

Intraoperative Acute Bronchospasm Mimicking Acute Chest Syndrome in a Patient with Sickle Cell Disease Undergoing Laparoscopic Cholecystectomy: A Case Report

NIKITA SAREEN¹, JAYASHREE SEN², MONIKA SAREEN³, MANOJ SAREEN⁴, DHWANI SHETH⁵

ABSTRACT

Sickle Cell Disease (SCD) is associated with significant perioperative morbidity due to an increased risk of hypoxia, acidosis, dehydration, and stress-induced vaso-occlusive crisis. Laparoscopic procedures may further exacerbate these risks through pneumoperitoneum-induced respiratory and haemodynamic alterations. Intraoperative bronchospasm in patients with SCD is uncommon and may mimic or precipitate Acute Chest Syndrome (ACS), creating diagnostic and therapeutic challenges for the anaesthesia team. The present case is of a 59-year-old female with known SCD who presented with abdominal pain and was diagnosed with cholelithiasis, choledocholithiasis, a probable benign distal Common Bile Duct (CBD) stricture, and mild hepatosplenomegaly. Preoperative optimisation included correction of anaemia, Endoscopic Retrograde Cholangiopancreatography (ERCP), and CBD stenting. She subsequently underwent elective laparoscopic cholecystectomy under general anaesthesia. Shortly after Carbon Dioxide (CO₂) insufflation and placement in the head-low position, she developed a sudden rise in End-Tidal Carbon Dioxide (EtCO₂), increased peak airway pressures, and bilateral wheezing, consistent with severe intraoperative bronchospasm. Prompt management with bronchodilators and ventilatory optimisation was initiated; however, owing to persistent respiratory compromise, the procedure was converted to open cholecystectomy. The patient recovered uneventfully with postoperative supportive care, underwent successful CBD stent removal and transfusion, and was discharged in stable condition. This case is unique because severe intraoperative bronchospasm occurring during laparoscopic cholecystectomy in a patient with SCD closely simulated complications such as ACS and vaso-occlusive events. The report highlights the importance of early recognition, prompt airway management, careful intraoperative monitoring, and multidisciplinary collaboration to ensure favourable outcomes in this high-risk patient population.

Keywords: Airway pressure, General anaesthesia, Hypercarbia, Hypoxia, Pneumoperitoneum

CASE REPORT

A 59-year-old female patient was admitted to the Department of General Surgery with complaints of severe, continuous, aching pain in the right upper abdomen for the past four months. The pain typically occurred after meals, radiated to the right shoulder, and was associated with nausea and vomiting, lasting 6-8 hours and aggravated by movement since four months and she was subsequently planned for elective cholecystectomy. She had been diagnosed with SCD since infancy for which she was taking hydroxyurea capsules (750 mg/day orally once daily) and folic acid (1 mg once daily) tablets. No history of diabetes mellitus, tuberculosis, bronchial asthma, hypertension or major allergies was present. There was no noteworthy history of any addictions.

The patient was afebrile, cooperative, and cognisant during general examination. Her BMI was 23.0 kg/m²; blood pressure was 140/80 mmHg, respiration rate was 18 breaths per minute, and pulse rate was a steady 60 bpm. There was no evidence of elevated jugular venous pressure, icterus, oedema, or pallor. A cardiovascular examination showed that S1 and S2 were normal and no murmur was heard. Bilateral vesicular breath sounds were heard during the respiratory system assessment without any added sounds. The central nervous system testing came out to be normal.

A mouth opening of three fingers, Mallampati score 2 and an adequate range of neck and Temporomandibular Joint (TMJ) mobility were all recorded during airway evaluation. No major airway limitations were expected. Based on presence of SCD, the patient was categorised as American Society of Anaesthesiologists (ASA) Grade II and was judged suitable for surgery with the appropriate perioperative care.

A laboratory analysis showed anaemia with a haemoglobin level of 7.6 g/dL. The platelet count was 1.85 lakh/mm³, and the total leucocyte count was 9,000/mm³. Coagulation profile showed Prothrombin Time (PT) to be 12.1 seconds and Activated Partial Thromboplastin Time (APTT) to be 30.3 seconds with an International Normalised Ratio (INR) value of 1.01. Her blood group was B Rh positive with late sickling positive with SS pattern and Direct and Indirect Coombs tests were negative.

Because of anaemia, the patient was given a Packed Red Cell (PRC) transfusion preoperatively with no premedications given prior. During transfusion, she developed a febrile reaction with chills and rigours. Intravenous hydrocortisone (100 mg i.v. STAT) supportive measures were used to manage the transfusion reaction after it was temporarily stopped. Microscopic examination of the peripheral blood smear revealed predominantly normocytic, normomorph erythrocytes with occasional sickle cells and no significant evidence of active haemolysis or sickling. Transfusion was restarted later under careful observation following stabilisation. No other transfusion reactions were observed with the careful administration of one more subsequent transfusion. Chest radiography showed a normal cardiothoracic ratio without focal consolidation, pleural effusion, or pneumothorax.

Using T1- and T2-weighted axial, sagittal, and coronal images, Magnetic Resonance Cholangiopancreatography (MRCP) showed a well-distended gallbladder with few calculi that were around 4-5 mm in size. There was no sign of cholecystitis or pericholecystic collection. There was no indication of intrahepatic biliary radical dilatation or calculi, and the whole CBD was dilated, with a maximal

diameter of 8.2 mm, suggesting a likely benign stricture at the ampulla. Mild hepatosplenomegaly was indicated by the slightly enlarged liver (15.6 cm) and spleen (12.3 cm) that showed normal shape and signal intensity. The size and shape of the kidneys and adrenal glands were both normal.

The endoscopic examination revealed normal stomach and oesophageal mucosa. In the duodenum, periampullary diverticulum was seen. The gallbladder was enlarged and the cystic duct was severely dilated.

The results of ERCP and abdominal ultrasound were indicative of cholelithiasis with choledocholithiasis. There was mild hepatosplenomegaly. T-wave inversion was seen in lead V1 of the ECG. Preoperative optimisation included correction of anaemia, arrangement of blood products, obtaining high-risk informed consent and nil-per-oral guidelines with fasting for solids for eight hours and allowance of clear liquids up to two hours before surgery.

The patient subsequently underwent elective laparoscopic cholecystectomy after initial stabilisation and completion of antibiotic therapy under general anaesthesia. After obtaining informed written consent, the patient was shifted to the operating room and standard monitoring including ECG, non-invasive blood pressure and pulse oximetry was applied. After preoxygenation with 100% oxygen for three minutes; premedications - Inj. glycopyrrolate 0.2 mg i.v. and Inj. midazolam (1 mg i.v.) were given. General anaesthesia was induced with intravenous propofol (2 mg/kg), analgesia was provided with fentanyl (2 µg/kg). Neuromuscular blockade was achieved using vecuronium (0.1 mg/kg) to facilitate endotracheal intubation. Anaesthesia was maintained with a mixture of oxygen, air and sevoflurane with intermittent doses of vecuronium for muscle relaxation. EtCO₂ was kept within normal range by starting mechanical ventilation in a volume-controlled mode. CO₂ was insufflated to create pneumoperitoneum and patient was placed in head low position as per the Surgeon's requirement. At the same time, delivered tidal volumes decreased, peak airway pressures increased, and later auscultation revealed audible wheeze. The mode of ventilation was changed from volume-controlled ventilation to pressure-controlled ventilation to limit airway pressures and optimise ventilation. The patient presented with hypotension and tachycardia and oxygen saturation demonstrated a downward trend. Intraoperatively, monitor showed heart rate to be 101 beats per minute, SpO₂: 96%, EtCO₂: 45 mmHg, respiratory rate 22 breaths per minute and blood pressure -117/55 mmHg.

Acute bronchospasm, elevated CO₂ absorption, endotracheal tube blockage or migration, CO₂ embolism, and early ACS were among the differential diagnoses taken into consideration. The patient was immediately manually-ventilated to ensure adequate ventilation.

Arterial Blood Gas (ABG) analysis indicated suboptimal ventilation. consequently, the operating table was returned to the supine position and pneumoperitoneum was released to improve respiratory parameters. The patient was immediately managed by deepening the level of anaesthesia, administering intravenous hydrocortisone (100 mg/i.v.) and dexamethasone (8 mg/i.v.), delivering salbutamol (4-8 puffs, 100 µg/puff) via a metered-dose inhaler with its mouth piece connected to the endotracheal tube connector, and administering intravenous deriphylline (2 mL given over 15-20 minutes, slowly). Despite partial improvement, persistent ventilatory difficulty necessitated a timely decision to convert the procedure from laparoscopic to open cholecystectomy. Following desufflation and conversion, ventilation improved significantly, wheezing subsided, and airway pressures and EtCO₂ gradually returned to normal [Table/Fig-1].

Intraoperatively, the gallbladder was found to be distended with multiple calculi, without evidence of acute inflammation. A standard four-port laparoscopic approach was initiated; however, persistent

Parameters	During bronchospasm	Repeat assessment	Reference range
pH	7.41	7.42	7.35-7.45
pCO ₂ (mmHg)	62.6	44	35-45
pO ₂ (mmHg)	162	151	80-100
Oxygen Saturation (SpO ₂) (%)	99.4	100	95-100
HCO ₃ ⁻ (mmol/L)	36.3	27.7	22-26
Base excess (mmol/L)	+14.2	3.9	-2 to +2

[Table/Fig-1]: Serial Arterial Blood Gas (ABG) analysis during and after intraoperative bronchospasm.

bronchospasm with rising airway pressures necessitated conversion to an open cholecystectomy. The cystic duct and cystic artery were identified, ligated, and divided, and the gallbladder was removed through a right subcostal incision. Haemostasis was achieved, and the abdomen was closed in layers.

The postoperative course was uneventful, with gradual improvement in respiratory and haemodynamic status. She was discharged in stable condition and remained asymptomatic during 15 days of follow-up. Bronchodilators, corticosteroids, and supportive care were continued in the postoperative period. Following surgery, another PRC transfusion was given. A pulmonology consultation was sought postoperatively. There was a noticeable clinical improvement over time. Abdominal drains were withdrawn one after the other while the surgical incisions stayed intact. After successful removal of the CBD stent and transfusion of packed red blood cells, she remained clinically stable, ambulatory, and tolerated oral feeds well.

DISCUSSION

In the present case, a 59-year-old SCD patient experienced acute, severe bilateral bronchospasm immediately following CO₂ pneumoperitoneum and Trendelenburg positioning during laparoscopic cholecystectomy. The patient's symptoms included wheezing, elevated peak airway pressures, and an abrupt increase in EtCO₂. Rapid improvement after bronchodilator therapy, desufflation, and conversion to open surgery, along with normal ventilatory parameters on a repeat ABG analysis [1]. This distinction is clinically important because bronchospasm, ACS, pulmonary embolism, mucus plugging, endobronchial intubation, and other pulmonary vaso-occlusive complications can produce overlapping respiratory manifestations in patients with SCD. As hypoxia, acidosis, hypothermia, dehydration, and surgical stress can produce sickling and pulmonary problems, current perioperative literature also emphasises careful monitoring of oxygenation, ventilation, acid-base status, temperature, and intravascular volume [2].

This patient's respiratory decline specifically followed pneumoperitoneum and Trendelenburg positioning, indicating that the incident may have been caused by changed respiratory mechanics related to laparoscopy. While pneumoperitoneum can raise intra-abdominal pressure and hinder diaphragmatic excursion, and placement can further change pulmonary mechanics, laparoscopic surgery is generally regarded as safe in SCD when the proper measures are performed. In SCD surgery series, similar issues have been documented. Careful control of hypovolemia, hypothermia, acidosis, and discomfort was stressed in a prospective study of 20 children undergoing laparoscopic cholecystectomy; yet, two patients developed ACS after the procedure [3]. Similarly, Vecchio R et al., reported satisfactory outcomes with extensive perioperative monitoring, hydration, temperature control, and selective transfusion in a series of seven people with SCD having laparoscopic cholecystectomy; just one patient experienced lower-limb pain after surgery [4]. These results support the use of laparoscopy in patients who are suitably optimised, but they also highlight the necessity of promptly identifying respiratory impairment, as the current instance does.

Since ACS continues to be one of the most dangerous postoperative pulmonary consequences of SCD, it was a crucial differential diagnosis. According to a recent comprehensive study by Khalaf Z and Mahmood M postoperative desaturation, pulmonary infiltrates, and respiratory symptoms were typical signs of ACS, which happened in 4-23% of SCD patients after abdominal surgery. Crucially, the research discovered contradictory data about whether open or laparoscopic surgery increases the risk of ACS [2]. Diarra B et al., reported significantly higher SCD-related morbidity following laparoscopic compared with open cholecystectomy (10.5% vs 4.5%, p-value=0.04), with a significantly higher incidence of ACS in the laparoscopic group (8.5% vs 1.1%, p-value=0.01). These findings suggest that laparoscopic cholecystectomy may carry an increased risk of ACS in patients with SCD [5].

A recent 2026 case report highlighted the diagnostic challenge of ACS in SCD, where VOC-associated ACS following a distal femur fracture mimicked aspiration pneumonitis, emphasising the importance of early differentiation from other causes of respiratory compromise. Similarly, the present case demonstrates that acute bronchospasm during pneumoperitoneum may mimic ACS, underscoring the need for careful assessment of clinical findings, timing, airway pressures, capnography, and response to treatment [6].

The current patient's perioperative care followed to the guidelines advised for high-risk SCD patients. When ventilatory compromise persisted, desufflation and conversion to open surgery were employed in addition to bronchodilator treatment, deepening of anaesthesia, intravenous hydrocortisone, and deriphylline. Because of their bronchodilatory qualities, adjunctive medications such as intravenous magnesium sulphate and ketamine may be taken into consideration in cases of severe or refractory bronchospasm; however, in this instance, neither was necessary because the patient responded to standard treatment. Current perioperative guidelines highlight the significance of preventing hypoxia, hypothermia, acidosis, dehydration, and excessive fluid delivery. Individualised fluid therapy is more important than indiscriminate hyperhydration because excessive intravenous fluid administration itself may worsen pulmonary oedema and lead to ACS [1]. Similar to standard transfusion, risk-based transfusion is better; choices are made based on baseline haemoglobin, the severity of the disease, surgical procedure and the expected blood loss [1,2].

Lastly, the current case shows that minimally invasive surgery does not completely eliminate the respiratory hazards associated with SCD, despite the fact that laparoscopic cholecystectomy is still a desirable option because to less postoperative pain, earlier mobilisation, and shorter recovery. De'Angelis N et al., found that patients having mini-laparoscopy with low, stable-pressure pneumoperitoneum had no ACS, while the incidence of ACS in the laparoscopic cholecystectomy group was 1.6% [7]. The

current case thus highlights the significance of customised surgical planning, close communication between the anaesthesia team and the surgeon, ongoing capnography and airway-pressure monitoring, early ABG assessment, and a low threshold for desufflation or conversion to open surgery when respiratory mechanics worsen. To find out if low-pressure pneumoperitoneum, improved laparoscopic procedures, and standardised perioperative respiratory protocols can lower intraoperative pulmonary problems in individuals with SCD, more prospective research is required.

CONCLUSION(S)

In SCD patients, intraoperative bronchospasm can cause hypoxia, increasing EtCO₂, increased airway pressures, and respiratory distress, similar to vaso-occlusive crisis or early ACS. Because some conditions have overlapping clinical indications, misunderstanding might cause delays in receiving proper therapy. Rapid detection of bronchospasm requires thorough monitoring of ventilatory parameters and auscultation. Immediate airway optimisation, administration of bronchodilators and corticosteroids, appropriate oxygenation, and correction of triggering variables, as well as coordinated input from anaesthesia, surgery, and pulmonology teams, are critical to avoiding major complications and ensuring positive results.

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REFERENCES

- [1] Adjepong KO, Otegbeye F, Adjepong YA. Perioperative management of sickle cell disease. *Mediterr J Hematol Infect Dis*. 2018;10(1):e2018032. Published 2018 May 1. Doi: 10.4084/MJHID.2018.032.
- [2] Khalaf Z, Mahmood M. Acute chest syndrome in post-operative sickle cell disease patients: A systematic review of predisposing factors and interventions. *Surg Pract Sci*. 2022;11:100143. Published 2022 Nov 12. Doi: 10.1016/j.sipas.2022.100143.
- [3] Ndoye MD, Bah MD, Pape IN, Diouf E, Kane O, Bèye M, et al. Prise en charge périopératoire de la cholécystectomie par voie laparoscopique chez l'enfant drépanocytaire homozygote [Perioperative management of laparoscopic cholecystectomy in children with homozygous sickle cell disease]. *Arch Pediatr*. 2008;15(9):1393-97. French. Doi: 10.1016/j.arcped.2008.06.012.
- [4] Vecchio R, Cacciola E, Murabito P, Gambelunghe AV, Murabito R, Cacciola RR, et al. Laparoscopic cholecystectomy in adult patients with sickle cell disease. *G Chir*. 2001;22(1-2):45-48.
- [5] Diarra B, Roudié J, Coulibaly A, Somian FE, Kanga-Miessan JB, Franco D. Cholecystectomy in sickle cell disease patients: Is there more acute chest syndrome after laparoscopy? A case controlled study. *Int J Surg*. 2008;6(3):220-23. Doi: 10.1016/j.ijsu.2008.03.009.
- [6] Chethan HK, Panda CK, Sainath J, Patel S, Agarwal G. Acute chest syndrome with vaso-occlusive crisis in a sickle cell disease patient: A mimic of aspiration pneumonia. *Indian J Crit Care Case Rep*. 2026;5(2):38-40. Doi: 10.5005/jp-journals-11006-0212.
- [7] De'Angelis N, Abdalla S, Carra MC, Luzzi V. Low-impact laparoscopic cholecystectomy is associated with decreased postoperative morbidity in patients with sickle cell disease. *Surg Endosc*. 2018;32(10):1-12. Doi: 10.1007/s00464-017-5925-y.

PARTICULARS OF CONTRIBUTORS:

1. Junior Resident, Department of Anaesthesia, DMIHER, Sawangi, Wardha, Maharashtra, India.
2. Professor, Department of Anaesthesia, DMIHER, Sawangi, Wardha, Maharashtra, India.
3. Consultant, Department of Obstetrics and Gynaecology, Pushpanjali Hospital, Delhi, India.
4. Consultant, Department of Pathology, Jaipur Golden Hospital, Delhi, India.
5. Junior Resident, Department of Anaesthesia, DMIHER, Sawangi, Wardha, Maharashtra, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:

Dr. Nikita Sareen,
Tower V - 036, CGV DLF New Moti Nagar, Shivaji Marg, Delhi-110015, India.
E-mail: nikitasareen1997@gmail.com

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