Anaesthesia management of 85-year-old male patient with 15% Ejection Fraction and multiple comorbidities posted for Spine Fixation

Dr. Runjhun Jain
JR2
Department of Anaesthesiology

85 year old male came to our hospital with chief complaints of :

- Excruciating pain in lower back region since 4 months
- Pain radiating to B/L Lower limbs

Past Medical History:

- K/C/O Hypertension since 2007, On T. Metoprolol 25 mg OD, T Sacubitril + T. Valsartan 50 mg BD and T. Epleronone 25 mg OD.

- K/C/O Diabetes Mellitus since 2009, On T. Linagliptin 5 mg OD and T. Dapagliflozin 5 mg OD

- K/C/O Ischemic Heart Disease since 2009, PTCA was done. On T. Atorvastatin 10 mg HS and T. Ecosprin 75 mg HS.

- K/C/O Asthma since 2012, was on treatment, discontinued since 1. year.

- K/C/O Interstitial Lung Disease since September 2022, not on any medication

Medical history continued :

- Psychiatric disorder, on T. Haloperidol 0.5 mg BD since 1 month
- AKT– Day 18 in view of FNAC of vertebra : ?Tuberculosis
- No history of Thyroid disorder/ Seizure/ Stroke

Surgical History:

- B/L cataract surgery in 2009 under local anaesthesia
- Right Knee Replacement surgery in 2015 under Spinal + Epidural Anaesthesia

History of Blood Transfusion:

- 2 pints of PCV transfused during Right knee replacement surgery in 2015.
- 1 pint PCV transfused in March 2023 i/v/o Low Haemoglobin

History of ICU stay for 6-7 days in 2018 i/v/o Diabetic Ketoacidosis.

History of Recent Hospitalization:

- 7^{th} – 10^{th} February 2023: Admitted in Military Hospital in view of Typhoid.
- 14^{th} – 22^{nd} Feb 2023: Admitted at a local hospital for urosepsis.

History of Allergies:

Allergic to Metformin

Personal History:

- Diet –mixed
- Appetite- normal
- Sleep- disturbed
- Bowel and bladder –normal
- Denies addictions

Family History:

No h/o similar complaints in other family members

General Examination:

- Patient was conscious, cooperative, well oriented to time, place and person.
- Weight: 68Kg, Height: 158 cm, BMI- 27.2 Kg/m^2
- Afebrile
- Mild pallor, No icterus, cyanosis, clubbing, lymphadenopathy or oedema
- Pulse: 86 bpm, right radial artery, regular in rate and rhythm, all peripheral pulses palpable

- BP: 140/86 mmHg recorded over the right brachial artery, supine posture and 130/70 mmHg in sitting posture
- RR: 17/ min
- JVP: Normal
- Spine- Skin normal, No kyphoscoliosis. Mild tenderness present on palpation in lumbar area

Airway Examination:

- Teeth - Edentulous
- Mouth opening - adequate
- Mallampati Score – II
- Temporomandibular joint mobility : Normal
- Thyromental distance: > 6.5 cm
- Neck Movement: Normal

Cardiovascular System:

- Inspection – No precordial bulge
- Apical impulse in 5th ICS just medial to the midclavicular line
- No dilated veins/ abnormal pulsation/ scars
- Palpation. – Apex beat confirmed in left 5th ICS, No abnormal pulsations felt
- Percussion: Left border corresponded to apex beat, right border was retrosternal
- Auscultation : S1S2 normally heard in all areas, Grade 3 Ejection systolic murmur heard in aortic area

Central Nervous System :

- Patient was anxious
- Higher functions- conscious, cooperative and oriented to time, place and person
- Power, tone and reflexes were normal
- Tenderness was present over lumbar spine on palpation

Respiratory System :

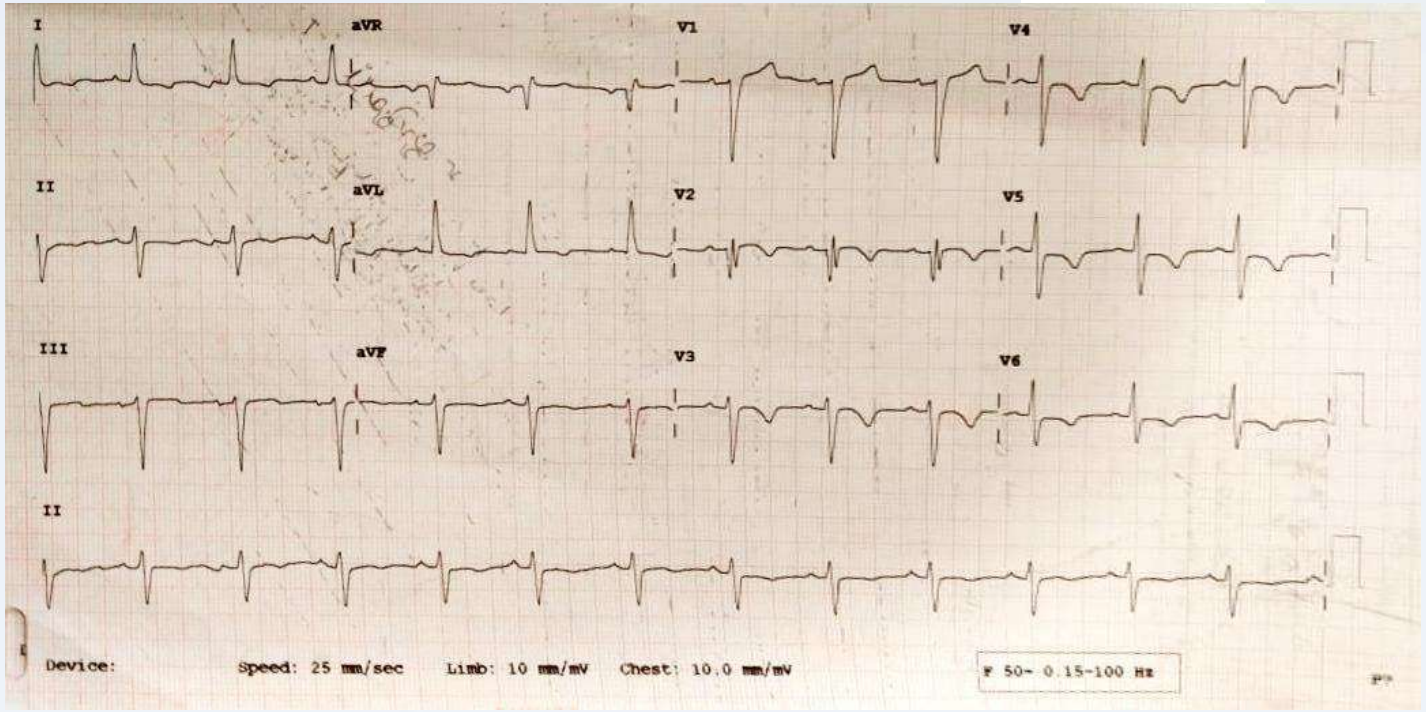
- RR – 17/min, Abdominothoracic
- No tracheal deviation
- Chest B/L symmetrical in shape, moves equal bilaterally
- On auscultation – Air entry bilaterally equal, no adventitious sounds

Per abdomen:

- Soft, non tender, no rigidity/ guarding, no distension
- Bowel sounds +, no organomegaly

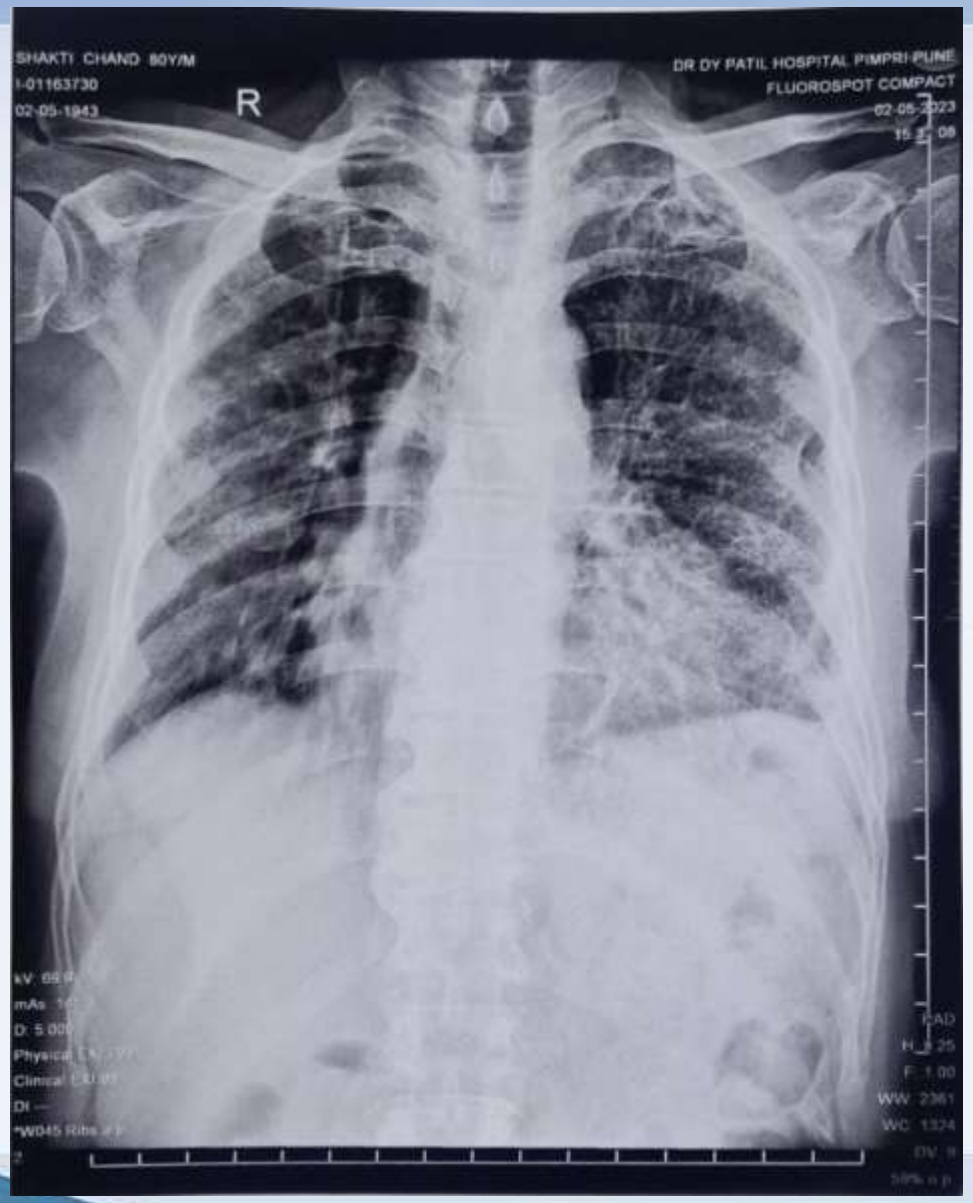
INVESTIGATIONS:

- Hb-9 g/dl
- TLC 4200 /µL
- PC-2.09/µL
- PT/INR- 12.9 sec /1.02
- Blood group: A+
- BSL-120 mg/dl
- HbA1c- 6.2%
- Urea- 19 mg/dL
- Creat- 1.03 mg/dL
- Na- 135 mmol/L
- K- 4.19 mmol/L
- T. Bil- 0.37 mg/dL
- SGOT- 22 U/L
- ALP- 10 U/L



- **ECG** showed NSR, LAD, Altered T wave axis, Nonspecific intraventricular conduction delay in V2

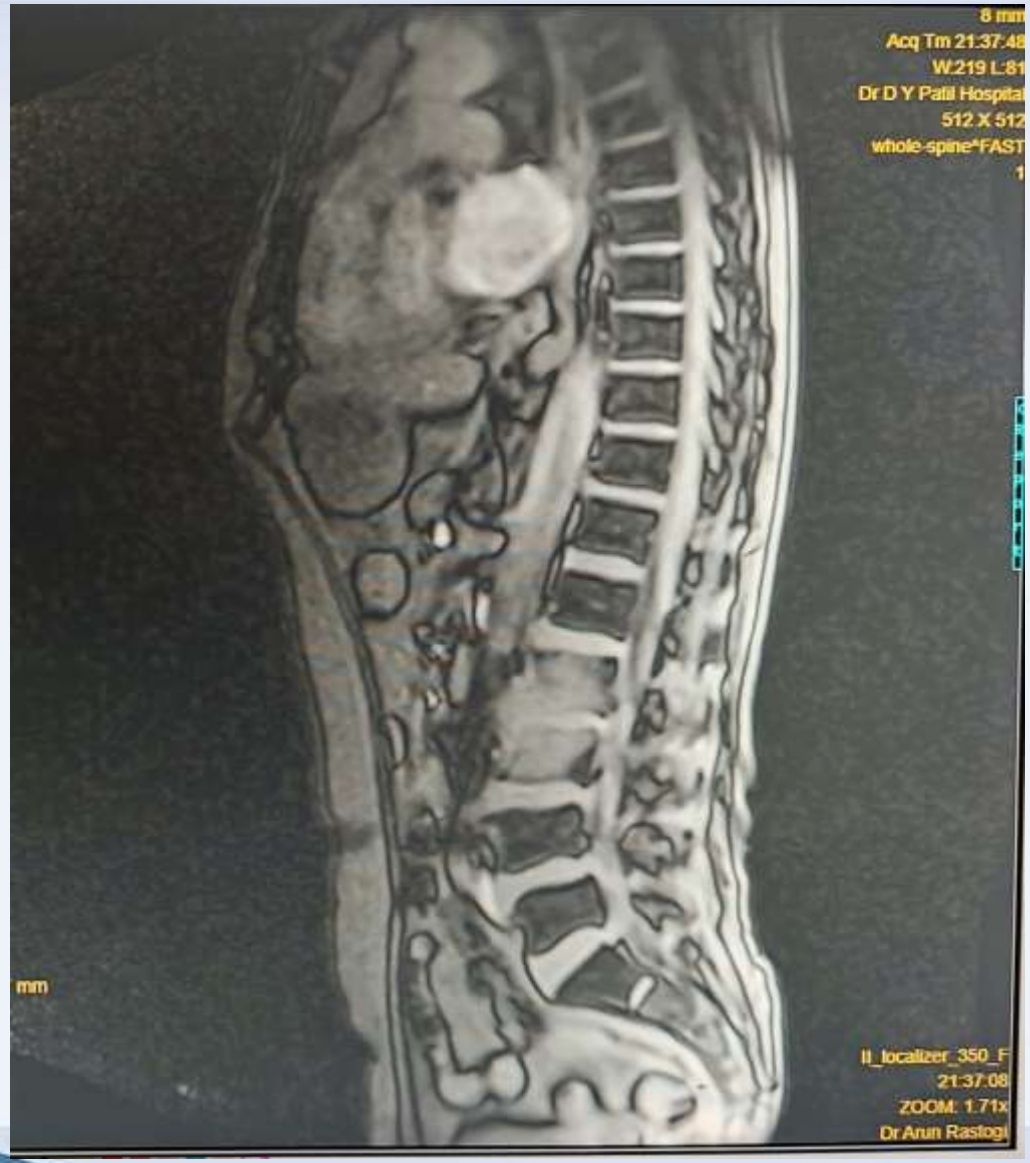
- **2 D echo** revealed EF 15%, Global LV Hypokinesia, Severely depressed LV Systolic function, Grade 1 Diastolic dysfunction, Aortic Valve sclerosed, Trivial AR, Mitral annular calcification present, Trivial MR and Trivial TR.



- **Chest X Ray** showed blunting of left costophrenic angle. Features of Interstitial lung disease +

- **HRCT Chest** revealed Fine subpleural reticulations with basal coarse reticulations, Traction bronchiectasis - s/o ILD.

- **PET CT** scan showed Metabolically active Left pleural thickening. Focal metabolic activity along L2 vertebral body.



- **Radiograph of Lumbar Spine – AP and Lateral view:** Degenerative changes of lumbar spine involving discs. Reduction in intervertebral disc space at multiple vertebral levels. Lumbar spondylosis at L3-L4 vertebral level. Possibility of infective spondylodiscitis at L2-L3 level.

- **MRI Lumbosacral Spine (Plain) :** Possibility of infective spondylodiscitis at L2-L3 level. Degenerative disc changes and prolapsed disc at multiple levels (L1-L5)

DIAGNOSIS

85-Year-old male patient k/c/o Diabetes Mellitus, Hypertension, Interstitial lung disease, Asthma, Ischemic Heart Disease with severely low LVEF of 15% with L1-L5 prolapsed disc posted for Spine Fixation

Anaesthetic Challenges :

1. Geriatric patient with Diabetes Mellitus and Hypertension

2. Multivalvular involvement and its complex pathophysiological changes- with low ejection fraction- which cardio supportive drug to use

3. Ventilation difficulties in view of Interstitial lung disease and asthma and compromised cardiac function

4. Negative Impact of prone position on already compromised cardiac function

5. Poor fluid tolerance

Anaesthesia Management and steps taken to overcome challenges:

- In view of low haemoglobin and poor tolerance to even minimal blood loss intraoperatively, we optimised the haemoglobin preoperatively to 12.3 gram/dl with due precaution to prevent volume overload
- Patient was nebulized with Duolin and Budecort thrice daily and on the morning of surgery
- Patient was given fitness under ASA 3

PREOPERATIVELY:

- Patient was explained about anaesthesia and associated risks
- Written and informed consent taken.
- All medications except oral hypoglycemic drugs and T.Valsartan were continued on the morning of surgery with sips of water.
- One 20 G and one 18 G IV canula was secured
- Left IJV was secured with 7 French Triple lumen central line using USG guidance under local anaesthesia.

- Patient was NBM for >8hours
- Preop Vitals : Pulse – 84/min, BP: 140/74 mmHg , SPO_2: 96%
- Fasting BSL checked – 110 mg/dL
- Adequate blood and blood products were reserved.

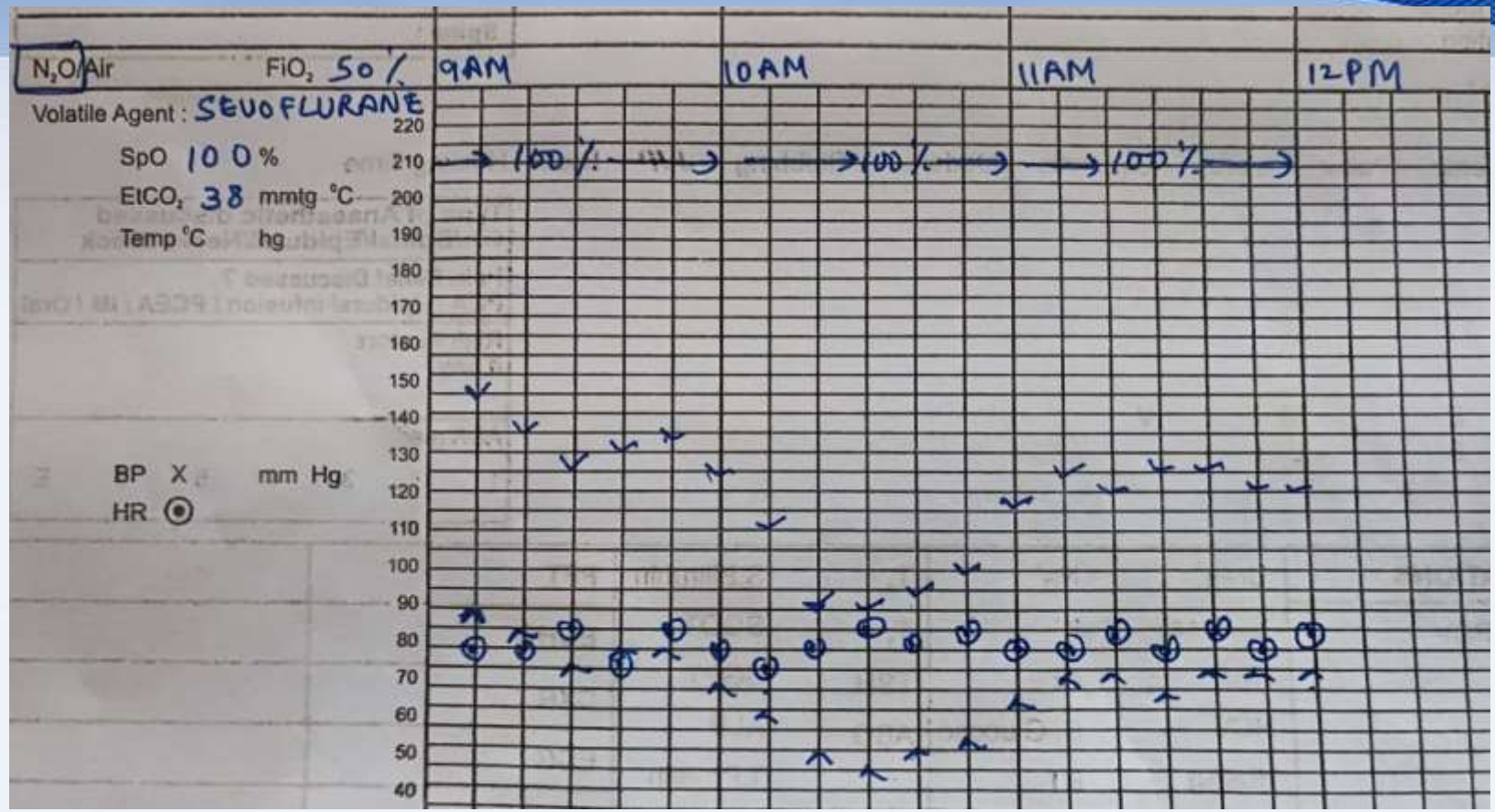
INTRAOPERATIVELY:

- All standard monitors like -Pulse oximeter, ECG, Noninvasive Blood pressure, End-tidal carbon dioxide, nasal temperature probe were attached.
- Elastocrepe bandage was applied on both lower limbs to prevent DVT and hypotension.
- Right radial arterial line was secured.
- All emergency drugs including Milrinone, Amiodarone, Adrenaline and Noradrenaline were kept ready.

- Patient was premedicated with Inj Midazolam 0.02 mg/Kg, Inj Fentanyl 1 µg/Kg, Inj Lignocaine 50 mg and Inj Ondansetron 0.1 mg/Kg.
- Preoxygenated for 3 min.
- Induction with Inj Etomidate 0.3 mg/Kg given slowly and Inj Succinylcholine 2mg/kg.
- Intubation was with 8.5 oral, cuffed, flexometallic tube.
- Muscle relaxant: Inj Cisatracurium 0.15mg/Kg
- Tube secured and throat pack inserted.
- Anaesthesia was maintained with $O_{2.}$ N_2O (50:50), sevoflurane and intermittent Inj Cisatracurium.

- Patient was made prone with due precaution to prevent IVC compression.

- In view of Asthma, low cardiac output and pulmonary fibrosis, expiratory time was kept longer (I: E ratio = 1:3) with low tidal volume, respiratory rate 18/min and PEEP zero.



- As patient was intolerant to even minimal blood loss, we started Infusion Norepinephrine (0.05mcg/kg/min) and minute-to-minute monitoring and balancing of input-output. Infusion Norepinephrine was required only for initial 45 min after which there was no requirement during surgery.

Procedure : L1-L5
Spine Fixation

Total Duration of
Surgery: 3 hours

- Intraoperatively total input was 760 ml (300 ml Crystalloid + 200 ml of colloid + 160 ml of FFP + 100 ml of PCV) and output was 600 ml (Blood loss 400 ml and Urine output 200 ml).
- Patient was made supine and reversal done with 2.5 mg Neostigmine and 0.4 mg of Glycopyrrolate.
- Patient was extubated after adequate tone and reflexes and response to verbal command.
- Post operative vitals - Pulse: 90/min, BP: 150/90 mmHg, SpO_2: 100 % on room air.
- Patient was shifted to CCM for observation.

DISCUSSION

- Even minimal blood loss in a compromised cardiac output may lead to precipitous hypotension. If this is associated with multivalvular involvement and compression of IVC during prone position, chances of precipitous hypotension increases multifold.

- That is why we optimised the haemoglobin to 12.3 gram/dl by transfusing two pints of PCV preoperatively with due precaution to prevent volume overload.

- Elastocrepe bandage was applied on both lower limbs to prevent DVT and hypotension.

- Due precaution was taken to prevent IVC compression in prone position

- In a trial comparing Epinephrine vs Norepinephrine, Norepinephrine was associated with lower occurrence of organ dysfunction, improved shock resolution and low mortality in cardiogenic shock [1] and a better balance between oxygen delivery and oxygen consumption in splanchnic area.

- One may think that an increase in blood pressure and thus afterload would rather be associated with decreased cardiac output. But it increases cardiac output as it acts on all four determinants of cardiac output i.e., Heart rate, preload, contractility and afterload.[2]

- Study by Montex et al showed that noradrenaline increased CVP, left ventricular end-diastolic area, global end-diastolic volume and cardiac index, the reason being noradrenaline increases preload due to venoconstriction causing redistribution of blood from unstressed to stressed volume.[3]

- Norepinephrine reserves vascular tone capacitance of vein, thereby decreasing the amount of fluid needed to restore venous return gradient which is not seen in milrinone. Milrinone causes vasodilatation which increases capacitance and may lead to precipitous hypotension. Hence more fluid may be required.[4]

- Further, most stroke volume could be used up to accommodate increased capacitance in an already compromised ejection fraction. However, we kept it ready to supplement norepinephrine if required.

- The advantage of the combination of a rise in arterial pressure, contractility and improved coronary perfusion improving ventriculoarterial coupling with noradrenaline again supported our choice to use this drug. [6]

Take home message,

Along with adequate preoperative optimisation, DVT prophylaxis, appropriate ventilatory setting, minute-to-minute monitoring and balancing of input-output, we suggest choice of vasopressor as norepinephrine and preoperative optimisation of haemoglobin in such cases.

REFERENCES:

1. Levy B, Clere-Jehl R, Legras A, et al. Epinephrine Versus Norepinephrine for Cardiogenic Shock After Acute Myocardial Infarction. *J Am Coll Cardiol* 2018;72:173-82. 10.1016/j.jacc.2018.04.051
2. Vincent JL, De Backer D. ICU nephrology: the implications of cardiovascular alterations in the acutely ill. *Kidney Int* 2012;81:1060-6. 10.1038/ki.2011.389
3. Monnet X, Jabot J, Maizel J, et al. Norepinephrine increases cardiac preload and reduces preload dependency assessed by passive leg raising in septic shock patients. *Crit Care Med* 2011;39:689-94. 10.1097/CCM.0b013e318206d2a3
4. Ayres JK, Maani CV. Milrinone. [Updated 2022 Sep 6]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 Jan-https://www.ncbi.nlm.nih.gov/books/NBK532943
5. Hamzaoui O, Jozwiak M, Geffriaud T, et al. Norepinephrine exerts an inotropic effect at the early phase of human septic shock. *Br J Anaesth* 2018;120:517-24. 10.1016/j.bja.2017.11.065


THANKYOU