was performed that day and findings were consistent with a through and through puncture at the time of SPC insertion. Bowel resection, was carried out with stapled re-anastomosis, closure of fistula and omental interposition. Post-operatively she was given IV antibiotics, subcutaneous heparin, and chest physio. She went on to develop an infective exacerbation of asthma as well as post-op MI, and she deteriorated despite HDU support. A decision was made not to resuscitate and she succumbed 5 days post-op.

Comments

Bowel perforation is a recognised complication of percutaneous SPC siting. The decision to change to SPC and the method used to site it were appropriate. In this case it appears that initially the catheter was seen to drain urine and the patient was therefore discharged as anticipated. Presumably she and her family were instructed as to what to look for should problems arise, but this was not documented in the notes. It is possible that language difficulties may have contributed, as it seems to have done on subsequent presentations.

Once the complication was recognised, operative repair was carried out speedily and appropriately. However, it could be argued that the delay in recognising the complication may have impacted on her preoperative fitness and ultimately her outcome.

There were two occasions where this lady presented with problems associated with her catheter and on both occasions discharge was arranged without the perforation being recognised (although in fairness to the last ED doctor it was included in the differential diagnosis). It is fortuitous that she was admitted eventually, but this had little to do with her medical assessment at the time.

It is also interesting to note that the nursing observations in ED remark on "? faecal stained" urine. It was also the nursing staff that later recognised her distress in retention which would have hinted that the fluid draining from the SPC was not in fact urine.

This lady had also not been given a follow-up appointment as expected two weeks post-catheter insertion. The reasons for the delay in follow-up are obscure in this case. Despite this, first hand review by the specialist team appears not to have been carried out until the morning after admission. One wonders whether early review (as opposed to telephone consultation) by the team may have expedited recognition of her perforation.

Recognition may have been made more difficult as the patient did not mount a significant reaction to sepsis and, as stated, language issues could have hindered history taking. Also the drainage from the SPC was reported as 'free flowing''. At each presentation, the urine was recorded as being turbid and/or with bacteria in abundance. Whilst a UTI is a common complication of catheter insertion, occasionally such a finding is indicative of more serious problems as this case illustrates.

I stress that there has been no one major adverse event that has impacted on this lady's management. Rather, a lowered threshold of suspicion for a potential complication.

A small criticism should be made in regards to the discharge letter, which lacks detail, but it is noted that the GP had been telephoned and presumably more information would have been given.

A Higher Index of Suspicion for Early Post-Operative Bleeding following Laparoscopic Surgery

Summary

A patient in his early 70s with a history of asbestos exposure and chronic airways limitation was noted to have a discrete opacity in the right upper lobe on a routine follow up chest x-ray. Investigations confirmed a small early primary lung malignancy. A CT scan staging for the lesion also identified an incidental 2.5cm solid mass arising from the left kidney consistent with a primary localised renal cell carcinoma. The lung primary took precedence and the patient underwent a wedge resection of the right upper lobe. He made a good recovery aside from readmission for a chest infection 6 weeks post-operatively. At 3 months following his surgery, he was given a complete clearance from a medical point of view to undergo definitive treatment for his left renal tumour.

A transperitoneal laparoscopic left nephrectomy was undertaken. A haemostatic clip was applied to a single renal artery and a vascular staple to a single renal vein. There was no documentation on the operative record of any intraoperative difficulties or excessive bleeding. However, a subsequent surgeon report on the operation described a greater than normal general ooze during the procedure that required the consultant surgeon to take over from the senior registrar half way through the procedure to complete the operation. However, there was no note made of any intraoperative complications and at the end of the operation, there was no evidence of active bleeding. In recovery, however, the patient's systolic blood pressure dropped from 130 to 96 over a period of 1 hour and 50 minutes. The patient was given 4 litres of crystalloid in theatre and 2 litres in recovery, but despite this fluid intake his blood pressure was systolic 96 and diastolic 59. The anaesthetist was informed and the patient was given clearance to return back to the ward.

As soon as the patient arrived on the ward, a systolic blood pressure measurement was noted to be 80 associated with a reduced urine output. There was minimal drain output, but the patient was not complaining of any significant abdominal pain. He was given further IV fluids and 2 units of Haemaccel. His blood pressure temporarily increased to a systolic of 90, but within 30 minutes dropped to 80. At that stage, blood for a full blood picture was taken by a surgical resident, but due to failure to label the specimen bottle there was significant delay in receiving the result which was 78. His blood pressure at this stage was still systolic 78 and diastolic 58. Exactly 3 hours and 40 minutes after returning to the ward, the patient arrested and the

emergency resuscitation team was called. Four units of packed cells of blood were given immediately and the patient returned to theatre.

A midline laparotomy was performed. Blood within the peritoneal cavity was evacuated and bleeding was noted at the adrenal bed and oversewed. However, blood was seen to be arising from the left paracolic gutter. This was shown to be from the splenic hilum, but the bleeding was unable to be controlled with sutures or clamps. A general surgeon was called and an emergency splenectomy was performed. An injury to the short gastric and tail of the pancreas was also noted. The patient was transferred to ICU but died of cardiogenic shock from an inferior myocardial infarction.

Comments

Assessment of post-op bleeding in the early immediate post-operative setting can be difficult especially in a situation where the surgeon feels that there was no worrisome bleeding at the end of the surgical procedure. In addition, there are several other causes of hypotension in recovery aside from bleeding such as inadequate fluid replacement during the operation, the effects of an epidural anaesthesia or myocardial causes. However, in this case there was sufficient evidence in the recovery that the patient's worsening hypotension was likely to be due to bleeding. The patient had no new changes on the ECG in recovery and was given 6 litres of crystalloid in theatre and recovery that was more than adequate. In addition, the patient did not have an epidural. In this setting, it is surprising that the patient was allowed to return back to the ward with unexplained hypotension without even a haematocrit reading or request for a consultant surgeon review in recovery.

Upon return to the ward, the surgical resident and senior registrar assessed the patient on several occasions, but all of the entries in the notes clearly showed a failure to appreciate the possibility of active bleeding. This led to a very long delay of 4 hours and 40 minutes before the patient was returned to theatre to evaluate and treat the bleeding. There were also several entries in the notes made regarding the absence of blood output via the drains and this is a good reminder that drains are generally very ineffective at draining blood and should never be used as an indicator of intra-abdominal bleeding.

Lastly, it should have been appreciated that patients who have laparoscopic surgery should have perhaps a high index of suspicion for early post-operative bleeding compared to patients with open surgery due to the possible "masking" of intraoperative bleeding due to the increased intra-abdominal pressure from the pneumoperitoneum. In fact, there are many reports especially in the case of laparoscopic nephrectomy of reducing the pneumoperitoneum pressure to 5mm Hg at the end of the procedure to allow a more accurate thorough abdominal inspection for bleeding. Certainly, I am not recommending that this should be a universal technique in all patients undergoing laparoscopic surgery. However, it may be a technique that can be used in patients where there may be some concern with greater than normal intraoperative bleeding as appeared to be the case with this patient.

In summary, early post-operative bleeding should be suspected in patients with unexplained increasing hypotension not responding to fluid replacement and the index of suspicion for bleeding should be heightened even further in patients following laparoscopic surgery.

General Surgery (8 cases)

Unexpected Death Following Minor Hernia Surgery

Summary

This elderly man was admitted for elective repair of umbilical hernia under general anaesthetic in the morning, had his surgery three hours later, and was dead approximately 11 hours after surgery. He became hypotensive post-operatively despite fluid replacement, intravenous steroids and dopamine infusion.

There were no medical notes regarding his pre-operative medical status, but note was made in the nursing admission of previous myocardial infarction and coronary bypass grafting. His weight was 92kg and he was ASA 3.

Comments

This patient died unexpectedly after routine elective minor surgery. There were no identifiable adverse events in the peri- or post-operative periods that contributed to his death. However, there is no documentation of this man's pre-operative cardiac status, despite there being evidence of significant cardiac events in the past. As he was not seen in a hospital outpatient clinic then this is a common event. Therefore, no comment can be made regarding whether his cardiac status was stable enough for elective surgery.

His cause of death was given as myocardial infarction, yet there is no evidence to support this. No postmortem was requested, and this is a case where useful information may have been obtained from such an examination.

No DVT prophylaxis was given, despite the patient being elderly, overweight and having a past history of ischaemic heart disease.

Inadequate Large Bowel Surgery

Summary

This 84 year old gentleman was admitted for a left hemicolectomy after a colonoscopy showed a carcinoma of the splenic flexure. Past medical history included Parkinson's Disease, myocardial infarctions and back pain. He has had a TURP and multiple joint replacements in the past.

At laparotomy a transverse colon carcinoma was identified. A segmental resection was performed. The anastomosis was made using a two-layered hand-sewn technique. Histology was consistent with a Dukes A carcinoma.

Post-operatively, the patient appeared to be recovering well, except for signs of confusion. On post-operative day 7, the patient was noted to be unwell and had abdominal distension. An abdominal x-ray showed pneumoperitoneum. At laparotomy, a perforation of a descending colon diverticulum was identified. This was oversewn with O silk. A 22 Fr French catheter caecostomy tube was inserted. His second post-operative period was complicated by atrial fibrillation and periods of low urinary output. His general condition deteriorated over the course of the next few weeks and died 28 days after admission.

Comments

This case highlights a few issues.

With this gentleman's pre-morbid condition, a segmental colectomy may have had merits, but I suspect that in most situations either an extended right or left hemicolectomy would be the operation of choice. This would be more oncologically correct. It would also eliminate the possibility of vascular insufficiency when the proximal and distal parts of the transverse colon are anastomosed. The marginal vasculature can be compromised in the elderly especially if the middle colic vessels are ligated.

It is unfortunate that this gentleman developed a perforation of the descending colon diverticulum postoperatively. I was unable to identify from the operating notes if the affected part of the descending colon was inflamed and thickened. The extent of peritoneum contamination was not mentioned. The mortality rate from faecal peritonitis in this age group is high, regardless of surgery. However, to give this gentleman the best chance of survival a Hartman's type procedure with resection of the perforated diverticulum \pm anastomosis would certainly be the safest. Oversewing the perforation, especially if the colon was inflamed, is not an ideal situation, and O silk may not be the suture of choice. This is more so if there is evidence of faecal peritonitis.

This gentleman remained quite confused after his second procedure. He never quite got back on to a diet. Perhaps this is because of his gradual deterioration. Whether a referral to a tertiary institution and use of TPN would have made any difference is difficult to tell. I suspect not. Nevertheless, I was not able to tell from the inpatient notes if this was considered.

I did not see any contraindications to the use of heparin for DVT and PE prophylaxis in this patient. Heparin was actively withheld by the surgeon in this case. Most of the documentation was done by the nursing staff with regards to the care of this patient. The record keeping from the surgical staff is lacking, especially in light of the serious nature of this patient's condition.

In summary, this gentleman had an unexpected complication of perforation of a descending colon diverticulum after segmental colectomy. In order to give this gentleman the best chance of recovery, a Hartmann's type procedure would be preferable to simple oversewing. However, given his age, premorbid condition and the unfortunate complication, it probably would have made no difference to the eventual outcome of the situation. As no autopsy was carried out, the actual cause of death is not known.

Death After Cholecystectomy in a Frail and Elderly Patient

Summary

A frail 89 year old woman presented with a five day history of abdominal pain, vomiting and dyspnoea. She had been admitted for assessment some six months earlier for investigation of deterioration in mobility and function. Her medical history included breast carcinoma managed non-surgically, hypothyroidism and medical therapy for presumed congestive cardiac failure. At presentation, she was physiologically compromised with leucocytosis, metabolic acidosis and some respiratory failure.

A diagnosis of acute cholecystitis was promptly established by ultrasound despite a technically difficult study. Initial conservative management was unsuccessful. Given her frailty, it was not unreasonable to proceed to a percutaneous drainage of the gallbladder, and this was performed four days following presentation. Resolution of the sepsis was disappointing and she subsequently underwent open surgery approximately two weeks later. Open cholecystectomy and extraction of a common bile duct calculus was achieved in an operation spanning 3½ hours.

Within the immediate peri-operative period, large amounts of intravenous crystalloid were administered amounting to 6.5 litres on the operative day and up to 3.5 litres daily subsequently. During this period her urine output remained low, but there was no unexpected respiratory compromise. Peritoneal sepsis, operative blood loss, and drain losses reasonably explained the fluid requirement. The intravenous fluids were reduced

with resumption of diet, but a sudden deterioration in her condition occurred on the fourth post-operative day coinciding with a large vomit. A chest x-ray performed at this stage demonstrated some pulmonary collapse with small effusions, but a normal cardiac outline. This examination was supportive of the clinical diagnosis of aspiration pneumonia and effectively excluded cardiogenic pulmonary oedema.

Her subsequent course was of an inexorable decline and in consultation with her family, the services of the palliative care unit were employed. Her death approximately one week later was expected.

Comments

The poor outcome in this case can be readily attributed to poor physiological reserve in a frail elderly patient with significant peritoneal sepsis. Significant co-morbidity was present and I suspect this was underestimated due to her relatively good health up to six months preceding her final admission. There is no evidence that her fluid management was a contributing factor to her demise. I believe her management was well within reasonable expectations and the decision to allow death with dignity is to be commended.

Sudden Pre-Operative Death of an Elderly Hyponatraemic Woman with Right Colonic Carcinoma

Summary

This 87 year old woman was admitted electively for a right hemi-colectomy for a carcinoma of ascending colon diagnosed at colonoscopy performed for iron-deficiency anaemia. Her co-morbid factors included blindness, asthma, congestive heart failure, hypertension, atrial fibrillation and gastro-oesophageal reflux disease.

On admission, she was hyponatraemic (121 mmol/L) and hypochloraemic (86 mmol/L). Her serum potassium was normal. Consequently, surgery and bowel preparation were postponed and a general physician consulted. The electrolyte depletion was attributed to her frusemide 80mg bd which she took with potassium supplementation. The patient was infused 1L Hartmann's over 12 hours and 1L iso-osmolar sodium chloride infusion over 6 hours, and the diuretic ceased. Regular clinical review showed no exacerbation of heart failure and her serum sodium improved to 127 mmol/L. She was transfused with 2 units of packed red cells (each over 2 hours) in view of a Hb of 97 g/L.

The patient sustained a cardiac arrest with ventricular fibrillation on the fifth admission day and failed to respond to resuscitation. There was no clinical evidence of deterioration before this sudden event. There was no post-mortem, so it is unknown whether the cause of death was pulmonary embolism, a cardiac or neurological event.

Comments

I cannot be overly critical of the management of this lady as my management would have been similar and we don't know the mechanism of death. However, I would make the following points. I agree that the likely cause of hyponatraemia was the high-dose diuretic, but drug-induced inappropriate ADH (SIADH) is common (50%) and was not excluded by checking serum osmolality. Hypo- or hyper-magnesaemia can lead to arrhythmias and was not excluded. Although the total volume of fluid infusion was small, the rates (1L over 6 hours and 500 mL packed cells in 4 hours) were too fast for an elderly lady with CCF. Having said that, there was no clinical evidence of CCF and the saline infusion was several days before the arrest. The correction of serum sodium was not overly aggressive, but it seems the initial fluid regimen written by the resident (4 litres of Hartmann's over 24 hours!) was crossed out, indicating a revised decision to go carefully. There was no post-mortem, so the cause of death is unknown. Therefore, I would class this as a minor adverse event that probably made no difference to the outcome.

The documentation in the case notes and subsequent typed correspondence from the surgeon was adequate. Although not a criterion on your check-list, I would make comment that the handwritten account of the cause of death on the surgical proforma was largely illegible and it would have been better to ask for a typed account.

Addendum

I carried out a brief Medline search, as electrolyte imbalance and SIADH are common pre-operative problems in our elderly patient population. In by far the majority of cases, no adverse outcomes are recorded. However, severe hyponatraemia, hypomagnesaemia and hypokalaemia can cause fitting and arrthymias, but overly rapid correction can equally cause adverse events. Thiazide diuretics are usually implicated in hyponatraemia rather than loop diuretics such as frusemide. In asymptomatic hyponataemia in a patient with CCF, temporarily stopping the diuretic may resolve the problem; intravenous saline replacement should be reserved for severe or symptomatic hyponatraemia (< 115 mmol/L). Rapid saline replacement (> 20 mEq rise in serum Na+ in 24 hours) can produce fitting, central pontine myelinolysis and sudden death. "Duplex" saline should be rarely used.

As pre-operative electrolyte disturbance is so common and correct management a bit cloudy, does WAASM consider a proper literature review in conjunction with an endocrinologist or renal physician appropriate to draw up some guidelines? Has there been any other pre-operative sudden deaths associated with electrolyte disturbance recorded in the audit?

Prolonged Medical Therapy Contributed to Death in Severe Ulcerative Colitis

Summary

A 58yr old woman with a 20 year history of ulcerative proctitis presented to her gastroenterologist with deteriorating symptoms despite increasing doses of oral corticosteroids and olsalazine. She was admitted systemically unwell some two weeks later to a peripheral private hospital where severe colitis was confirmed endoscopically. High dose parenteral steroids were administered in association with oral/enema delivered steroids and mesalazine, with little clinical improvement. Ten days later, immunosuppression therapy with mercaptopurine was commenced, and this appeared to be of some initial benefit to her colitis. However, the mercaptopurine was ceased ten days later due to an induced leukopenia. Development of a high fever subsequently prompted transfer to a large metropolitan private hospital where care was assumed by a multidisciplinary team including a physician, gastroenterologist and surgeon.

Broad-spectrum antimicrobial therapy was commenced despite negative blood cultures, and within 24 hours of transfer a surgical decision to perform colectomy was made. Active colitis was still present despite the treatment to date, but not unreasonably a decision was made to defer surgery until the leukopenia was corrected. Unfortunately, respiratory failure occurred within four days resulting in admission to an ICU where she required ventilatory support.

Her subsequent course was that of a general decline associated with ARDS, punctuated by an emergency total colectomy six days after the ICU transfer. The development of multi-organ failure including worsening ARDS, coagulopathy due to DIC, an ischaemic CVA, and renal failure ended with her death eleven days post-operatively.

Comment

With the benefit of hindsight, the 'golden window of opportunity' to perform lifesaving colonic resection in this patient was missed. Arguably this window of opportunity was open prior to her assessment by a surgeon, and the unfortunate outcome was all but inevitable by the time of surgical review. I would like to make two observations about her initial treatment.

Firstly, the failure to respond to maximal conventional medical therapy over a period of 2-3 weeks in association with significant systemic toxicity is a reasonable indication for timely surgical intervention. Unfounded optimism concerning medical therapy that is failing can lead to a delay in appropriate surgical

management that ends in unnecessary mortality. Earlier surgical consultation in this case may have led to a much happier outcome.

Secondly, the use of immunosuppressive therapy in severe colitis that has failed to respond to conventional therapy is surely controversial and has in this case arguably led to the demise of the patient. The secondary leukopenia led to significant management difficulties, and placed the treating surgeon in a difficult position. It is my opinion that the immunosuppression closed the window of opportunity, rather than extended it.

With the remainder of her management I can find nothing to be critical of, and it is evident that the level of expertise and cooperation within the treating hospital was of the highest standard.

Splenectomy and Large Bowel Surgery Should be Separated Whenever Possible

Summary

The patient was an 85 year old male with many diagnoses. These were complex partial seizures, hypertension, chronic renal impairment, deafness, peripheral vascular disease, anaemia (either iron deficient or haemolytic), type II diabetes, arthritis and aortic valve disease. His notes support these diagnoses and describe the management of these conditions.

He was referred by his general practitioner with PR blood loss and earlier had been a patient of the haematologists and carried a diagnosis from them of Evans' syndrome to account for his anaemia and thrombocytopaenia. Colonoscopy identified an infiltrating sigmoid carcinoma at 18 centimetres from the anal verge. The colonoscopist recorded an intention to refer to the colorectal surgeons, but I cannot find any documentary evidence of discussion between the surgeon and the haematologist about elective splenectomy.

His first operation proceeded approximately one month later and high anterior resection was carried out for a Dukes B tumour, which would have given him a good prognosis. An elective splenectomy was also performed, its elective nature being confirmed by the consent form and the fact that he had been given pneumococcal vaccination. However, this had been given on the day of operation and therefore was of no use to him post-operatively to resist pneumococcal infection.

The patient's post-operative course was complicated by a large amount of blood loss from his drains requiring transfusion (with packed cells only), renal failure, mental confusion and atrial fibrillation.

Seven days after the first operation he had a laparotomy and loop ileostomy, performed but there was no anastomotic leak or pus collection although serous fluid had been found in the pelvis and cultured, yielding only a staphylococcus undetected by microscopy and sensitive to all antibiotics - not a life threatening organism. He returned to ICU after developing respiratory failure and after haemodialysis, inotropic support, ventilation and prostacyclin for pulmonary hypertension (aortic regurgitation?) as well as IV antibiotics, he had a cranial CT scan which showed multifocal infarcts and he was unresponsive. Respiratory support was withdrawn and he died 12 days later.

Comments

There had been a delay in diagnosis of this patient's sigmoid carcinoma (uninvestigated Fe deficiency anaemia in a male subject was first noted in 2000).

The evidence for a diagnosis of Evans' syndrome (combined auto immune haemolytic anaemia and auto immune thrombocytopaenia) was far from overwhelming, being based only on a positive result for direct Coombs' test and the presence of platelet antibodies. The red cells were not spherocytic and haemolysis had not been assessed by a serum haptoglobin level. Low ferritin was found (25 mg/L) previously and the diagnosis of iron deficiency anaemia had already been made (presumably due to blood loss from his

carcinoma). He also had renal impairment as a possible cause. The immunologist found that his classical and alternate haemolytic complement pathways were both low at 20% and 65% of normal.

With regard to the thrombocytopaenia, he had been taking sodium valproate for his seizures and quinine for cramps as well as clopidogrel, and these drugs can cause thrombocytopaenia as a side effect. The presence of a lupus type antibody as well could have confused the result of the platelet antibody test.

When the spleen was examined histologically, there were no changes of reactive follicular hyperplasia, ceroid histiocytosis, or extra medullary haemopoiesis, and plasmacytosis of the red pulp was not seen.

It seems therefore that the decision for splenectomy was in error as the patient had drug-induced thrombocytopaenia and an iron deficiency anaemia due to his sigmoid carcinoma, and that Evans' syndrome (for which splenectomy could be beneficial) was not the correct diagnosis.

Did the splenectomy contribute to the patient's death?

I think that it certainly did and should not have been done. In an 85 year old man with many co-morbidities and no adequate pre-operative vaccinations, it is a dangerous procedure and made further so by combination with large bowel surgery. However, the patient did have post-operative bleeding presumably replaced by "storage injured" packed cells and these cells contributed to multi-organ failure.

A better pathway would have been to perform the large bowel surgery and treat the anaemia and thrombocytopaenia (if they persisted and if proper evidence of their auto immune origin - Evans' syndrome - had been obtained) with safe doses of prednisolone and gammaglobulin to maintain platelets for say six months, and then undertake laparoscopic splenectomy if these measures had failed and it was certain that splenectomy would be beneficial.

Complication of Warfarin Therapy

Summary

A 79-year-old lady with significant comorbidity was admitted as an emergency to hospital with abdominal pain. She was investigated and a decision was made to operate on the presumptive diagnosis of ischaemic bowel and after discussion with her family. In fact, she had a retroperitoneal haematoma secondary to excessive warfarin therapy and died two days after laparotomy after withdrawal of support in consultation with her family.

This lady was on warfarin for atrial fibrillation. She had undergone a carotid endarterectomy earlier in 2003 following a TIA. The admission diagnosis list also included ischaemic heart disease, but it is not certain on what basis this was formed. There was comment made of end stage cardiac failure and she was in renal failure with a creatinine of 300.

She had undergone a colonoscopy about 2 months prior to this acute presentation because of similar pain and this has shown no major abnormality, and she had her appendix removed some time ago.

She was initially admitted under a physician who made arrangements for investigations including a CT scan and was reviewed by the surgeon prior to and after the CT scan on the day of admission. The final CAT scan report did suggest the possibility of haemorrhage or inflammation in the area around the right colon but the hand written notes on the day of admission suggested inflammation alone. In retrospect, there was evidence of a raised INR of greater than 5 several days prior to presentation and a haemoglobin of 97 the day prior to presentation combined with a haemoglobin of 88 after two units of blood were transfused on presentation. The low haemoglobin just prior to presentation was probably acute as there was a haemoglobin of 130 earlier in 2003. The patient was resuscitated and observed overnight for review the following day and as she had not improved and had ongoing pain with the possibility of gut ischaemia, and after discussion with the family, a decision was made to operate.

At operation the diagnosis of retroperitoneal haematoma affecting the right colon and mesentery of the small bowel was made. Her raised INR had been corrected by that stage.

After surgery a decision was made with the family not to resuscitate further. Initially she was ventilated and then removed and transferred to a palliative care unit where she died two days after surgery. The actual cause of death was respiratory failure.

Comments

I believe that although the possibility of retroperitoneal bleeding would have been considered, in a patient who has an uncertain diagnosis and where the patient and the family request surgery for possible treatment it is reasonable to proceed. This situation is often encountered where the family request surgery even though there is a low chance of a positive impact and, after surgery, withdraw request for further treatment.

Although the consultant who filled in the report felt that he would have done something differently, it is very difficult at the time in the presence of a family who want to proceed with treatment to advise them against this decision. If a definitive diagnosis could be accurately obtained prior to surgery then the management of this patient may have been different.

Therefore, from the evidence presented, I would have proceeded in the same fashion. It is all very well to retrospectively put the high INR combined with a recent low haemoglobin together with the CT scan into a definitive diagnosis of retroperitoneal haematoma, but equally there could have been a different diagnosis and possibly surgery could have been effective.

The only comment I would make on the record keeping is related to entries made by the surgical registrar and by the physician. The surgical registrar wrote notes on a date which was actually the wrong date. He also failed to put any patient label or write the patient's name at the top of the notes on either side of the record sheet.

There is also a wrong date entry by the physician. In an emergency situation such as this it is often difficult to adhere to such detail, but it does become obvious in retrospect.

I am a little critical of the decision to give the patient 160mg bd of gentamicin when antibiotics were started, rather than a stat dose and then the plan for a gentamicin blood level. The patient's creatinine was 300 and I cannot see an indication of weight.

Then there is the overall question of giving elderly patients warfarin for uncomplicated atrial fibrillation. I am aware of the figures that suggest that warfarin is beneficial in patients in chronic atrial fibrillation, but it appears in this elderly lady there was a great deal of difficulty in maintaining a stable INR. Records indicate her INR fluctuated between 1.5 and 5 in the weeks leading up to this acute admission. I believe this query of warfarin in elderly patients on atrial fibrillation needs to be redirected to the cardiologists and physicians.

On a very positive note, I think this lady was treated promptly on her emergency admission with adequate treatment commenced and arrangements made for a CT scan. She was reviewed by the surgeon within hours of presentation, which is often difficult to achieve in a non-teaching hospital.

Death After Surgery for Advanced Cancer

Summary

A 57 year old male underwent palliative gastrectomy at a private hospital. This was performed following a preceding endoscopy for upper GI bleeding which demonstrated the presence of a large cancer in the mid

portion of the gastric body which was ulcerated and actively bleeding. He also required transfusion of two units of blood at the time. A decision was made to offer the patient surgery due to the symptomatic nature of the cancer, which appears to be correct and quite an appropriate decision. He subsequently underwent palliative gastrectomy on the same day and made good post-operative recovery. Intra-operatively he was found to have extensive disease in the stomach with peritoneal seeding as well as extensive lymphadenopathy.

Post-operative the patient was offered palliative adjuvant chemotherapy which he accepted and therefore insertion of an Infusaport was performed two months later. He underwent chemotherapy with epirubicin, cisplatin and 5FU through a continuous infusion pump. The treatment was tolerated quite well until a few days prior to completing the treatment when he developed some redness and discomfort at the port site which later settled on antibiotics.

The patient managed quite well following completion of chemotherapy for several months. However, approximately six months later he developed symptoms and signs of incomplete small bowel obstruction. He was managed conservatively for some time until his symptoms progressed gradually to complete small bowel obstruction. This was judged to be due to recurrent disease. It appears that the patient was given a complete and honest explanation of his problem. However, at the time he requested an attempt to excise the recurrent disease to try to relieve the small bowel obstruction.

Endoscopy was performed the following month which showed recurrent disease at the site of the anastomosis. He also developed extensive malignant ascites. He underwent a limited laparotomy which confirmed extensive peritoneal disease - the ascites and the disease was judged to be inoperable. He made gradual recovery and was discharged from the hospital.

Unfortunately he deteriorated rapidly at home and died at home three weeks after his discharge from the hospital.

Comments

In conclusion, my view is that this is a sad but routine case of a patient with advanced gastric cancer presenting with bleeding which was managed quite appropriately, both surgically and in terms of adjuvant therapy. The decision to operate was quite appropriate in terms of timing as well as the choice of surgical procedures. The second operation was very limited, and again justifiable in view of the patient's wishes.

There is not much to add to the management and it appears that at all stages the management was appropriate and relevant to the patient's condition.

Vascular Surgery (1 case)

Death From Poor Peri-Operative Administration of Anticoagulation

Summary

A 75 year old man with stable ischaemia sustained minor trauma to his left leg. He was admitted to hospital for assessment. He was a significant 'arterio-path' and relevant medical conditions, apart from his known peripheral vascular disease, included diabetes and atrial fibrillation. He was taking amiodarone, digoxin and warfarin. A previous echocardiogram showed mild to moderate dysfunction of the left ventricle with inferior and septal hypokinesia. The left atrium was dilated.

On the day of admission the cardiologist requested that warfarin be withheld and that Clexane be administered, and 70mg of Clexane was given that night. The cardiologist reviewed the patient personally the following day and the INR that morning was noted to be 1.4. Although instructions were given to withhold the Clexane that day in case the patient went to theatre, Clexane 70mg was given twice that day. A second INR performed in the afternoon was 1.6.

On the second day following admission he underwent a lumbar sympathectomy. This appeared to proceed uneventfully. The Clexane was withheld in the morning prior to surgery, but 70mg was administered in the evening. An INR that morning was 1.6.

When reviewed on the first post-operative morning the patient appeared to be well. For reasons that are not clear from the notes the cardiologist made arrangements for him to have a Persantin Thallium scan the following day. Instructions were given to fast the patient, presumably from midnight. Clexane 70mg was administered twice that day, but no INR was measured.

In the early morning hours on the second post-operative day (some 40 hours after surgery) the patient was found on the floor. He was cold and clammy with a BSL of 17.1. The blood pressure then was 117/70. A few hours later, the BP was recorded as 90/60 and the BSL was 19.1. The patient advised the nurses that he had not taken his hypoglycaemics because he was starving for his scan.

During the same morning, a MERT call was placed. The patient had collapsed a second time and probably lost consciousness whilst moving to the shower. An ECG at that time showed rapid AF with inferior elevation of the ST segment. He was given amiodarone IV. A subsequent ECG showed resolution of the ST changes and a heart rate of 88, and still in AF. The patient was administered aspirin and clopidogel although the exact time they were given was not recorded. Sometime that afternoon he was moved into the Intensive Care Unit. Investigations on admission to ICU revealed a haemoglobin of 114. In the evening, a repeat haemoglobin was 77, he was hypotensive at 105/50 and there was fullness in the left flank in relation to the lumbar sympathectomy incision.

It appears that before anything could be done about this, the patient collapsed. Hypovolemia was felt to be the principle cause of the problem and in the absence of cross-matched blood, he was given O negative blood. Despite ongoing resuscitation by appropriate senior staff the patient died. Blood taken at the time of resuscitation revealed an INR of 4 and AAPT of 88.

Notes made after the patient's death by the attending anaesthetist and a nurse both suggest that hypovolaemia secondary to a post-operative bleed was the initiating event for the arrest. The Coroner's Office was notified of the death, but chose not to carry out a post-mortem.

Comments

This gentleman died of post-operative hypovolaemic shock less than 48 hours following surgery. I am surprised the Coroner did not review this case. The peri-operative anti-coagulation regime must have played an important part. His pre-operative INR was 1.4 to 1.6 which is higher than optimal for elective surgery. Despite these high levels, no post-operative INR was measured until his terminal collapse, so the increase to an INR of 4 following surgery went unnoticed. In the absence of further warfarin this rise may have been secondary to a synergistic effect of the flucloxacillin started 24 hours prior to surgery.

The effects of the high INR were undoubtedly compounded by the administration of Clexane 70mg bd during the 36 hours prior to surgery and for 48 hours post-operatively. This is a high dose for a patient undergoing surgery. Although the notes clearly document that the Clexane was to be withheld on the day prior to surgery, it was not crossed off the drug chart, and was therefore administered.

Following his collapse he was administered both aspirin and clopidogel. These appear to have been given without regard to the impact this might have on a patient who had surgery only 36 hours earlier.

It is difficult to escape the conclusion that his anticoagulation regime could have been managed better. This was almost certainly the cause of the post-operative bleed that caused his death. The principle failure appears to have been a lack of communication between the cardiologist and surgeon.