

Anaesthetic Management of Subtrochanteric Femur Fracture in a Morbidly Obese Patient with Multiple Co-morbidities: A Case Report

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ABSTRACT

Morbid obesity, defined as Body Mass Index (BMI) greater than 40 kg/m², poses significant challenges for anaesthetic management, particularly in emergency orthopaedic procedures. The authors present the case of a 62-year-old morbidly obese female (BMI-58.76 kg/m²) with multiple co-morbidities including hypertension, diabetes mellitus, hypothyroidism, chronic kidney disease, and bronchial asthma, who presented with left closed displaced subtrochanteric femur fracture following a fall at home. The patient underwent Proximal Femoral Nailing (PFN) with Tension Band Wiring (TBW) under Combined Spinal-Epidural (CSE) anaesthesia after comprehensive preoperative optimisation for ten days. Preoperative management included correction of anaemia, electrolyte imbalances, glycaemic control, and respiratory optimisation. Intraoperatively, invasive arterial and central venous monitoring were established under ultrasound guidance. Low-dose spinal anaesthesia with 0.5% heavy bupivacaine (2 mL) was administered, supplemented by epidural catheter placement for postoperative analgesia. Sensory block was achieved to T10 dermatome with Bromage grade 2 motor block. The surgery was completed successfully in 125 minutes with stable haemodynamics maintained throughout. Postoperative pain management was achieved through multimodal analgesia including epidural tramadol administration. The present case highlights the importance of meticulous preoperative optimisation, appropriate selection of regional anaesthesia technique, and intensive perioperative monitoring in managing morbidly obese patients with multiple co-morbidities undergoing major orthopaedic surgery.

Keywords: Combined spinal-epidural anaesthesia, Haemodynamics, Perioperative management

CASE REPORT

A 62-year-old morbidly obese female presented to the Department of Orthopaedics with complaints of sudden onset, severe, continuous, dull aching pain in the left thigh and hip region following a fall at home one week prior. The pain was aggravated by movement and weight-bearing, with no relief in any position, and had progressively worsened since the injury, rendering her unable to ambulate. On examination, the patient was morbidly obese with height 165 cm, weight 160 kg, and BMI 58.76 kg/m² [Table/Fig-1]. The patient was classified as American Society of Anaesthesiologists (ASA) Physical Status III. Her medical history was significant for hypertension for 20 years (on tablet amlodipine 5 mg twice daily, hydrochlorothiazide 12.5 mg twice daily, and olmesartan 40 mg twice daily), diabetes mellitus for 20 years (on tablet metformin 500 mg twice daily, glimepiride 2 mg once daily, and teneligliptin 20 mg twice daily), hypothyroidism for 20 years (on tablet thyroxine 100 µg once daily), dyspnoea on exertion (Modified Medical Research Council (mMRC) dyspnoea grade 2) for one year, bronchial asthma (off-treatment for one year), and Chronic Kidney Disease (CKD stage 4, Estimated Glomerular Filtration Rate (eGFR) 22 mL/min/1.73m²). She had a history of breast abscess



[Table/Fig-1]: Preoperative photograph of the patient demonstrating morbid obesity (BMI 58.76 kg/m²).

incision and drainage under general anaesthesia 40 years ago without complications.

General examination revealed pulse 82/min, blood pressure 130/80 mmHg, oxygen saturation (SpO₂) 94% on room air and respiratory rate 18/min. Airway assessment showed mouth opening of three finger breadths, Mallampati grade 2, and normal neck extension and flexion. Systemic examination revealed bilateral air entry equal with no adventitious sounds, normal heart sounds, and the patient was conscious and oriented.

Initial investigations showed significant derangements including severe anaemia (haemoglobin 8.0 g/dL), azotaemia (serum creatinine 3.2 mg/dL, serum urea 266 mg/dL), severe hyponatraemia (serum sodium 120 mmol/L), hyperglycaemia, and respiratory alkalosis [Table/Fig-2].

Chest X-ray showed poor exposure due to obesity with bilateral haziness. High-Resolution Computed Tomography (HRCT) thorax revealed well-defined hypodense lesions in the superior basal segment of the right lower lobe. These were likely benign cysts or post-inflammatory changes; however, the differential diagnosis included bullae, infectious granuloma, or neoplastic lesion. Given the inability to perform contrast-enhanced CT due to impaired renal function, the pulmonology team was consulted, who opined these were likely benign based on morphology and stable clinical correlation, with no significant impact on ventilation-perfusion matching. Contrast-enhanced CT thorax was not possible due to altered renal function. Ultrasound of kidneys, ureters, and bladder showed minimal cortical scarring on the left kidney. Electrocardiogram showed poor progression of R wave in V1-V6. The poor R wave progression was attributed to the concentric left ventricular hypertrophy identified on echocardiography, rather than prior myocardial infarction, given the preserved ejection fraction of 60% and absence of regional wall motion abnormalities on two-dimensional echocardiography.

Parameters	Preoperative	Post-optimisation	Normal range
Haemoglobin (g/dL)	8.0	11.7	12.0-15.5
Total leucocyte count (cells/mm ³)	9100	-	4000-11000
Platelet count (Lakhs/mm ³)	3.8	-	1.5-4.0
PT/INR- Prothrombin Time (PT)/International Normalized Ratio (INR)	9.8/0.78	12.0/1.0	11-13.5/0.8-1.2
Random blood sugar (mg/dL)	169	Within normal	70-140
Serum urea (mg/dL)	266	102	7-20
Serum creatinine (mg/dL)	3.2	1.2	0.6-1.2
Serum sodium (mmol/L)	120	127	136-145
Serum potassium (mmol/L)	5.3	4.7	3.5-5.0
pH (ABG)	7.556	7.426	7.35-7.45
Po ₂ -Partial pressure of oxygen (mmHg)	106.2	193.9	80-100
PCO ₂ -Partial pressure of carbon dioxide (mmHg)	22.3	31.9	35-45
HCO ₃ (mmol/L)	22.3	21.7	22-26
TSH-Thyroid Stimulating Hormone (mIU/L)	4.145	-	0.4-4.0

[Table/Fig-2]: Comparison of preoperative and post-optimisation laboratory values.

Two-dimensional echocardiography revealed ejection fraction 60%, concentric left ventricular hypertrophy, and mild mitral, aortic, and tricuspid regurgitation.

Diagnosis was left closed displaced subtrochanteric femur fracture without distal neurovascular deficit. The patient was planned for left subtrochanteric femur PFN with TBW. In view of multiple co-morbidities and deranged parameters, preoperative optimisation was performed for ten days. Surgery was delayed to allow adequate correction of severe metabolic derangements (hyponatraemia, azotaemia, anaemia) that posed unacceptable perioperative risks. During this period, the fracture was stabilised with skin traction (5 kg) to provide pain relief and prevent further displacement. Thromboembolism prophylaxis was initiated with subcutaneous enoxaparin 40 mg once daily (dose-adjusted for renal function) and intermittent pneumatic compression devices.

Preoperative optimisation: The patient received one unit of packed red blood cell transfusion for correction of anaemia. Antihypertensive medications were changed to tablet metolazone 2.5 mg once daily, tablet nifedipine 20 mg thrice daily, and injection furosemide 20 mg thrice daily. Oral hypoglycaemic agents were switched to sliding scale insulin. Oxygen support at 4 L/min via nasal prongs was initiated. Daily nebulisation with ipratropium bromide 500 mcg + salbutamol 2.5 mg (Duolin) and budesonide 0.5 mg nebulisation was administered thrice daily. Fluid restriction of 1-1.5 L/day with salt-restricted diet was implemented, and serum electrolytes were monitored 12-hourly.

Following optimisation, repeat investigations demonstrated significant improvement in all deranged parameters [Table/Fig-2]. Although the serum sodium remained at 127 mmol/L, this represented chronic hyponatraemia with a 7 mmol/L improvement from the initial value of 120 mmol/L. Rapid correction was avoided to prevent osmotic demyelination syndrome. Importantly, despite the persistent hyponatraemia, the patient remained conscious and fully oriented throughout the preoperative period with no signs of confusion, disorientation, or altered sensorium, suggesting chronic adaptation to the low sodium levels. The decision to proceed with surgery was made after multidisciplinary consultation, weighing the risks of prolonged immobilisation (thromboembolism, pressure ulcers, hypostatic pneumonia) against the risks of moderate chronic hyponatraemia in a neurologically intact patient.

Intraoperative sodium levels were monitored and remained stable. The persistent mild respiratory alkalosis (pH-7.426, PCO₂-31.9 mmHg) was attributed to chronic respiratory compensation for underlying metabolic acidosis secondary to CKD, compounded by obesity-related restrictive pulmonary physiology causing relative hyperventilation, and was considered an acceptable baseline for this patient.

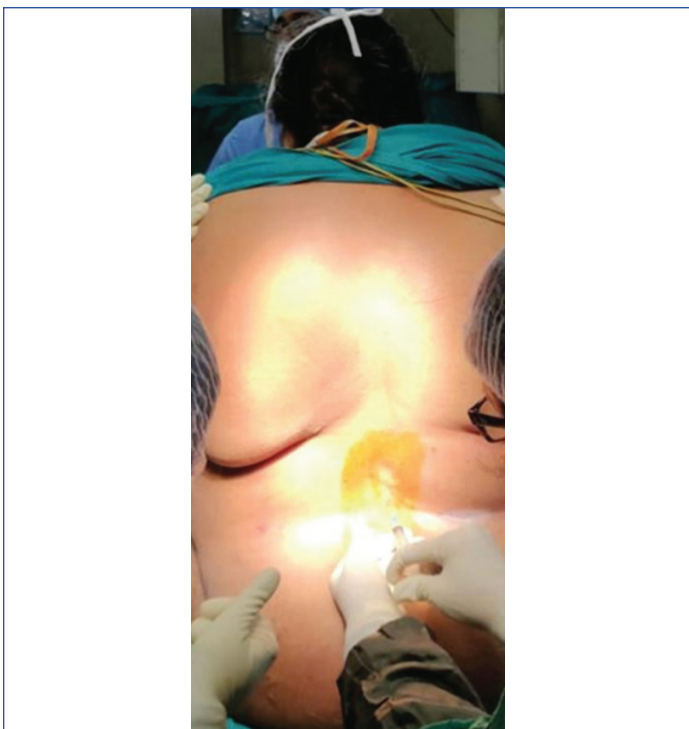
Anaesthetic management: The patient and relatives were explained about the anaesthetic risks in relation to her weight and co-morbidities, and high-risk consent was obtained (ASA grade 3). On the day of surgery, morning doses of antihypertensive medications (tablet metolazone 2.5 mg and tablet nifedipine 20 mg) and tablet thyroxine 100 mcg were administered, while the morning dose of insulin was withheld. Fasting blood sugar and serum electrolytes were within acceptable limits, and serum acetone was absent.

In the operating theatre, appropriate tables were prepared considering the patient's weight. Peripheral intravenous access was secured with 20G cannula on the dorsum of the left hand. Standard monitors including electrocardiogram, non-invasive blood pressure, pulse oximetry, and nasal temperature probe were attached. A warming device was utilised to prevent hypothermia, and urinary catheter was inserted for urine output monitoring.

Under local anaesthesia, invasive arterial line was placed in the right radial artery for continuous blood pressure monitoring and arterial blood gas sampling. Central venous catheter was inserted in the right internal jugular vein under ultrasound guidance for fluid management and vasopressor administration. The risk of performing central neuraxial blockade in the setting of hyponatraemia (Na-127 mmol/L) was carefully considered. The lowered seizure threshold associated with hyponatraemia increases vulnerability to Local Anaesthetic Systemic Toxicity (LAST). To mitigate this risk, a low-dose spinal technique was employed, intravenous lipid emulsion (Intralipid 20%) was kept readily available, and serum sodium was monitored intraoperatively. Epidural space was located at L2-L3 intervertebral space using the hanging drop method, and an epidural catheter was inserted under aseptic precautions. The image demonstrates the technical challenges encountered in morbidly obese patients, where excessive subcutaneous adipose tissue obscures bony landmarks of the spine, making palpation of spinous processes extremely difficult and spinal anaesthesia administration technically challenging [Table/Fig-3].

Spinal anaesthesia was administered at L3-L4 subarachnoid space using a 23G spinal needle. Low-dose 0.5% heavy bupivacaine (2 mL) was given with relative ease. The patient was immediately returned to supine position for 15 minutes with head elevation to prevent the dermatomal level from exceeding the required limit. Sensory block was assessed by pin-prick method and a bilateral T10 level was achieved within 10 minutes. Motor block assessment showed Bromage grade 2 (able to flex knees but unable to lift legs). Surgical anaesthesia was confirmed within 15 minutes of spinal injection. No supplemental sedation or analgesia was required during the procedure. Subsequently, the patient was positioned in the right lateral position for surgery [Table/Fig-4]. All pressure points were carefully padded to avoid pressure sores and neurological injury.

Goal-directed fluid therapy was employed based on central venous pressure readings, estimated blood loss, and urine output with strict input-output charting. Vasopressor support with injection noradrenaline infusion at 0.02-0.05 µg/kg/min was titrated to maintain mean arterial pressure above 65 mmHg. Injection noradrenaline bolus was kept available for use if blood pressure fell below 20% of baseline. Intraoperatively, vital signs remained stable with mean arterial pressure maintained between 75-85 mmHg, heart rate-78-88/min, and SpO₂-96-98% on supplemental oxygen via face mask. Estimated blood loss was approximately 350 mL. A total of 1000 mL of Ringer's lactate and 500 mL of normal saline were administered. Urine output was maintained at approximately



[Table/Fig-3]: Administration of Combined Spinal-Epidural (CSE) anaesthesia in the sitting position.



[Table/Fig-4]: Intraoperative positioning showing patient in right lateral position with C-arm fluoroscopy, invasive monitoring lines, and pressure point padding.

0.5 mL/kg/hour. Body temperature was maintained between 36.2-36.8°C with the use of forced-air warming device. Intraoperative arterial blood gas showed pH-7.38, PO₂-165 mmHg, PCO₂-34.2 mmHg, HCO₃⁻-20.1 mmol/L, and serum sodium-128 mmol/L. The surgery was completed in 125 minutes with stable haemodynamics throughout.

Postoperative: The patient was shifted to the post-anaesthesia care unit and monitored for 24 hours. Haemodynamics remained stable throughout. Respiratory monitoring in the Post Anaesthesia Care Unit (PACU) showed SpO₂ of 95-97% on supplemental oxygen at 2 L/min via nasal prongs, with no episodes of desaturation or bronchospasm. Postoperative analgesia was provided using a multimodal approach: epidural catheter with injection tramadol 50 mg in 10 mL normal saline administered every eight hours, intravenous paracetamol 1 g every six hours, and rescue analgesia with intravenous fentanyl 25 mcg as needed. The Visual Analogue Scale (VAS) was explained preoperatively for appropriate pain assessment. VAS scores were maintained at 2-3 at rest and 4-5 on movement during the first 24 hours. The epidural catheter was removed after 24 hours with the blue tip intact. Deep vein thrombosis prophylaxis was continued with subcutaneous enoxaparin 40 mg once daily and intermittent pneumatic compression devices.

Bedside sitting was initiated on Postoperative Day (POD) 1, and assisted ambulation with a walker was commenced on POD 3. The patient's pain was well controlled with VAS scores remaining below 4 at rest throughout the hospital stay. No major complications such as wound infection, deep vein thrombosis, pulmonary embolism, or respiratory complications were encountered. Serum creatinine

at discharge was 1.4 mg/dL, and serum sodium had improved to 131 mmol/L. The total hospital stay was 14 days. At 6-week follow-up, the patient showed satisfactory fracture healing on radiographs and was ambulating independently with a walker. Renal function had returned to near-baseline levels with serum creatinine of 1.3 mg/dL.

DISCUSSION

The global prevalence of obesity has reached epidemic proportions, with morbid obesity (BMI >40 kg/m²) presenting unique challenges for anaesthesiologists [1]. Obesity predisposes patients to multiple chronic diseases including peripheral vascular disease, cardiorespiratory derangements, liver disease, diabetes mellitus, diaphragmatic hiatus hernia, polycythaemia, and malignancies [1]. The anaesthetic management of morbidly obese patients requires careful consideration of altered pharmacokinetics, difficult airway management, and increased perioperative complications [2]. In the present case, a 62-year-old morbidly obese female with BMI 58.76 kg/m² and five major co-morbidities (hypertension, diabetes mellitus, hypothyroidism, CKD, and bronchial asthma) was successfully managed with CSE anaesthesia for PFN. The use of reduced local anaesthetic dose (2 mL of 0.5% heavy bupivacaine) achieved adequate surgical anaesthesia to T10 while minimising haemodynamic disturbances, consistent with the recommendation that morbidly obese patients require 20-25% less local anaesthetic due to epidural fat and distended epidural veins [3,4]. Regional anaesthesia offers several advantages in obese patients, including minimal airway intervention, reduced cardiopulmonary depression, improved postoperative analgesia, decreased opioid consumption, and reduced postoperative nausea and vomiting [3]. However, regional anaesthesia can be technically challenging in obese patients due to difficulties in patient positioning, identifying anatomical landmarks, and determining appropriate needle penetration depth [3].

Several case reports and series have documented the anaesthetic management of morbidly obese patients undergoing orthopaedic procedures. Ingrande and Lemmens reported that CSE anaesthesia provides the most reliable block in the obese population, with the spinal component ensuring rapid surgical anaesthesia and the epidural catheter allowing dose titration and extended analgesia [3]. In the present case, the CSE technique proved similarly effective, with the added advantage of avoiding general anaesthesia in a patient with multiple risk factors for difficult airway management. De Jong A et al., highlighted that morbidly obese patients undergoing emergency orthopaedic procedures have a significantly higher risk of respiratory complications when managed under general anaesthesia, further supporting the choice of CSE in the current case [4].

The anaesthetic challenges in morbidly obese patients are multifaceted. Several factors increase perioperative risk in morbidly obese patients, including rapid oxygen desaturation, predisposition to deep vein thrombosis, sleep apnoea, and regurgitation and aspiration [2]. In diabetic patients, the aim of intraoperative management is to provide adequate anaesthesia with proper positioning while avoiding hypoglycaemia, hyperglycaemia, ketoacidosis, and electrolyte disturbances [5]. Regional anaesthesia in CKD requires assessment of coagulation adequacy and exclusion of uraemic neuropathies [6]. Co-existing metabolic acidosis may decrease the seizure threshold for local anaesthetics. In patients with asthma, there is increased risk of desaturation, metabolic acidosis, and bronchospasm [7]. Obese patients with long bone fractures have a high predisposition for complications such as compartment syndrome, nerve injuries, pressure ulcers, fat embolism, and pulmonary embolism [2].

The management of severe hyponatraemia in the patient deserves special mention. The initial serum sodium of 120 mmol/L was likely multifactorial, secondary to dilutional hyponatraemia from CKD-related fluid retention, thiazide diuretic use (hydrochlorothiazide), and

possible Syndrome of Inappropriate Antidiuretic Hormone (SIADH). Management with fluid restriction (1-1.5 L/day), discontinuation of hydrochlorothiazide, and switching to metolazone resulted in gradual correction to 127 mmol/L over ten days. Rapid correction was deliberately avoided to prevent osmotic demyelination syndrome, with a target correction rate of <8 mmol/L per 24 hours as per current guidelines [8]. The dramatic improvement in renal function (serum creatinine from 3.2 to 1.2 mg/dL) was attributed to correction of prerenal factors including optimisation of volume status, discontinuation of nephrotoxic medications, and avoidance of contrast agents. This suggests a significant prerenal component to the initial renal impairment. Regarding the persistent respiratory alkalosis, this was likely a combination of chronic hyperventilation secondary to obesity hypoventilation pattern and compensatory response to CKD-related metabolic acidosis. Current Kidney Disease: Improving Global Outcomes (KDIGO) guidelines recommend goal-directed haemodynamic management with avoidance of nephrotoxic agents and maintenance of adequate renal perfusion pressure as the mainstay of perioperative renal protection [9].

Several alternative anaesthetic strategies were considered for this patient. General anaesthesia was deemed high-risk due to anticipated difficult airway (morbid obesity, Mallampati grade 2), increased risk of aspiration, postoperative respiratory complications, and the need for large-dose opioids. Peripheral nerve block alone (fascia iliaca or femoral nerve block) was inadequate for the proximal extent of surgery involving the subtrochanteric region. Epidural anaesthesia alone was considered but carries the risk of slower onset, patchy block, and unpredictable spread in obese patients. The CSE technique was ultimately chosen as it combined the reliability and rapid onset of spinal anaesthesia with the flexibility of epidural catheter for dose supplementation and postoperative analgesia, representing the optimal balance of efficacy and safety for this high-risk patient [3].

CONCLUSION(S)

The present case demonstrates successful anaesthetic management of a morbidly obese patient with multiple co-morbidities using low-dose spinal anaesthesia combined with epidural anaesthesia. Key factors contributing to the successful outcome included comprehensive preoperative optimisation, careful selection of regional anaesthesia technique, intensive haemodynamic monitoring with invasive arterial and central venous access, judicious fluid management, and effective postoperative pain control through multimodal analgesia. Meticulous attention to thromboembolism prophylaxis, early mobilisation, and close monitoring of metabolic parameters were equally crucial. This approach can be considered for similar high-risk patients undergoing orthopaedic procedures.

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