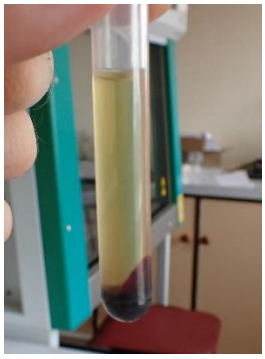
Top them up and send them home.

A 50 y.o lady presented from an aid outpost about a day's walk away, however she was flown into Kompiam as she was unable to walk the distance. Her main complaint was abdominal pain which had been present for 3 months, however she had not been able to walk secondary to shortness of breath and global oedema. She reported that her oedema had only started 2 weeks previous when seen at the aid outpost, she had been given a litre of IV fluids. By the time I had seen her, the abdominal pain had resolved, although she had only been given paracetamol, a penicillin class antibiotic and albendazole. She was not particular concerned regarding the oedema, only being thankful that we had alleviated her pain.

On examination she was saturating at 97%, HR in the 90s, BP 88/48, normal respiratory rate and was afebrile. She appeared comfortable, but was grossly fluid overloaded with severe pitting oedema, ascites and even swollen breasts and forearms. She had pale palmer creases and pale sclera. She had an ejection systolic murmur. She had good air entry with fine crackles in the bilateral bases, percussion note resonant. Sacral oedema was present. On abdominal examination, an enlarged liver edge was palpated, approximately 5cm below the costal margin, it was difficult to get the texture of the liver due to the ascites. Her pitting oedema extended well up her tights. No lymph nodes in the neck nor the inguinal region where palpable. A chest XR showed and enlarged heart, pleural effusions and some signs of pulmonary oedema.

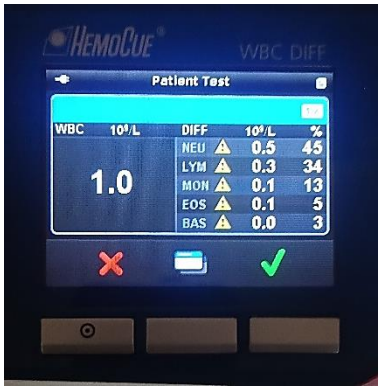


The clinical suspicion of the origin of the anaemia was initially that of either liver or kidney origin. A finger prick Hb, Rapid Hep B/ Hep C/ HIV/ malaria stirps were complete, urine dipstick and the temperamental electrolyte/liver function test machine was fired up. Her Hb was 19 g/L, was Hep B positive, Hep C positive, with + protein on urine dipstick and her albumin came back as 10. The LFTs were normal or failed to produce a result on our machine.

I suspected her having synthetic disruption of her liver, causing a decrease in clotting factors and resulting in the gross oedematous state. Therefore, causing occult GI bleeding and the oedema relatively contributing to her anaemia. Once the experience SMO reviewed the patient, he suggested an alternative and indicating politely that I was wrong. If she had that severe an impairment, then why was she not jaundice or encephalopathic? that all the symptoms are likely to be secondary to severe anaemia and that the symptoms will resolve with transfusions. We are unable to treat nearly all causes of liver disease or occult GI malignancy regardless, therefore our only goal of treatment was to top her up with red cells.

A 20-year-old female presented to hospital with a scattered history of abdominal pain, fatigue and a recent event of hematemesis. The abdominal pain was the sensation of tightness in her epigastric region, was inconsistent in nature, did not move anywhere else and did not impact significantly on her working in the garden. She had a single episode of hematemesis 5 days ago without an episode since, it was frank blood mixed with gastric content, with no coffee ground type content. She had travelled a days walk to arrive to Kompiam and could only speak a small amount of pidgin, speaking only Engan. Therefore, it was difficult to establish a past medical history or much of a further history.

She was tachycardic, normotensive and afebrile. She had pallor of the palmer creases and of the sclera. Her chest was clear and although rapid, her heart sounds were dual without an added sound. On abdominal exam, she had a distended abdomen and a large mass extending inferiorly to below the umbilicus. This mass was firm in nature, moved with breathing and seemed to arise from the R side of her abdomen. It appeared to be a large spleen.



A pregnancy test was performed which was negative. Her Hemaglobin was found to 25 g/L, this blood result was conducted on a point of care test and assumed to be incorrect, however on marcoscopic examination of her blood, 80% of the blood content appeared to be serum. Her total white cells were 1.0 x 10^9/L with no specific dominance of subtypes.

A transfusion was arranged immediately, although she was clinically well.

She was reviewed the next day, with an Engan translator, these issues of abnormal bleeding, fatigue had been chronic for the past 7 years. She had been monitored with this enlarged spleen and had multiple blood tests with similar results as above. She had no history of weight loss, enlarged nodes and was otherwise well. She had to reduce the amount of garden work she could do and had to take her time walking distances.

It was assumed that due to chronic nature of her symptoms and multiple objective hemoglobin tests over the past 7 years that a diagnosis of a leukemia or another primary bone marrow disease was unlikely. It was likely her spleen that was causing her pancytopenia rather than the process causing the pancytopenia to be enlarging her spleen. Therefore, these symptoms were attributed to tropical splenomegaly syndrome

Topical splenomegaly syndrome is a hyperactive spleen which is effective at protecting against encapsulated organisms and malaria, however it can get carried away and start behaving as a destructive autoimmune disease. In the above case, this over-zealous tropical Insurance was causing pancytopenia. We considered whether removal of the spleen was the best course of management. This was anticipated to be a complicated surgery, due to the size of the spleen. But aside from the acute difficulty which faced surgery, would the removal of her spleen improve or worsen her immune function. The leukopenia due to the spleen vs the disarmed immune defenses secondary to being spleenless. The decision was made, after consultation with multiple surgeons around PNG, to not perform the splenectomy. Rather to manage the patient conservatively with multiple transfusions and long-term oral malaria treatment, which has evidence for reduction of splenic size.