Dysentery or not dysentery, that is the question

50 y.o male admitted to Hospital post falling out of the back of vehicle and falling onto his L side. He is assessed, FAST scanned and Xrayed, revealing a 9th rib fracture. The patient is admitted and prescribed antibiotics and simple analgesia. He is reviewed again the next day and antibiotics are ceased and he is advised to take deep breaths even if it hurts to help with chest physio. He is discharged the following day as he is well, taking deep breaths and mobilising well. He is from a different area in Highlands but has friends locally he can stay with.

Returns the morning one day post discharge with decreased conscious state, perfuse diarrhoea, nausea and vomiting. Rousable to pain, very faint thready peripheral pulse. People who brought him report that they found him outside on the ground in the early hours of morning, unconscious. He was covered in feaces and looked like he had vomited, they reported that there was blood in the diarrhoea. His vitals were HR 90, BP 88/50, RR 22, Sats 99, Temp 35. He had equal and bronchovesicular breath sounds, a soft abdomen and normal heart sounds. He had a finger prick Hb of <60, a baseline haemoglobin had not been previously recorded. A peripheral line was inserted with difficulty and he was fluid rehydrated with Normal saline. He had multiple blankets placed on him to try and warm him up and he was treated with broad spectrum antibiotics for septic shock.

He became more rousable, further history revealed that he had gone to the market after discharge and eaten some pig, that evening he felt nauseated, was vomiting and had perfuse dysentery. After so much vomiting and diarrhoea, he felt so weak that he fell and was unable to get back up in the outhouse and collapsed on the ground. There he was exposed to the elements for an uncertain period of time.

Therefore, the working diagnosis was Dysentery with hypothermia, causing the combination of symptoms. The antibiotics were narrowed to local bacteria which cause dysentery (Likely salmonella Typhi) and blood transfusions were arranged (sourced from friends, as he had no relatives in the area).

After an unfortunate, prolonged and intimate encounter with the patient's stool, it was thought to not be containing blood, however this was debated amongst the medical team.

Never the less, he had been transfused two units, had 3 bags of saline and his observations had mostly normalised and his haemoglobin raised to 85.

I was called to review him overnight due to him having ongoing pain. On review, he appeared similar to earlier in the day. Observations were in line to previous. His abdomen was diffusely tender, without specific focus, however was not peritonitis, as previous. Although, his abdomen did seem grossly more distended than previous with a loud percussion note in the epigastric region. His diarrhoea had ceased, he felt nauseated but had not vomited much recently. We had reached the pinnacle of the local pain medicine pyramid. I decided to pass an nasogastric tube, in attempt to relieved his nausea and maybe his pain was secondary to distension of an oncoming bowel obstruction (sometimes patients with typhoid can have obstructive/constipated type symptoms) . Although, minimal output came from the NGT and was removed a couple of hours later due to patient discomfort and minimal drainage.

The next morning, he was similar, ongoing abdominal pain, seeming out of perspective with the diagnosis of gastroenteritis. His relatives had arrived from a different area, as they had heard that his condition had worsened. They told a different story regarding his initiation injuries. He had gotten into a fight nearby to Kompiam whilst intoxicated. He had lied as he had been told that he would be

charged a higher Hospital fee if he had been fighting. This initial injury had been 6 days previous to the 2nd admission. We rechecked his Hb and found it to be 72. We repeated the fast scan and found significant abdominal free fluid in Morrisons pouch, throughout the abdomen. An IDC was inserted which drained straw coloured urine.

The diagnosis of splenic rupture secondary to assault was made and the call for significant blood donors went out. The family members were screen and venesected where appropriate (Hep B and HIV and strife throughout the community, therefore only about 80% of blood can be used). The patient had few family members present, therefore and I had the strange experience of running the group and cross match on my own blood freshly venesected blood. Throughout the afternoon his Hb continued to drop despite continual bags and the decision was made to go to theatre at 1700. 6 different bags of blood were cross matched to the patient.

Later that evening I had the same interesting experience of suctioning some of the my donated blood from his abdomen. A total of 3 litres of blood was extracted from his abdomen as well as an actively bleeding spleen. With the bleeding under control post removal, some bits of spleen were replanted via being sowed into his omentum. It is thought that the spleen can regrow somewhat from a 50cent coin size of tissue. (tropical diseases such as malaria do cause significant issues with those spleenless)

Was the diarrhoea, nausea and vomiting due to gastroenteritis, causing sufficient intra-abdominal strain leading to clot breakdown and delayed splenic bleed? Or did the peritoneal blood, secondary to the bleeding spleen, cause enough irritation to cause diarrhoea and nausea and vomiting?