A Stick and A Stone. A pair of acute abdomens

We received a call from our aid outpost radio service. A lady, 3 days walk away from Kompiam, had fallen overnight onto a stick. She had come into the health outpost in the morning, after attempting to sleep off the injury, as she was now in terrible abdominal pain, nauseated and was febrile. These remote aid outposts are often poorly equipped and stock little more than simple analgesia, a sporadic spread of antibiotics and vaccines. She was transported by plane to Kompiam and arrived to the hospital via Landcruiser/ambulance. She got herself out of the back of the Landcrusier, carrying her own IV fluids and IDC bag, and walked into the ward.

On arrival she was tachycardic, febrile to 38.5, tachypneic and had a stable blood pressure. On history she had fallen onto a stick which had punctured, within the region of her vagina and that this stick had penetrated about 18cm. The exact distance given seemed oddly specific, until a family member produced that stick which had caused the injury, which had been measured. On examination, she was stable with a normal airway, breathing, circulation. She did not have a grossly peritonitic abdomen however it was locally hardened in the LIF. The site of injury was briefly examined, determined not to be actively bleeding and decided that further examination in theatre was indicated. She had two large IV cannulas placed, fluids were run and state triple IV antibiotics. Blood donations were sort and crossmatches performed as intraoperative bleeding was expected.

The perpetrator

She was taken to theatre and a speculum exam found the puncture wound located the anterior vaginal wall, anterior to the cervix, and had a mild amount of active external bleeding. She had a midline incision made for the exploratory laparotomy. Initially the first clue of injury was the enflamed and erythematous small bowel. However, shortly, a pocket of blood and feaces was found in the abdomen. The source was found to be a punctured transverse colon, sigmoid colon, small bowel, and lacerated left uterine artery, without puncturing the bladder, rectum or uterus. Surprising to me, all these structures seemed to have been somewhat walled off by the omentum, therefore likely causing the clinical lack of generalised peritonitis. I would have suspected that a traumatic injury would have resulted in widespread immediate peritonitis, rather than examination findings that were found. Regardless, the perforated structures were repaired and the abdomen was washed, the muscle and fat layer closed without an abdominal drain. The skin left open for delayed primary closure. An NG tube was placed and the patient was started on maintenance IV fluids.

A pethidine infusion was initiated for pain relief, a new luxury in Kompiam with flash electronic infusion pumps. The NG tube drained approximately 4L over the next 24hrs. Fortunately, we are able to test potassium, and she was low, therefore IV replacement was initiated. At 48hrs she started passing wind and her NG tube was spigotted and she was started on the PNG equivalent to clear fluids (Oral Tang, similar to cordial but with a bit of bicarbonate fizz). At 72hrs the NG was removed and she was hesitantly eating and drinking. At a week she was mobilising normally, eating normally and was just awaiting removal of her sutures, which was completed prior to discharge on day 10 post operatively.

A 40 y.o male, Fred, presented late in the afternoon with severe abdominal pain, nausea and reduced oral intake for the last 2 weeks. He had reported that he had not eaten anything for the entire two weeks, only drinking small amounts of water. The pain was unchanging and constant. Bowels were still open, however he was only making small amounts of urine. History taking was challenging due to the language barrier between all involved. I suspected that this guy may be exaggerating the story as to justify coming to hospital, to ensure he got the “good stuff”, as some other patients had done previous.

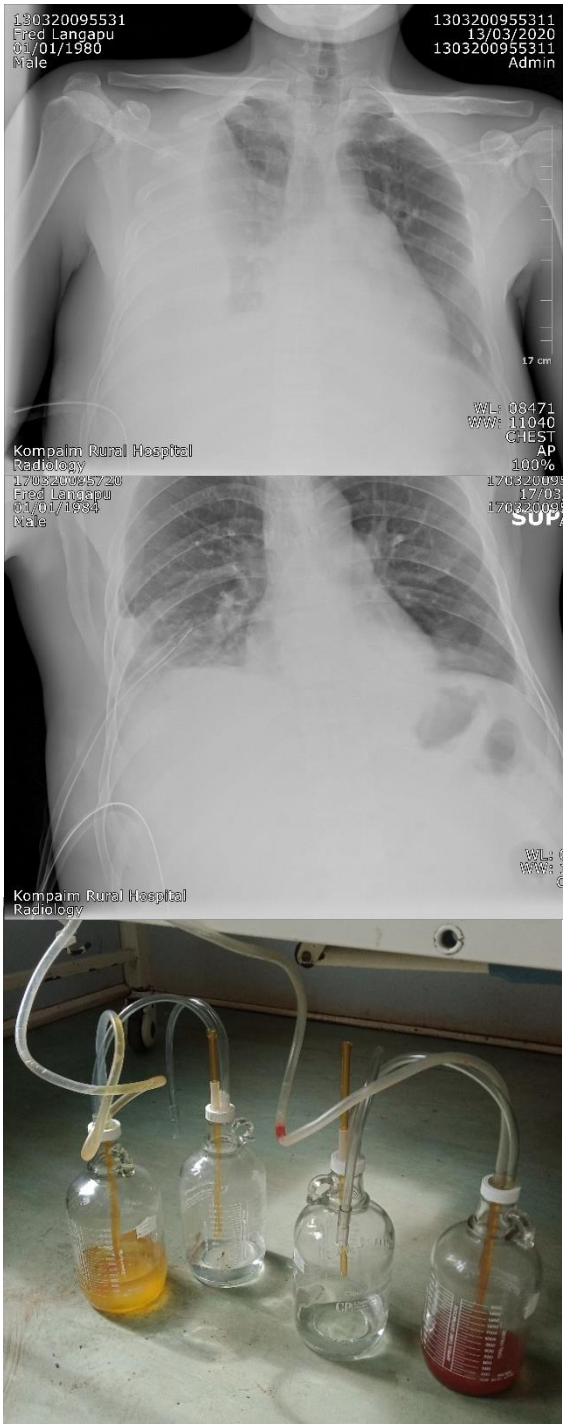
Frustrated by the lack of ability to differentiate specific symptoms from the history, examination was expedited. He was tachcardiac, tachypnoeic, and had a stable BP. His abdomen had board like rigidity, with percussion tenderness and rebound tenderness throughout. Startled by the discovery of a peritonic abdomen the patient was urgently taken around to the XR machine for an erect chest XR. The R sided free gas under the diaphragm, supported by clinical peritonitis, proved the patient as truthful and not exaggerating. OT was activated and blood from family members crossmatched. He was given triple antibiotics immediately and aggressively fluid replaced. His starting Hb was 150.

Dr Mills and Dr Williams operating on the appendiceal abscess.

It was initially suspected that Fred had had a perforated gastric ulcer, as this area is thought to have less bacteria than other bowel region. It was theorised that if his perforation had come from his large and small bowel then he was likely to have perished from sepsis, due to the high bacterial load. He was given a general anaesthetic with Ketamine Infusion, Low dose Halothane, and paralysed with acuronium. An epigastric midline incision was made, and non-purulent, transudative fluid was evacuated from his superior abdomen. However, no perforation was found and the omentum seemed tethered to the lower abdomen. The incision was widened to the suprapubic region and the omentum interrogated. A purulent collection in the Right iliac fossa was identified and the omentum was adhered to the posterior aspect of the ceacum. This was dissected and the remnants of an appendix, well bound to the omentum was found. The diagnosis of a ruptured retroceacal appendiceal abscess was made. The necrotic and fibrotic tissue were debrided, an assumed appendix was in this mess but not fecalith was found (no stone). This tissue was exceptionally friable, and the ceacum was damaged, requiring repair. The abdomen was washed, with muscle to sub cutaneous closed.

He had a NG tube, IDC, two large cannulas inserted and no abdominal drain was inserted. After initial rapid IV therapy, his IV was slowed to maintenance and his pain post operatively was through another slow pethidine infusion. Post operatively his Hb was stable, had a normal potassium and sodium. He pasted flatus at about 72 hrs and opened his bowels shortly after. However, he was not out of the woods. The night of day five he started to get worsening abdominal pain/tighteness, tachypnea, tachycardia, dropping oxygen saturations and a subjective fever (the nursing staff for the shift overnight had no thermometer to make it objective). On review his abdomen was showing signs of worsening peritonitis, rather than improving. It was decided to start again, NG placed, his large bore IVCs reinserted and he was prepared to return to OT for a suspected ruptured bowel from the previously damaged ceacum.

On reopening, no gross faecal contamination was noted thought there was lots of peritoneal fluid generally. The bowels showed signs of early adhesions. The caecal area, which was suspected for having a leak, appeared to not have any active leak nor bleeding. However, the cause of the peritonitis did not take long to be found, just inferior to the liver, extending up to the diaphragm was an infraphrenic collection. This was yet to form a well organised abscess like that of the old appendiceal abscess, however this collection of bacteria created a new home. The collection broken down, the adhesions manually discouraged and the abdomen washed out. At the washing of the abdomen, with the large bacterial load he became more tachycardic, decreased his blood pressure and his saturations dropped into the mid 80s. Once closed and the surgery completed, his halothane was replaced by a ketamine infusion to ensure tolerance of his tube whilst we waited to see what would come of his saturations. His saturations improved over the next two hours and was successfully extubated and wardable, with a pethidine infusion for pain relief. He was generally oedematous, with pitting oedema of his legs and arms bilaterally. This was attributed to absolute protein deficiency, and with no IV alternative in Kompiam, his oedema only worsened.



Over the next few days he developed worsening saturations and on CXR a unilateral consolidation in the right lower zone of his lung was found. It was hoped that this represented a reactive effusion, as the abdominal collection had been separated only via the diaphragm. Eventually, Fred spiked a fever and we promptly inserted a chest drain. The “chest tube” initially drained a large quantity of blood-stained puss, despite the large output from the drain, his symptoms only improved slightly. The next day the chest tube had ceased to swing and repeat XR showed the drain was very inferior with an ongoing large effusion superior to the chest tube site, therefore another chest tube was inserted 2 rib spaces above. The new tube drained a large quality of straw-coloured serous fluid and saw his symptoms of shortness of breath improve rapidly. The contrast between the content of the two drains was immediately evident. We assumed the empyema must have been extremely loculated to cause such a difference in the drains. The next day he was taken off oxygen, ceased his pethidine infusion and was somewhat mobilising. His mobilisation is limited as the glass underwater sealed drains are quite cumbersome.

Once the drains had stopped swinging, which the inferior one struggled with immediately, both drains were retracted about 5cms, both continued to drain some fluid, however the inferior drain was not

swinging well. Another CXR was done, showing the superior drain in the upper lobe, however the inferior drain dived down, outside of the CXR view. Eventually, the inferior drain was chased radiologically and was found to be located next to ASIS. It is assumed that this was never a chest drain, but a pan thoracic abdominal drain. It had drained the hemopurulent fluid still residing in the abdomen. This raised the question of the potential damage the drain could have produced, and how should removal be done.

As the patient continued to improve, his drains were removed with fingers crossed and solemn prayers. He recovered with nil observable significant defects. He was seen in the ward a month after discharge. His two big scars on his chest and midline incision scar were accompanied by the much-needed return of some fat and a big smile.