The Complex and Co-morbid Perioperative Patient

As the obesity epidemic swells and people are living well (and well) into their 90s; there is increasing demand for surgeons to serve complex and co-morbid perioperative patients. Regional settings such as the Shoalhaven face distinct challenges in optimising patients for surgery, with expectations on patients to travel to tertiary centres and numerous expensive investigations and specialty services.

Many general physicians such as myself have tried to bridge this gap through post-graduate training and practice in Perioperative Medicine. Decisions on when, if and how to operate and provide perioperative care should not be left upstream to surgeons and anaesthetists for complex patients. Especially if optimisation or surgery itself is potentially life-saving.

Case Study:

Jim Bean is a 54-year-old obese male who has decided to visit a GP for the first time in 20 years as he is barely able to mobilise due to bilateral knee pain. He has struggled along using ibuprofen and panadol three times a day, and 6-8 glasses of whisky a night to manage the pain. He has a sedentary job as a truck driver but lately he has been scared to climb up and down from his truck. He also is falling asleep at traffic lights and rest-stops. He denies any other medical history or medications. He quit smoking a year ago after his friend passed away from lung cancer. His father died of kidney disease in his 60s after 3 months on dialysis.

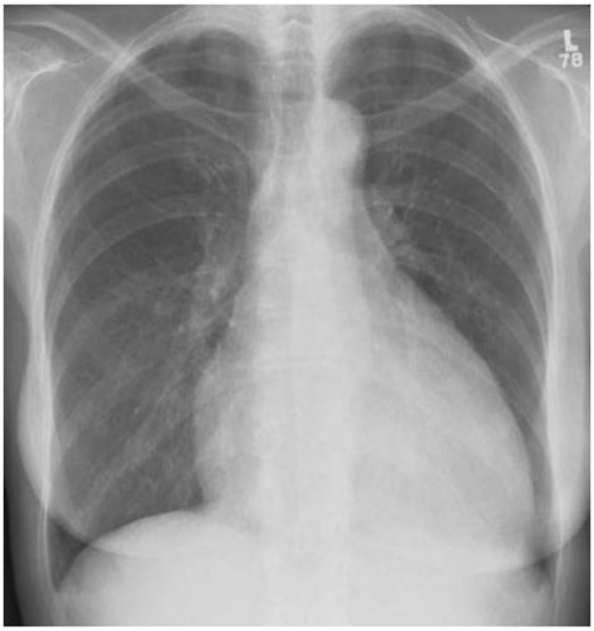
You are immediately alarmed by this history, (especially if he is to require orthopaedic surgery) and decide to do a comprehensive assessment.

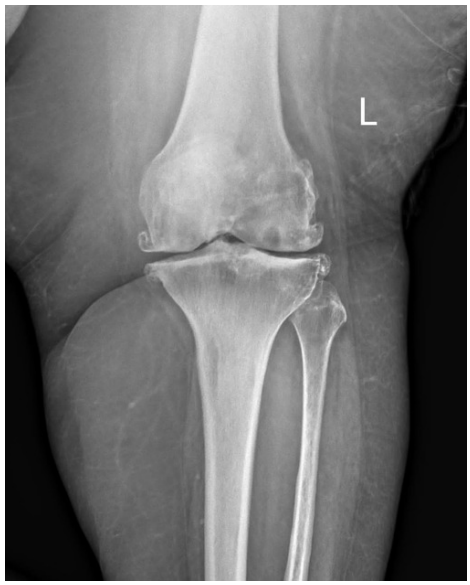
Blood pressure:180/ 98mmHg: pulse: 108 bpm and irregular; saturations are 93% on room air.
BMI is 38 with significant central adiposity
Neck circumference 42cm; Mallampati score: 3; STOPBANG: 7
Heart sounds are dual, no murmur, apex beat is displaced to the axilla.
Globally reduced air passage throughout the lungs, no extra sounds.
Minor pedal oedema
Unable to palpate liver or spleen due to habitus. No shifting dullness.
He has very limited range of movement of both knees due to pain. There are no marked effusions or heat.

Bloods and xrays return the following day:

Hb: 185g/L and normocytic, haematocrit is elevated at 52% (H)
Platelets: 118 $x10^9$/L (L)
WCC/ CRP: normal

Creatinine: 152umol/L with an eGFR of 42mL/min
Sodium, potassium and chloride: within normal limits
HCO3: 44mmol/L; VBG is run showing a CO2 of 56mmHg (H)
AST: 88; ALT: 64; GGT: 500 (H)
Bilirubin: 17umol/L (borderline)
Albumin: 32 (L)
HbA1c is 7.4%
ECG: Left ventricular hypertrophy (LVH) and atrial fibrillation (AF)
Bilateral Knee Xrays: loss of joint space, osteophytes and subchondral cysts.
Chest Xray: Cardiomegaly secondary to LVH and RVH plus atrial dilation. Likely systemic and pulmonary hypertension plus AF





Further Investigations?

1. Sleep Study +/- nocturnal oximetry: Hb suggests erythrocytosis with an elevated hct likely secondary to **hypoxia.** Hypercapnia with renal compensation is concerning for chronic ***Obesity Related Hypoventilation Syndrome***
2. Spirometry: above also concerning for advanced emphysema with
3. Urine albumin + albumin: creatinine ratio: hypertensive nephropathy main differential given BP and low albumin.
4. Renal tract imaging to exclude obstruction and polycystic disease given family history.
5. BNP – this is now coming into some Perioperative Cardiology guidelines however, cannot trust in an obese patient: often falsely low due to high levels **neprilysin** (cleaves proBNP) associated with adiposity. Also can be artificially raised in renal impairment.
6. TTE; May have poor views but essential. Given history, HFpEF by diastolic impairment from LVH or pulmonary hypertension. AF often a consequence of increased atrial pressures from right or left heart failure.
7. Liver screen: for completion, note ferritin will be high given active alcohol use and fatty liver causing inflammation

8. Fibroscan can provide stage of fibrosis / cirrhosis but limited by body habitus
9. Ultrasound with doppler of portal venous system is a must as low platelets are the first sign of portal hypertension which would also cause splenomegaly, ascites and dilated or occluded portal venous system. It is pertinent to always check for hepatocellular carcinoma (+AFP). We now know that F3 fibrosis can incur a **higher risk of HCC than F4 in NAFLD** patients (Sanyal et al., NEJM, 2021)

Management

1. Pain management:
    - AVOID OPIOIDS if concerns regarding daytime drowsiness/ nocturnal apnoea
    - Stop NSAIDs –contraindicated in hypertension, renal disease and cirrhosis!
    - Image guided or bedside injections of corticosteroid/ anaesthetic would be treatment of choice
2. Alcohol counselling/ abstinence. Tests reveal cirrhosis and a likely central sleep apnoea syndrome (OHS) and so avoid sedatives before bed.
3. Weight loss and significant diet modification. Focus should be lowering sugar content (insulin is the driving force behind NAFLD) and increasing fibre and protein for satiety + regular bowel motions. Must avoid fasting and constipation as increases risk of encephalopathy and accelerates sarcopenia. Encourage late night protein snack.
    a. Dietitian and Diabetes Educator
    b. Physiotherapist to ensure non weight bearing exercises / techniques. Patient would only benefit from an exercise program e.g. hydrotherapy!
4. Likely will be prescribed BiPAP given hypercapnia and hypoxia. Note for obesity related hypercapnia it takes a greater EPAP setting to stint airways.
5. Anticoagulation as $CHADVAS_2$ is at least 2
6. Diabetes: metformin often contraindicated in decompensated cirrhosis so need to work hard on lifestyle measures. Good candidate for an SGLT2 inhibitors and GLP-1 agonists given cardio + renal + weight-loss benefits
7. Renal optimisation
    a. STOP NSAIDs – removing constriction of afferent arteriole (Aa) increases glomerular filtration
    b. Treat blood pressure-
        i. Reluctant to start ACE or ARB in this setting. Allow for NSAID wash out, encourage home readings, and then add as cornerstone of care.
        ii. Consider a beta blocker which, whilst not having a big effect on blood pressure, will allow increased filling time for LVH and AF. However, starting a beta blocker in perioperative setting can increase risk of death and stroke (POISE TRIAL, The Lancet, 2008). BUT Trial was looking at ACS RISK, not AF!
        iii. Ultimately will likely need 2 or 3 agents and combined in a single pill for ease and efficacy!
    c. Reduce salt> fluid:

    i. Hypertension and renal disease both cause increased hydrostatic pressure
    ii. Aldosterone is the primary driver behind fluid retention in cirrhosis
8. It would be in the interest of the patient and public to suspend his licence!

SUGGESTED GUIDELINES

- **Australian Diabetes Society Of Australia and NZ (ADS-ANZCA)** guidelines for Perioperative Diabetes and Hyperglycaemia in Adults
- **American College of Cardiology, American Heart Association and European Cardiology Society** Guidelines on Perioperative Evaluation and Management of Patients undergoing Non-cardiac Surgery
- **American Society of Anesthesiologists** guidelines on perioperative management of OSA, Pulmonary Disease
- **Association of Anesthetists of Great Britain and Ireland, American Diabetes Association and European Association or the Study of Diabetes**