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CASE NOTE REVIEW BOOKLET

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Acute Cholecystitis

An elderly patient presented to a district general hospital four days following discharge after shoulder surgery. There was a background of NIDDM, hypertension, and asthma requiring steroids, reflux and a previous sigmoid colectomy for diverticulitis. The patient had generalised abdominal tenderness and a fever of 39.3 with a tachycardia of 100/min. A CT scan revealed an impacted stone in the neck of the gallbladder with marked distension, but no free fluid. IV antibiotics were commenced and the patient transferred to a tertiary hospital.

On arrival the antibiotics were ceased and the patient observed for seven days during which the pain seemed to be intermittent. The fever persisted and the patient's condition seemed to gradually deteriorate. The antibiotics were re-started two days after they had been ceased. The patient spoke no English so it was difficult to confirm the level of discomfort.

At laparoscopic cholecystectomy seven days after presentation the gallbladder was perforated with omental wrapping. Calot's triangle was friable. Diathermy and Flo-seal seal were used for haemostasis, and a 19F Blake's drain placed. Noradrenaline was required intra-operatively and the patient was then ventilated in ICU. The poor renal function was treated with noradrenaline and frusemide. There was significant on-going blood loss (Hb dropped to 58 despite nine units packed cells transfusion), which was treated conservatively.

Whilst being turned on the fifth day in ICU there was an accidental extubation with significant desaturation and aspiration. This was recovered and elective extubation occurred two days later. Transfer to the ward was the following day at 23:45 on a Saturday night. The IV access consisted of a femoral line that had been placed soon after surgery. This remained in use for a total of two weeks. The antibiotics were ceased over the weekend, but started two days later. The patient was noted to be drowsy, but distressed on the Monday morning (nine days postoperatively) and the fevers returned, with delirium and oliguria. A CT revealed a 12 cm sub hepatic collection with gas locules. Although the drain was not draining, it looked to be in a good position on the CT.

Delirium and fevers persisted for a further week before microbiology was consulted. The sub hepatic collection was radiologically drained 18 days postoperatively when TPN was also commenced. Following further deteriorated further three days later it was agreed that aggressive resuscitation was not warranted.

Comments

It can be difficult to assess pain in patients who do not speak English. The surgical notes recorded the abdomen was soft and non-tender and with no pain, but the nursing notes recorded pain while pointing to the right upper quadrant.

Flo-seal has limitations. Also the extent of bleeding can be underestimated in laparoscopic surgery due to the head-up position. A return to theatre for haemostasis and washout - whether open or laparoscopic - is unlikely to impede recovery in the first few days postoperatively, but may have been effective in preventing the ongoing slow blood loss and the sepsis, which was potentiated by the rich intra-peritoneal culture medium.

Although the plan for discharge from ICU was well telegraphed before the weekend, a transfer at midnight on Saturday night to an understaffed surgical ward without appropriate IV access ought to be preventable. This is an area of significant concern and staffing levels ought to reflect clinical needs, whether in the ICU or in a step-down unit.

It is regrettable that TPN was instituted just two days before the arrest and a decision not to pursue active resuscitation. The postoperative care after discharge from ICU reads as though there was little senior surgical input.

Malignant Bowel Obstruction

A young patient was admitted under the care of a General Physician with a unilateral DVT. By virtue of mild intellectual impairment

there was a poor history, but there was clear 12-month history of abdominal pain, recurrent urinary tract infections and recent pneumaturia. On examination the patient was cachectic, anaemic (Hb = 50, iron deficiency picture) and a MSU confirmed a UTI.

The patient was anti-coagulated and transfused of four units of packed cells. An upper gastro-intestinal endoscopy was performed, but failed to identify the cause of anaemia.

In the early stages of the admission abdominal distension was a prominent feature. On the second day an abdominal x-ray identified distension of both large and small bowel. A colonoscopy was booked for the following day, but cancelled and not formally re-booked.

On day five an abdominal ultrasound scan identified ascites and on day six a General Surgical team was asked to review the patient. The General Surgical Registrar ordered a CT scan that was performed the following day. However, the Registrar did not follow-up on the result. The abdominal CT scan identified enlarged iliac lymph nodes, sigmoid thickening in two segments with dilatation of both small and large bowel.

The CT scan was reviewed for this report and shows a clear-cut sigmoid carcinoma with apple core constriction producing large bowel obstruction. In addition, there was obvious small bowel obstruction with decompressed loops of small bowel evident on the films as well as a probable point of obstruction due to adherence of the small bowel to the main tumour mass.

A flexible sigmoidoscopy performed on day nine confirmed the presence of a recto-sigmoid carcinoma. A new surgical team was consulted, an IVC filter was inserted the following day (day 10) and the patient proceeded to laparotomy on day 11.

At surgery two colonic tumours were confirmed. A mobile, proximal sigmoid lesion was identified as well as a locally advanced, recto-sigmoid cancer that invaded the bladder and the left pelvic side wall. The surgeon opted for a Hartmann's resection that involved an extensive bladder resection but clearly did not achieve clear surgical margins. The patient subsequently required re-operation because of urinary leak, developed progressive fungal sepsis and died on day 23 of this admission.

Histopathological analysis confirmed extensive lymph node involvement by tumour and involved surgical margins.

Comment

Notwithstanding the patient's young age the combination of his abdominal pain, colonic distension, recurrent urinary tract infections, pneumaturia, iron deficiency anaemia and cachexia should have pointed the managing clinical team and the first surgical team to the diagnosis of colorectal cancer.

Despite the patient's debilitated state, flexible sigmoidoscopy at the very least should have been undertaken as a matter of much higher priority than upper gastro-intestinal endoscopy or even abdominal ultrasound. Matters were not helped by the under-reporting of the abdominal CT scan that quite clearly demonstrated a sigmoid cancer.

Having ordered the CT scan, the Surgical Registrar should have not only reviewed the result but should have acted upon it. It was left to the admitting clinical team to organize the flexible sigmoidoscopy and to involve a second surgical team in this patient's ongoing management.

Given the patient's very poor general medical condition and the clearly advanced local stage of the recto-sigmoid cancer a much less ambitious intra-operative strategy would have been better. On this occasion a defunctioning stoma to allow neo-adjuvant chemo-radiotherapy and later surgical resection would have been a safer strategy.

This patient was admitted late in the course of his overall illness and the most significant delay in diagnosis actually preceded his admission. Just the same a much more prompt progress to diagnosis by CT scan and flexible sigmoidoscopy and a less ambitious intra-operative strategy might well have seen him survive this presentation even if not see him ultimately cured.

Delay In Surgery For Colonic Bleeding

An elderly patient had mild Parkinson's disease and was hard of hearing, but living alone independently, was admitted at 15:30 hours after large per rectal bleed with syncope and hypotension. The haemoglobin was 86. Three units packed cells were given.

Some five and half hours later (20:00 hours) a colonoscopy showed active sigmoid diverticular bleeding. Two more units packed cells were given. During the early hours of the following morning (about 12 hours after admission) there was further rectal bleeding with fresh blood and clots, each estimated at 300+ ml.

Later that morning (11:30, 18 hours after admission) a Hartmann's resection was performed. Three more units of packed cells were required. Recovery in ICU was unremarkable and 24 hours later the patient was transferred to the ward.

The next morning, 48 hours after surgery the patient was sitting in a chair, walking with the physiotherapist and using the PCA appropriately.

The next morning, 72 hours after surgery the patient was found vomiting and then became unresponsive. The patient was intubated and returned to ICU, but died six hours later.

Comment

This patient was actively bleeding at the time of colonoscopy having had a significant bleed before coming to hospital. The patient continued to bleed significantly early the next morning. By the time of the operation the patient needed eight units of packed cells and two units of FFP.

I believe earlier surgical intervention could have been performed. However, I doubt that this would have made a difference to the post-operative course. The patient seemed to recover well, but developed respiratory complications presumably from aspiration of the vomitus.

Haematemesis and Melena I - Teams Should Exist in all Tertiary Referral Hospitals.

An elderly patient was admitted to a regional hospital with haematemesis and melena. Co-morbidities included severe COPD (on daily steroids and puffers) and IHD (AMI six years earlier). When endoscopic attempts fail to control the bleeding the patient proceeded to an emergency laparotomy.

Operative findings were of a massive posterior duodenal ulcer (DU) with an active arterial bleeder at the base that was managed by oversewing of the bleeding point and pyloroplasty. Post-operatively the patient failed extubation and was transferred to a tertiary hospital ICU. Soon after arrival to the ICU the patient was extubated and transferred to a general ward the next day. Over the following the days the patient received two MET calls and investigations show a non-STEMI and a drop in haemoglobin from 110 to 70.

An urgent gastroscopy (five days post initial laparotomy and oversewing of DU) showed an actively bleeding DU that could not be controlled endoscopically. In view of the high anaesthetic risk, angiographic embolisation was undertaken. This showed a small bleeding aneurysm at the tip of the gastroduodenal artery. The artery was coiled, but overnight the patient continued to bleed, leading to a second emergency laparotomy. The operation, undertaken by a trainee surgeon, commenced at 00:30hrs and was completed at 02:15hrs. The previous duodenotomy was re-opened and the coils found in the base of a large duodenal cap ulcer that was bleeding. The duodenotomy was extended and the bleeding controlled with large 2/0 prolene sutures at 4 corners. The duodenum was then repaired and an omental patch applied.

Post-operatively the patient was managed in ICU for three days and then transferred to the general ward. Thereafter there was a steady recovery until day 12 post-operatively when more episodes of melena are noted and the haemoglobin fell to 76. A PPI infusion was commenced with a transfusion of six units of packed RBCs. The patient's haemoglobin

subsequently remained stable with no further transfusions, and the passage of normal stools until eventual discharge/transfer (29 days after the redo-operation) to a small local rural hospital for ongoing convalescence.

Two weeks after discharge the patient complained of abdominal pain and had three coffee-ground vomits and was transferred to a regional hospital. A CT scan shows proximal small bowel obstruction (SBO) with gross small bowel dilatation. The haemoglobin was normal and a gastroscopy showed no evidence of bleeding, but faeculant fluid was noted in the stomach. The patient was managed conservatively and after three days was transferred to a tertiary referral hospital for further management for a non-resolving SBO. At the tertiary hospital a surgical team reviewed the patient and a plan made for a laparotomy the next day. However, overnight the SBO resolved spontaneously. Over the next few days the respiratory status deteriorated. A CT pulmonary angiogram shows centrilobular emphysema, early bilateral pneumonia and a cavitating lesion in the lung. Six days post-admission frank blood and clots were passed per-rectum and the haemoglobin fell by two grams. An urgent colonoscopy and gastroscopy was declined due to the high anaesthetic risk and likely low yield. It was thought this last presentation was secondary to ischaemic colitis. The patient died 19 days after the re-admission.

Comment

The first line assessor has raised two issues:-

1) A laparotomy and oversew of a DU was performed six days after the first operation failed. Was the repeating of the oversewing technique adequate in the second operation, or should the gastroduodenal artery been ligated above and below the duodenum? While the wisdom of repeating the same failed operative technique is a valid point, in this case it was successful and the patient was eventually discharged. Post-operative ongoing melena was successfully managed with high dose PPI infusion. There was no follow-up gastroscopy.
2) Was the surgeon at the second operation experienced enough or should a more experienced surgeon been called? Given that a re-bleed occurred six days after previous surgery it would have been prudent to consult an upper GIT specialist soon after admission. In this case it would not have affected the final outcome. There was plenty of time during day-light hours for such consultation. The eventual hour of the re-do surgery was in the middle of the night, which is less conducive for seeking opinions from more experienced colleagues.

Haematemesis and Melena II - A Case for a Haematemesis and Melena Teams

A previously well and independent elderly patient was urgently transferred by RFDS to a tertiary referral hospital following a sudden collapse earlier that day and epigastric pain. Prior to transfer, the haemoglobin at the rural hospital showed a drop from 140 (recorded several months before) to 85. One unit of packed cells was commenced. A presumptive diagnosis of a leaking abdominal aortic aneurysm was made.

During transfer by the RFDS the patient was haemodynamically stable and no further blood transfusion was given. At the tertiary referral hospital, surgical review of the patient by the overnight surgical registrar revealed a recent history of several episodes of melena. Vital signs, abdominal examination and PR were all normal, with no blood, melena or masses noted. Admission blood tests showed an Hb of 101, creatinine of 157 and an elevated troponin of 0.11, but no ECG changes to suggest ischaemia or infarction. Two hours later the same surgical registrar witnessed the passage of about 500mls of melena, but no fresh blood or clots. The patient was assessed as having had a large upper GIT bleed, but presently haemodynamically stable. Also noted was a NSTEMI due to hypotension. The registrar suggested the patient was not for surgical admission and was to be referred to the on-call gastroenterology team, given an IV infusion of esomeprazole and in view of the risk of bleeding anticoagulant medication for

the NSTEMI was to be withheld.

At 11:30hrs the next day the patient underwent a gastroscopy by the gastroenterology registrar, where an 'ulcer of the second part of the duodenum is noted with high risk features' and 'a large D2 ulcer with a visible vessel was injected with adrenaline and gold probed with good effect'. The suggested management plan was for 're-scoping if re-bleeds'. The endoscopy report noted a 'moderate risk of re-bleed'.

Post-endoscopy the patient was transferred to a general ward at 13:15hrs. Seven hours later the patient deteriorated with a BP of *77/52* and a MET call was made. Notes by the on-call surgical registrar indicate that after discussion with the ICU consultant that 'will not accept the patient, even intubated', post any procedure but 'may consider a HDU bed'. There was also discussion with the on-call general surgical consultant that concluded 'the patient will not be a candidate for laparotomy given advanced age, multiple co-morbidities, deteriorating condition and no ICU support'.

At 22:30hrs the patient was transferred to theatre for an endoscopy by the gastroenterology registrar. This took two hours. The surgical registrar and interventional radiology consultant were also in attendance. A large visible spurting vessel at the base of a posterior duodenal ulcer was seen, but not able to be controlled by adrenaline injection or haemoclips. Post-endoscopy the plan was for transfer to the radiology department for embolisation. However, the patient was haemodynamically unstable throughout the procedure and the anaesthetic consultant noted that the patient was moribund.

After discussion with the family it was agreed to cease inotropic support and high flow oxygen. The patient succumbed at 01:30hrs.

Comment:

This elderly patient was fit and independent with no significant co-morbidities. The medical record notes the patient lived in a retirement village, was independent of all ADLs, mobile indoors, ventured outside on his gopher and received community supports. The acute presentation in the patient had all the hall-marks of a major upper GIT bleed, with a history of collapse and drop in Hb. The first endoscopy identified a duodenal ulcer with high-risk features and a visible bleeding vessel. Given the patient's age and previous big bleed the question that must be asked is whether the plan for re-bleed should have considered surgical intervention, rather than re-scoping as the primary management plan.

When the patient did re-bleed (massively) the decision to transfer the patient to the operating theatre was correct. However, given the haemodynamic instability, the patient should have proceeded to an immediate laparotomy and over sewing of the bleeding ulcer rather than a two hour gastroscopy, which failed to control the bleeding. The plan for later embolisation (which often takes hours) was inappropriate in such a sick patient.

This case highlights the importance of having a multi-disciplinary haematemesis and melena team (gastroenterologist and surgeon) in a tertiary referral hospital setting, one that would allow for a better management in such high-risk cases. The transfer of the patient to a general ward and not an HDU after the first endoscopy showing a bleeding ulcer with high-risk features clearly demonstrates a lack of adequate system protocols which would be present if such a team existed.

Whilst many cases of upper GIT bleeding are successfully controlled endoscopically this case clearly illustrates the importance of a multi-disciplinary team approach and the need for protocols for managing upper GIT bleeding.

ERCP May Have Been a Better Option.

A frail elderly patient with severe recurrent biliary colic was a 'day of admission' case for a cholecystectomy. The operative risk was very high due to significant co-morbidities included stage II breast cancer, ischaemic heat disease with stable angina, early dementia, and hypothyroidism. This was discussed with the patient and their family. The preoperative abdominal ultrasound result was not available in the notes, but purportedly demonstrated a

large gallbladder calculus, without evidence of biliary obstruction. Liver function tests performed the day before surgery demonstrated significant elevation of all of the hepatocellular enzymes, but a normal bilirubin level.

Laparoscopic cholecystectomy was difficult due to a calculus that obscured the hepatobiliary anatomy. The dissected anatomy was defined by operative cholangiography. This demonstrated high-grade obstruction of the common bile duct due to an impacted 7mm calculus. Management advice was readily obtained intra-operatively from an available upper-gastoenterological surgeon. The cholecystectomy was completed laparoscopically and an urgent ERCP arranged.

On the first postoperative day the patient had a sudden syncopal episode and an elevated troponin I level in keeping with a peri-operative myocardial infarct. The patient was transferred to CCU and since otherwise well the planned ERCP delayed. Intravenous antibiotics were commenced for treatment of a concomitant urinary tract infection. On the second postoperative day an elevated lipase level was found in the absence of any significant abdominal tenderness.

ERCP was performed on the third postoperative day and was normal apart from mild biliary dilation. A sphincterotomy was performed. There were no obvious complications following the procedure. The next day the patient suddenly became hypotensive and confused. Multi organ failure rapidly ensued, and in consultation with the family, active treatment was withdrawn. The patient died several hours later. The cause of death was thought to have been due to pancreatitis, or possibly biliary sepsis.

Comment

It is hard to be critical of the decision to operate at all on this patient given the distressing symptoms and the appropriate consultative process. My only slight reservation about the management of this patient was that little apparent weight was given to the significantly deranged liver function preoperatively. In view of the high operative risk a pre-operative MRCP would have been justified and this would have mandated ERCP pre-operatively thus potentially avoiding surgery. Having stated this, it is likely that the outcome would have been the same in this case since there was evidence of pancreatitis *prior* to the ERCP. Confronted by biliary obstruction at surgery, the decision to proceed to therapeutic ERCP rather than bile duct exploration was the correct one and the outcome was largely determined by the severe co-morbidity of this patient

Surgery is the Preferred Treatment for Boerhaave's Syndrome

An elderly patient was admitted to a major teaching hospital with symptoms of chest and epigastric pain following a vomit. The patient had chronic airway disease, Parkinson’s disease and paroxysmal atrial fibrillation. A CT pulmonary angiogram was performed to exclude pulmonary embolism, but unexpectedly reveal Boerhaave’s syndrome with gas in the mediastinum.

The cardiothoracic registrar admitted the patient after discussion with the consultant. It was decided not to place an intercostal drain. A swallow and x-ray were performed the next day showed no evidence of an extraluminal leak. The patient was treated with intravenous antibiotics and kept nil by mouth. Warfarin for atrial fibrillation was ceased, presumably in anticipation of a possible operation. The CRP was 150 on day one.

The patient was given ice to suck and allowed clear fluids the day after admission. On day three a repeat chest x-ray showed a minimal amount of gas in the right and left paratracheal spaces. On the same day the patient complained of chest pain thought to be secondary to the pneumomediastinum. On the fourth day the patient reported more chest pain and feeling hot. A low grade temperature was recorded. The patient was commenced on a soft diet. The low grade pyrexia persisted. On day five the patient was given a full diet and was discharged without any further antibiotics. This despite a low grade

temperature, chest pain, a rising white cell and neutrophil count and a CRP of 320 on day 3. The patient was asked to contact the GP or the registrar if pain developed again.

Four days later the patient was re-admitted to another teaching hospital under the general surgeons. The patient was unable to keep food down, had chest pain, vomiting shortly after meals and had a haematemesis. The haemoglobin was 78 and WBC 20,000. A CT scan showed a large multiloculated para-oesophageal or mediastinal collection with a large right pleural effusion. A chest drain was inserted, antibiotic commenced and the patient was kept nil by mouth. Four days after this re-admission, and after a drop in haemoglobin to 57, an oesophageal stent was inserted. The patient was admitted to the ICU and ventilated, but rapidly developed multiple organ failure and died in ICU almost a month after admission.

Comment

This patient was not offered the conventional, standard management that would be expected for Boerhaave's syndrome. A few issues need to be addressed.

Boerhaave's syndrome is a life threatening surgical condition with an overall mortality of approximately 35% and a morbidity of approximately 55% that in most cases is due to contamination of the mediastinum combined with a delayed diagnosis. The high mortality rate (up to 70%) associated with conservative treatment makes surgical intervention the usual recommendation. Surgery includes simple repair with an autologous pleural flap or pedicled muscle flap from the intercostal muscles, chest wall musculature, diaphragm, or a mobilised pedicle of omentum, exclusion or diversion of the oesophagus or an oesophagectomy with or without reconstruction. Mortality rises dramatically with increasing delay to surgery. Primary repair of the ruptured oesophagus is the first consideration if the patient's condition is suitable and generally has a good outcome. Boerhaave's syndrome requires at least an urgent thoracotomy and laparotomy for both for pleuromediastinal debridement and adequate drainage.

Non operative management is very controversial and only recommended by some in stable patients with a well contained leakage and without any evidence of sepsis. Surgical intervention is necessary if any sign or symptom of sepsis is noted.

A negative contrast study does not mean that there is no perforation and in this case the management was apparently based on the fact that an active leak was not demonstrated. The patient had gas in the mediastinum initially which was sufficient evidence of a perforation. The later development of a rising white cell count, chest pain, a low grade temperature and a high CRP count all pointed to developing sepsis.

The patient should have been operated on as soon as possible with at the least a debridement and adequate drainage. There was no note about consideration of surgery in the file. Aggressive reversal of his warfarin and an early operation offered the only chance of survival.

'Aggressive conservative' management has been advocated by few but there is no convincing evidence of a better or at least equal outcome compared to the standard surgical approach. This patient was not even treated in an 'aggressive conservative' manner and the re-introduction of fluids within 24 hours and a full diet with discharge five days after a confirmed perforation seems was a high risk strategy. The patient should at least have had an adequate time of drainage and TPN to give the oesophageal perforation time to heal.

The patient represented to another major teaching hospital with signs of multiloculated mediastinal sepsis and a large pleural effusion. The patient was clearly septic and, although the presentation was now very late and the anticipated mortality much higher, there were clear indications for a thoracotomy and proper control of the sepsis with debridement, opening of infected planes and adequate drainage. Surgery to correct the perforation was clearly too late at this stage and I am not sure if a stent was even indicated. Percutaneous placement of an intercostal drain in a patient with loculated mediastinal sepsis was clearly inadequate as

evidenced by the fact that a second drain had to be inserted later.

The choice of a non-surgical approach for this patient unfortunately left them with a predictable mortality. Boerhaave's syndrome patients often have a deceptively mild initial presentation, but surgeons should be aware of the dismal outcome of a non-surgical approach.

Editorial note.

This is one of two patients with Boerhaave's syndrome that WAASM reviewed in 2009. The second case was also managed without an operation and died. The (different) assessor was equally critical of the conservative management and also expressed a firm view that early surgery was the strongly preferred option.

DVT and PE I - Multiple Failures Lead to Death

A middle aged patient holidaying in the country sustained an eversion injury of the right ankle. Three hours (14:50) post injury the patient arrived at local hospital A by private transport. The treating doctor felt a fracture was likely, but as no X-Ray facilities were available the patient was transferred by RFDS to regional hospital B arriving at approximately 22:00 hours.

An X-ray revealed a tibial fracture. The anaesthetist noted the patient was 105 kg, but otherwise fit. After discussion with the surgeon on call the Emergency DMO performed an MUA at 23:20 hours. Subsequently the patient was admitted to the ward under the care of the surgeon awaiting transfer to Perth for assessment at a teaching hospital injury clinic.

The patient remained in the regional hospital B for a further three days awaiting transfer to Perth. Four days after returning to Perth the patient saw a private orthopaedic surgeon who determined the bone alignment was adequate. The public hospital referral was thus cancelled.

Two days later (17:00 hours) the patient presented to the Emergency Department of hospital C complaining of a swelling above the knee. The treating doctor diagnosed thrombophlebitis and felt a DVT was unlikely. However, an ultrasound was arranged for the following day. No clexane was prescribed and the ultrasound does not appear to have been performed.

Two days later the patient collapsed at home. An ambulance was called. On arrival the patient was asystolic and CPR was commenced. The patient was transferred to local hospital C but was pronounced dead 90 minutes later. This was 10 days post injury.

An autopsy revealed bilateral pulmonary emboli and a large saddle embolus as the cause of death.

Comment:

The importance of DVT prophylaxis has previously been highlighted by WAASM. There would be very few surgeons who would not have a safeguard in place to ensure the appropriate DVT prophylaxis of all patients they operate on. This case however highlights areas that are often overlooked – for example, minor procedures by non surgeons and continuation of prophylaxis post discharge.

The current NHMRC Clinical Practice Guidelines list lower limb immobilization as a significant risk factor for the development of a VTE. The Cochrane Database systemic review in 2008 concluded that LMWH significantly lowered the rate of symptomatic and proximal DVT in such cases. Other significant patient risk factors in this case are age greater than 40 and obesity. Finally there were other risk factors of prolonged immobilization, three days of bed rest and substantial plane travel.

This patient had substantial risk factors for a DVT and in my view should have received chemoprophylaxis. The responsibility lies with the hospital organisations that should have policies in place to capture a patient's DVT risk and ensure that they are managed appropriately. Such policies should include the use of the Surgical Safety Checklist endorsed by the WHO and RACS, which would ensure DVT prophylaxis is at least thought of. No patient should be able to undergo a procedure in theatre without this

occurring and if such a policy existed there is no record in the notes of it having occurred.

The treating doctor was not a surgeon and may not have been aware of theatre protocol. This highlights our responsibility to educate our colleagues for the need for prophylaxis. The admitting surgeon and team reviewed the patient twice and did not consider DVT prophylaxis. Considering that all patients in remote parts of our state have significant distances to travel to arrive in Perth that will necessitate periods of immobilization DVT prophylaxis should be part of the transfer paperwork.

There is a substantial body of work that has studied the long increased risk of VTE after surgery. VTE prophylaxis may need to continue after discharge and the NHMRC guidelines recommend 7 to 10 days post cancer surgery. In patients with lower limb plasters the guidelines recommend prophylaxis for the entire period of immobolisation.

Regardless of the physical findings this patient should have been managed at hospital C as a DVT until excluded by an ultrasound.

In summary, it appears there were substantial failures at many levels in this patient care. All may have contributed to this patient's death.

DVT and PE II – Proper Prophylaxis Not Given.

An elderly patient died from a fatal pulmonary embolus 12 days after a radical cystectomy and right nephroureterectomy with ileal conduit formation. There was always at least a moderate risk of peri operative death as the patient has a pre-existing co-morbidities of ischaemic heart disease and renal impairment (ASA 3) as well as being of advanced age.

The patient was at high risk of postoperative DVT/PE and could probably have received more aggressive prophylaxis. One month prior to the operation the patient had undergone a transurethral bladder tumour resection and insertion of ureteric stents in a private hospital. This procedure was covered by s/c heparin for 72 hours. The notes provided are a little sketchy but it would seem the patient had difficulty walking after that operation (unstated reasons) and did not leave hospital between that operation and the cystectomy. It is unclear as to whether the patient received ongoing heparin during that time

On the day before the cystectomy the RMO's admission notes state the patient had a past history of DVT and PE. This was not recorded at the pre-admission clinic, nor by the Consultant Anaesthetist at the same clinic nor on the surgeon's admission/consent form.

The patient received s/c heparin 5,000 units the night before the cystectomy, but no heparin at all on the day of surgery. Calf-compressions was used during the operation and for the first 24 hours. Thereafter, the patient wore TED stockings and received 5,000 units of s/c heparin twice daily until death. Post-operatively, the patient had a prolonged ileus requiring TPN support. The physiotherapists clearly had considerable problems mobilising the patient, partly due to his clinical condition and partly due to his attitude, which was stated to be aggressive at times.

Comment

This patient was at considerable risk of DVT/PE. Yet, for unstated reasons, did not receive heparin on the day of surgery, these doses arguably being the most important. Consideration could have been given to more aggressive prophylaxis both pre and post operatively e.g. Clexane 40mg s/c daily or even a higher dose.

DVT and PE III - was DVT/PE prophylaxis adequate?

A young fit, non smoker presented as an emergency with a three day history of lower abdominal pain. At that stage the patient was unwell, but stable with a pulse rate of 110, normal blood pressure and a temperature of 38.3. The clinical diagnosis was peritonitis and plain films showed free gas under the diaphragm. After fluid resuscitation and antibiotics the patient underwent a three hour operation when a Hartmann's procedure was

performed. A Meckel's diverticulum was removed at the same time as an incidental finding.

There is no mention in the notes about pre or intra-operative DVT prophylaxis. The notes show TED stockings and administration of Heparin were written up, but only starting at nine o'clock the following morning. The patient did then receive post-operative Heparin 5,000 units bd and TED stockings.

The post-operative course was unremarkable and the patient discharged, apparently well, on day 10. Later that evening the patient was re-admitted as a Death on Arrival. The reporting surgeon states this was due to an iliac thrombus causing a fatal pulmonary embolism. I assume this was on the basis of post-mortem. I have not seen the report of that post-mortem.

The main discussion point clearly is the venous thrombo-embolism and whether anything could or should have been done further. My comments are based on the background of the Fourth Edition of the 'Best Practice Guidelines of Australia and New Zealand prevention of Venous Thrombo-embolism' produced by the Australian and New Zealand Working Party on the Management and Prevention of Venous Thrombo- embolism. Using this as a template this patient was of moderate risk only. The patient was a non smoker, not overweight, without pre-existing malignant disease and other risk factors. Against this measure the post-operative DVT prophylaxis was prophylaxis was probably appropriate.

However, there is no record in the notes that heparin was given either pre- or intra-operatively. In the absence of any entry the assumption has to be that it was not. This is when it should have started. There is also no record that that states whether TED stockings or compression pumps were used pre- or intra-operatively. If such devices or agents are used this should be documented.

Post-discharge prophylaxis is currently an area subject for great discussion. Nevertheless is it important to be cautious with early discharge patients as they may still be a risk and may need continued prophylaxis during the convalescence.

Editorial note

WAASM has previously noted that the use of TED stockings and calf compression, either intra-or post-operatively, is not recorded consistently. In particular, there is no way of knowing (as in this report) if no record means compression techniques were not used, or they were used but their application not recorded.

Doctors Prolong the Inevitable.

An elderly patient was admitted to a regional hospital with recurrent right foot cellulitis and ascities. Medical co-morbidities include NIDDM, chronic renal impairment, pulmonary hypertension, a previous TVR and pacemaker. The ascities was drained once every 5 to 6 weeks.

The patient had been admitted with an exact similar abscess 22 days earlier. It had been debrided under local anaesthetic (LA).

The notes of the admitting examination do not include a vascular examination and there is no entry regarding pedal pulses. The foot was drained/debrided on the ward under LA that night. The underlying bone was noted to be involved. The next day the ascites was tapped and over the next few days 8.5 litres drained.

The patient was treated with antibiotics and wound dressings over the next two weeks. During this time a substantial left pleural effusion was found, but for the surgeons were not keen to insert a chest drain. This may have been related to the fact that the patient was on warfarin.

There is a note 15 days after admission that the wound had further necrotic tissue. Three days later the renal function was deteriorating, probably secondary to the hypotension which is turn was secondary to sepsis. The renal function was discussed with a teaching hospital renal unit. A conversation was then had with the same teaching hospital ICU who felt the patient was not a suitable for ICU admission. On the same day the effusion was drained. Later in the same day the patient was

noted to be anuric. Another conversation was then held with a second, different teaching hospital that agreed to accept the patient. A transfer was arranged with RFDS for that night. An RFDS doctor reviewed the patient prior to transfer and determined that the patient would not survive regardless of treatment. After discussion with the patient it was agreed to introduce symptomatic treatment in the peripheral hospital. The patient died 48 hours later.

Comment

I have no major problem with the clinical care of this patient. I do think the decision making should have been faster and different. The chances of survival were very poor from the outset. If the hospital felt they could not cope then the patient should have been transferred shortly after admission. However, it was elected to treat locally, but the process was very slow. For example, the warfarin could have been reversed and the chest drain inserted earlier. The patient's health slowly deteriorated and it seems that 15 days after admission was pre-terminal and three days later terminal. This should have been accepted and terminal care introduced. Why arrangements were made to transfer the patient to Perth is not clear to me. To have the patient 'rejected' (in my view appropriately) by one teaching hospital, only for the local doctors to try later the same day to transfer the patient to another, different teaching hospital seems very odd. Fortunately the RFDS doctor appreciated the inevitable and an unnecessary transfer avoided. The principal doctors should have accepted the inevitable outcome several days earlier and made the patient's last few days more comfortable.

A Missed Strangulated Femoral Hernia

An elderly patient presented to an Emergency Department (ED) with recent history of abdominal pain and tenderness on the right side of the abdomen. The patient lived alone, had a background of left ventricular hypertrophy, atrial fibrillation and on Warfarin (INR on admission was 4.4), angina and a past CVA. There is no record groin or PR examination. The examining doctor documented the bloods were essentially normal, but they are not in the notes. An ultrasound was performed that showed fluid around liver and spleen as well as between small intestinal loops. The initial ED doctor (I presume an intern) discussed it with another ED doctor (perhaps the registrar) who advised to discharge patient to GP but for some reason (not mentioned) the ED consultant was then involved who considered it as 'undiagnosed abdominal pain, peptic ulcer, malignancy' and advised on outpatient referral to gastroenterology. The patient was discharged.

The patient continued to feel unwell with increasing pain and vomiting and eventually called an ambulance about a week after the first ED visit. The patient arrived in ED at 15:30 and the triage nurse documented that the patient was 'hypotensive with postural drop' and had a 'large red hot swelling in left groin' (the wrong side). The nurse determines a triage category of four and the patient placed in the waiting room. There were two further inaccuracies in the triaging notes as 'mode of arrival' was recorded as 'walking' (not ambulance) and the 'type of visit' was documented as 'ED - 1st visit'. All these must have affected the triage category.

An ED doctor reviewed the patient at 20:24 and diagnosed a "right inguinal hernia or abscess" and asked for a surgical review. The surgical registrar reviewed the patient at 23:40 and diagnosed an 'incarcerated inguinal hernia + SBO'. The Warfarin was reversed and taken to theatre at 00:30. Anaesthetic induction took an hour.

During the first operation, which was performed by the consultant assisted by a senior registrar, a high approach was utilised and strangulated small bowel was found in a right femoral hernia. While trying to reduce the bowel, it perforated contaminating the area. A small bowel resection was undertaken, the area was thoroughly washed and the inguinal canal was closed without mesh. A Blake's drain was left in the pelvis and a Penrose drain in the rectus sheath and the wound was closed. The patient was put on antibiotics and was taken to ICU.

Next day a pus collection developed in the femoral canal and at a second operation the surgical registrar made an incision in the right groin and drained the collection. The wound was debrided and packed. The consultant was well informed about the progress of the second procedure. The patient stayed in ICU until day five post operatively then transferred to the ward.

On day six the patient died of arrhythmia. A post-mortem examination confirmed 'acute myocardial arrhythmia due to coronary artery atherosclerosis'.

Comment

The obvious problem is the delay in diagnosis, which led to delay in management. On the first visit to ED the examination was not thorough and the groins not examined. Examination of the groins and PR examination are a routine part of any acute abdominal examination. Unfortunately this part of the examination was omitted and thus missed important signs. Moreover, the intra-peritoneal fluid seen on ultrasound remained unexplained. All this delayed the diagnosis by about a week.

The second poor assessment was by the ED triage nurse at the second presentation. Despite that fact that the patient was brought in by an ambulance and the clear documentation of hypotension with postural drop and the tender and red groin swelling, the patient was triaged as category four and was placed in the waiting room! This led to about five hours delay before a doctor assessed the patient. It is hard to criticize ED doctors for the delay. There was also 2-3 hours delay before the surgical registrar reviewed the patient.

The patient was taken promptly to theatre. The patient had the right procedure through the right approach. I have two comments

- The bowel perforated while trying to reduce it from the femoral ring. Dividing the lacunar ligament would have made the reduction easier.
- The operating surgeon commented that the femoral canal should have been drained. This could have been achieved by either packing, or leaving it open or by using a vacuum dressing. A tube drain would probably have been ineffective. Proper drainage was done during the second procedure the following day.

I hope the hospital through its internal audit and quality assurance pathways, picked up the poor triaging and addressed the issue with the nurse and ED management.

Urosepsis I - Exclude a UTI Before Urinary Tract Instrumentation

An elderly patient underwent investigation for haematuria. There was a background of IHD, angina, moderate aortic stenosis and sick sinus syndrome.

To investigate the haematuria a cystoscopy, retrograde study and ureteroscopy were undertaken. This confirmed a transitional cell carcinoma in the ureter that was stented. Definitive treatment for the ureteric tumour was planned for a later date following cardiology assessment. The patient presented to an Emergency Department with hypotension, fevers and rigors 48 hours later. The hypotension was poorly responsive to resuscitation and blood parameters confirmed high white count and deranged renal and liver function. Despite admission to ICU and inotropic support the patient succumbed to multi-organ failure, presumably from urosepsis.

Comment

Pre-operative and intra-operative documentation was scant, but a pre-operative urinalysis confirmed positive leukocytes. The retrograde study and ureteroscopy was undertaken with no documented peri-operative antibiotic cover and this may have contributed to the urosepsis. The indication and type of surgery was entirely appropriate and the patient certainly did have co-morbidities, particularly cardiac, that contributed to their death.

Urosepsis in the elderly, especially with co-morbidities, is extremely poorly tolerated and often a life-threatening event. The general adage that urinary instrumentation should not be performed in infected urine should be

particularly applied to such patients. In some situations this is unavoidable but in a procedure to investigate the upper tracts such as a retrograde or ureteroscopy.

Urosepsis II – UTI not Excluded Prior to Trial of Void.

An elderly patient developed urinary retention three months prior to this admission following a fall at home. The prior medical history included chronic renal failure, ischaemic heart disease, hypertension, and depression. The patient was admitted to the same hospital six weeks later for a trial of void that failed. On that occasion a urine culture isolated *Staphylococcus aureus* and he was commenced on a short course of antibiotics.

This admission was for a further trial of void. There is no documentation of a further urine culture being performed or ordered. At the time of admission the patient was not on antibiotics. The notes on the first day are fairly scarce. A bladder scan in the afternoon showed nearly a litre in the bladder. The nurses attempted to insert a catheter, but nothing drained. A catheter was then inserted by a doctor and drained creamy purulent-looking urine. The patient was mildly disorientated, but apyrexial and haemodynamically stable. Investigations showed the creatinine had risen from 300 the month before to 1,000 and there was a leucocytosis with a white cell count of 23,000 and a CRP of 185.

An ultrasound showed no evidence of upper tract obstruction. Intravenous antibiotics were commenced and a subsequent urine cultured enterococcus for which the antibiotics were appropriate. It was presumed the urinary sepsis was the cause of the renal deterioration. Clinical examination suggested the patient was euvolemic and the urine output was reasonable. However, daily weights were not obtained nor was a consultation with a nephrologist. On the second day the urology team passed the patient's care to the medical team.

Despite what seemed like adequate hydration the renal function failed to improve. Two days following admission under the medical team the patient was assessed by the after hours resident medical officer because of blood in the stool. A distended tender abdomen was noted. The impression was of possible upper gastrointestinal disease or diverticular disease. At that time the patient was receiving a three unit transfusion for anaemia. Two days following this the after hours medical resident was again asked to review the patient and noted generalised body pain with tenderness to palpation over the entire abdomen. The patient was mildly confused and the pain was put down to general hypersensitivity from the acute renal failure. Decreased urinary output was noted.

The following day a CT scan of the abdomen that showed marked colonic diverticulosis only. It also commented on inflammation in the left paracolic gutter with thickening of the left adrenal and left Gerotas fascia, but a homogeneous pancreas. The following day the serum lipase was 1,500 and a diagnosis of pancreatitis made. The patient continued to deteriorate and died three days following the diagnosis of pancreatitis and eight days following the original admission. The cause of death was listed as urinary sepsis and pancreatitis.

Comment

Urinary infection is a common complication of urinary catheterisation. This was treated on the first admission. However, no proof of cure was confirmed by obtaining further urinary specimens. The patient was readmitted for a second trial of void without a further urine culture performed prior to this and without being commenced on antibiotics.

The admission notes are fairly scant and the patient may not have been examined until found in retention that evening. Established urinary sepsis was probably present even prior to admission given the deteriorated creatinine and raised inflammatory markers. All of this may have been prevented with a simple urine culture and appropriate antibiotic treatment before removing the catheter. Removing a catheter in a septic patient with purulent urine probably contributed to the demise.

Transfer to the medical team was appropriate. Their management of the acute on chronic renal failure was possibly sub optimal. The fluid balance seemed to be an issue and daily weights were not performed nor was a nephrology consult obtained. With hydration and antibiotic therapy the creatinine would have been expected to fall whereas it remained at its elevated level throughout the patient's admission.

Finally there was a delay in the diagnosis of the pancreatitis of some four days from the original signs and symptoms of an acute abdominal process. It may be that an earlier diagnosis would not have changed management as the patient was already being nil by mouth and being treated with intravenous fluids.

Urosepsis III – Obstructed Kidney not Decompressed.

An elderly patient was admitted with symptoms of right renal colic. The significant past history included myelodysplasia, chronic renal failure, atrial fibrillation and urinary retention requiring long term IDC. On admission, a CT scan confirmed right hydronephrosis secondary to two distal ureteric stones measuring 6 mm and 4 mm.

In view of the past history, a conservative approach was suggested since on admission there was no clinical evidence of associated sepsis or infected obstructed system. The patient was admitted for analgesia and observation. In particular, several notes were made to inform the specialist in charge if the patient developed a fever. A catheter specimen of urine identified mixed growth only. The white cell count on presentation was elevated at approximately 20,000, which was not unexpected in view of the myelodysplasia. The pain was relatively well controlled requiring occasional doses of opiate analgesia.

At 48 hours the chronic renal failure was acutely exacerbated with a creatinine rise from 134 to 283. There was now lethargy with early onset respiratory difficulty and reduced oxygen saturation. The white cell count increased dramatically from 20,000 to 114,000 and an urgent haematological consultation was undertaken to exclude an acute leukaemic transformation of the known myelodysplasia. A medical review was also undertaken since at this stage the patient's mental state deteriorated with confusion and hallucinations. Throughout this 48 hour period the patient remained entirely afebrile. The tachycardia was stable and constant since admission and was presumed to be secondary to atrial fibrillation.

On the third and fourth day following admission the patient's mental state deteriorated and required Haloperidol for confusion. There was increasing respiratory distress requiring oxygen. Two medical reviews strongly considered sepsis and blood cultures were undertaken and intravenous antibiotics commenced. The renal function at this stage had stabilised and pain was reasonably well controlled. However, the patient's mental state and respiratory distress gradually worsened and on the sixth day the patient arrested and died. The subsequent blood culture results confirmed gram negative bacteria in the blood.

The surgeon in charge completing the audit form felt that the sepsis should have been diagnosed earlier so that possible decompression of the obstructed kidney could be undertaken.

Comment:

An infected obstructed kidney is an acute surgical emergency that requires urgent decompression of the obstructed system either by an ureteric stent inserted cystoscopically or a percutaneous nephrostomy. The urgency of this situation is so well known that every patient with renal colic is closely monitored for two important clinical parameters that may alert the specialist for infection. Firstly, a fever is generally a good indicator in well patients with renal colic and secondly, the presence of leucocytes or an infection on urinalysis is also an indicator for urgent decompression. The white cell count is often elevated generally to less than 20,000 in even non-infected renal colic. Renal tenderness is also a non-specific finding.

In this case these two clinical parameters were masked or not present due to the patient's underlying medical condition. The failure to mount a febrile response was most likely secondary to the underlying haematological condition. Secondly, a catheter specimen from a patient with a long-term catheter will always either show mixed growth or evidence of a UTI which is usually colonisation that does not require treatment based on the MSU result *per se*. The raised white cell count was higher would be expect from simple renal colic but again might have been explained by the haematological condition as was the tachycardia by the AF.

This patient was a difficult diagnostic dilemma whereby the usual indicators for sepsis were not present or masked. In retrospect, there were other indicators to suggest the patient was septic including deterioration of his mental state, agitation and confusion and worsening respiratory distress.

The record keeping in the notes was excellent and there were many entries in the notes requesting close monitoring of the patient's temperature so that intervention could be undertaken if the patient was febrile. This case is a reminder that the absence of a temperature does not exclude infection especially in patients who are elderly, immunosuppressed or have an underlying haematological condition. This requires a high index of suspicion of sepsis in such patients where evidence of sepsis can be as subtle as an alteration of mental state.

Urosepsis IV – Organisation Failure Resulted in No Pre-Operative MSU.

An elderly diabetic patient with a past history of ischaemic heart disease was admitted for elective bladder neck incision and removal of a bladder stone. The patient had previously undergone a TURP (transurethral resection of the prostate) the previous year.

Pre-operative urinalysis demonstrated blood and white cells. No formal MSU was sent for MCS pre-operatively. It was decided by the pre-admission nurse that the patient did not require pre-admission assessment.

The patient was given gentamicin on induction. The patient then underwent a bladder neck incision using a Mercedes technique with incisions at 12, 5 and 7 o'clock. Catastrophic bleeding was encountered and the procedure was abandoned with the stone left in the patient's bladder. A urethral catheter was placed to tamponade the bleeding and the patient returned to the ward.

Approximately three hours post procedure the patient became hypotensive and tachycardic. Possible sepsis was recognised and the patient was given intravenous cephalosporins and transferred to the intensive care unit. The patient was placed on inotropic support and, despite having cardiac and respiratory failure on admission, appeared to improve over the first twenty four hours.

By the next morning, blood culture results, and urine culture results taken from the previous day demonstrated *E. coli*.

On the second morning in intensive care he appeared to be improving with reducing inotropic requirements. The patient was given fresh frozen plasma for a mild coagulopathy and reduced platelets. The patient suddenly deteriorated on the afternoon of the second day of ICU admission and was unable to be resuscitated. The patient's family refused a post-mortem. However a coroner's report stated that the most likely diagnosis was of AMI (acute myocardial infarction) secondary to gram negative sepsis and subsequent multi-organ failure.

Comment

This patient suffered an adverse event, and there are several areas where comment can be made regarding pre-operative assessment and operative technique.

The decision not to have a pre-admission assessment, and therefore a lack of pre-operative MSU, is an area for concern. Treatment with antibiotics of his *E. coli* urinary infection prior to admission to hospital and surgery may well have altered the outcome for this patient. It is increasingly difficult to ensure that patients do have appropriate pre-operative investigations in an era of increasing utilisation of day of surgery

admission. However this does not excuse a lack of appropriate investigation and should be followed up by the hospital and surgeon involved.

The operative technique utilised is also an area for consideration. Incisions at 12 o'clock, particularly post TURP, have a high risk of entering the dorsal venous complex and being associated with catastrophic bleeding. This is what occurred in this case exposing the patient to direct venous exposure to infected urine. Incisions at five and seven o'clock, which might be considered more standard treatment, and would have been less likely to precipitate this event.

Once the patient's condition deteriorated, the standard of record keeping, and further management in the intensive care unit were excellent.

Urosepsis V – Death Despite Proper and Prompt Treatment.

An elderly patient was admitted for investigation of confusion and pyrexia of unknown origin. There was an extensive past history including mixed connective tissue disease requiring long term steroids, renal impairment, abnormal liver function from chronic hepatitis and pancytopaenia. The patient was taking steroids and immunosuppressives. Aside from a wide range of investigations including CT scanning, gallium scan, liver biopsy and input from Haematology and Microbiology, the patient underwent a renal biopsy to investigate acute deterioration of renal function. This suggested acute tubular necrosis in the background of mesangiocapillary glomerulonephritis.

The pre-biopsy and post-biopsy MSU were both negative for infection. Two weeks later the patient developed urosepsis and a dilated left renal pelvis presumed from a bleed into the kidneys secondary to the renal biopsy. This was treated with immediate decompression with a left nephrostomy tube. Cultures confirmed *E.Coli* and *Candida* in the urine and the patient was treated with the appropriate microbiological treatment. Due to bleeding, possibly from the nephrostomy tube, an indwelling left ureteric stent was inserted cystoscopically three days later to unobstruct the kidney.

Despite this the patient developed SVT, pseudomembranous colitis from *Clostridium* infection followed by ARDS which was non-cardiac in nature. Despite ICU support and appropriate antifungal and antibacterial treatment, the patient deteriorated and passed away 12 days following insertion of the nephrostomy tube.

Comment

I do not feel that there has been any area of concern or preventable adverse event that contributed to the ultimate outcome. The two most important rules to prevent life-threatening urosepsis are firstly to ensure sterile urine prior to any intervention on the renal tract, and secondly to unobstruct any possibly obstructed system. Both of these rules were adhered to in management of this patient and documentation and referral to ICU was appropriate.

The patient had significant co-morbidities including pancytopaenia and immunosuppression. The presence of urinary sepsis both by bacteria and fungal organisms was appropriately managed by urgent decompression of the obstructed renal unit by nephrostomy tube in the first instance. When there was doubt this may not be draining adequately due to blood an indwelling stent was placed which is the appropriate management in such a situation.

The ultimate outcome was not preventable and the nephrostomy and surgical intervention were both entirely appropriate. The patient's demise was more a reflection of immunosuppression and pancytopaenia and the inability to elicit an appropriate immune response to infection.

Editorial note

WAASM's sister organisation in New South Wales, the Collaborating Hospitals Audit Surgical Mortality (CHASM), has just published its second Case Note Review Booklet. It includes two deaths related to urinary sepsis.