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CASE REPORT

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Negative pressure pulmonary edema resulting from upper airway obstruction during the post-anesthesia recovery period: a case series and literature review

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Abstract

Introduction Negative pressure pulmonary edema (NPPE) is a non-cardiogenic pulmonary edema primarily resulting from upper airway obstruction, particularly following laryngospasm during the post-anesthesia recovery period.

Cases presentation We report five cases of NPPE that occurred during recovery from general anesthesia at our institution. Among these, four cases were attributed to glossoptosis following extubation, while one case resulted from laryngeal mask displacement. All patients exhibited typical clinical features and imaging findings consistent with pulmonary edema, and none experienced poor prognoses.

Conclusions Glossoptosis and improper positioning of the laryngeal mask during the recovery phase of anesthesia pose a risk for inducing NPPE.

Keywords Negative pressure pulmonary edema, General anesthesia, Glossoptosis, Laryngeal mask, Airway obstruction

Background

Negative pressure pulmonary edema (NPPE) is defined as an acute respiratory failure that results from increased negative intrathoracic pressure due to upper airway obstruction [1]. Various conditions may precipitate NPPE, including epiglottitis, upper airway tumors, and laryngospasm [2]. In adults, airway obstruction leading to NPPE is most frequently associated with laryngospasm occurring after extubation [3]. This paper presents several cases of NPPE that arose during recovery from general anesthesia at our institution, specifically linked to glossoptosis and laryngeal mask displacement. Despite the low incidence of NPPE, its associated mortality rate can be significantly high if not diagnosed and treated promptly, underscoring the need for heightened awareness among anesthetists. Furthermore, a review of the clinical characteristics documented in the literature over the past five years is provided to enhance the understanding of this condition among clinicians.

Cases presentations

The Table 1 and Fig. 1 summarize the characteristics of the cases. Ethical approval (approval number, KY2024-061–01) was provided by the Ethical Committee of Sichuan Province Orthopaedic Hospital, Sichuan, China. Written informed consent for publication was obtained

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Table 1 Summary of clinical characteristics

Case	1	2	3	4	5
Age (years)	54	37	66	41	50
Gender	F	M	M	M	M
BMI (kg/m^2)	18.3	26.3	28.4	22.7	24.8
Setting	LMA	LMA	LMA	LMA	ETI
Habitus	Depression	Snoring	/	/	Snoring
Presenting symptoms	Glossoptosis, delayed recovery, bloody cough	Glossoptosis, bloody cough	LMA displacement, dyspnea, pink secretions	Glossoptosis, dyspnea, bloody cough	Glossoptosis, bloody cough
SpO_2 (%)	89	78	80	86	80
Auscultation	Wet rales	Wet rales	Wet rales	Wet rales	Wet rales
CT scan	Bilateral extensive infiltrate and alveolar filling ground glass	Bilateral extensive infiltrate and alveolar filling ground glass	Bilateral extensive infiltrate and alveolar filling ground glass	Bilateral extensive infiltrate and alveolar filling ground glass	Bilateral extensive infiltrate and alveolar filling ground glass
Time to resolution (h)	24	72	48	24	24
Treatment	Oxygen, CS, diuretic	Oxygen, nasopharyngeal airway, diuretic, CS, prone position	Intubation PPV, oxygen, CS, diuretic	Oxygen, nasopharyngeal airway, CS, prone position	Oxygen, CS, non-invasive ventilator-assisted ventilation, diuretic

Abbreviations: *F* Female, *M* Male, *BMI* Body mass index, *LMA* Laryngeal mask airway, *ETI* Endotracheal intubation, *SpO2* Oxygen saturation, *CT* Computed tomography, *CS* Corticosteroids

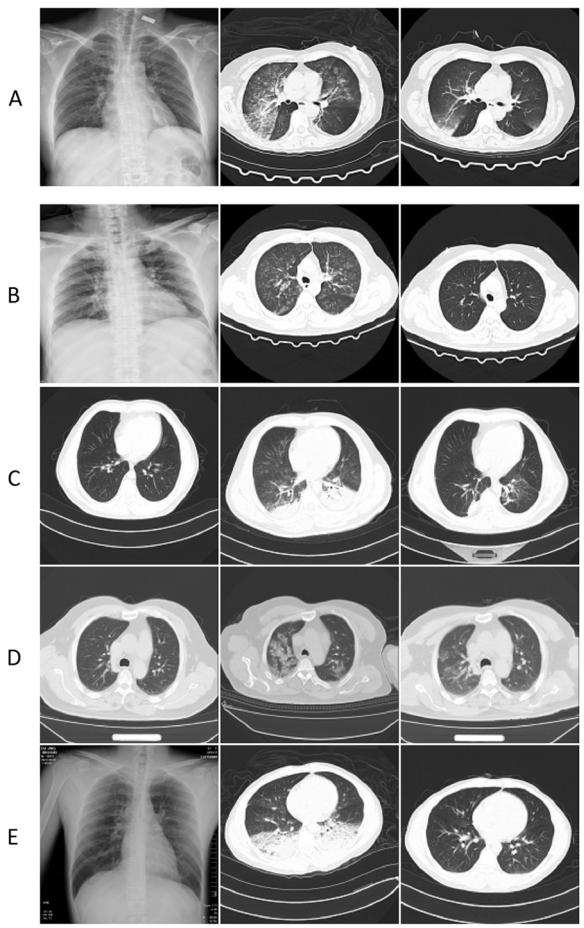

Fig. 1 Summary of the progression of chest imaging

from every patient for the publication of the date, images, etc.

Case 1

A 54-year-old female patient underwent intramedullary pin fixation for an intertrochanteric femur fracture under general anesthesia. The patient had a history of depression and had been treated with olanzapine and paroxetine hydrochloride for six months, presenting with sedation and apathy preoperatively. A single shot of fascia iliaca compartment block (0.2% ropivacaine 30 mL) was administered in the anesthesia preparation room. Following induction of anesthesia with propofol, sufentanil, and rocuronium, successful insertion of a laryngeal mask airway (LMA) was achieved, and anesthesia was maintained with sevoflurane and propofol. The surgical procedure was uneventful and lasted one hour. However, delayed emergence was observed postoperatively. Despite the administration of sugammadex, the criteria for LMA removal were not met until 45 min after the cessation of anesthetics. Upon removal of the LMA, the patient exhibited snoring respirations. Thirty minutes later, a decreased level of consciousness was observed in the Post-Anesthesia Care Unit (PACU), along with a peripheral oxygen saturation (SpO_2) level that dropped to 89%. The patient also expectorated pink, frothy sputum. Immediate oxygen supplementation via face mask was initiated, and intravenous dexamethasone (10 mg) was administered, resulting in no significant improvement. Two minutes later, 20 mg of furosemide was given while continuous oxygenation via face mask was maintained,

leading to an increase in SpO_2 to 95%. After being transferred to the Intensive Care Unit (ICU), the patient continued to receive supplemental oxygen through either a face mask or a nasal cannula. The following day, her symptoms improved, with SpO_2 rising to 99%. She reported feeling well and was returned to the ward. The patient was discharged three days later and remained stable during follow-up at one month post-discharge. Chest imaging studies are presented in Fig. 1A.

Case 2

A 37-year-old male underwent debridement of the left lower extremity and had a history of habitual snoring. A single femoral and sciatic nerve block was administered (0.2% ropivacaine, 30 mL and 20 mL, respectively) in the anesthesia preparation room. The anesthesia induction, maintenance protocol, and LMA insertion were consistent with those used in Case 1. The operation proceeded smoothly for approximately 1 h and 40 min, after which the patient regained consciousness and spontaneous respiration. Neostigmine and atropine were administered to reverse neuromuscular blockade, and the LMA was removed once extubation criteria were met. Shortly after extubation, the patient developed glossoptosis and snoring, along with a decrease in SpO_2 to 78% and cyanosis. Chin elevation was promptly performed, a 7.5 mm nasopharyngeal airway was inserted, and continuous mask oxygen was administered. Subsequently, methylprednisolone (80 mg) and furosemide (10 mg) were given. Following this treatment, the patient's SpO_2 was maintained at approximately 90%. Upon transfer to the ICU, the patient received oxygen therapy via mask or nasal catheter, nebulization therapy, and awake prone position. After being positioned in the prone orientation, the patient expectorated pink, frothy sputum. The patient demonstrated improvement and returned to the ward three days later, being discharged ten days post-operation with no abnormalities noted during a three-month follow-up. Chest imaging is displayed in Fig. 1B.

Case 3

A 66-year-old man underwent surgery for the removal of internal fixation from the left humerus. The anesthesia induction, maintenance protocol, and LMA insertion were consistent with those utilized in Case 1. Near the conclusion of the procedure, the patient's airway pressure abruptly increased to over 30 cm H_2O, accompanied by spontaneous movement and breathing. At this point, the anesthesiologist observed that the LMA had been displaced, the patient exhibited trismus with strong inspiratory efforts, and SpO_2 had decreased to 80%. Following the immediate intravenous administration of 70 mg of propofol and 30 mg of rocuronium, the LMA was removed, and SpO_2 increased to 92% after face-mask ventilation with 100% oxygen. Subsequently, an endotracheal tube was inserted with the assistance of a video laryngoscope. SpO_2 was maintained at approximately 94%, and airway pressure was recorded at 24–25 cm H_2O under mechanical ventilation with 100% oxygen. Pink secretions were observed in the oral cavity as well as within the anesthetic circuit. Methylprednisolone (40 mg) and dexamethasone (10 mg) were administered successively. The operation continued, and the endotracheal tube was removed 20 min post-procedure. The patient was transferred to the ward following surgery, where oxygen therapy via mask or nasal catheter, nebulization, and furosemide were provided. The patient was discharged on the 10th postoperative day and was followed up one month later, with no abnormalities noted. Chest imaging is displayed in Fig. 1C.

Case 4

A 41-year-old male with no significant medical history underwent arthroscopic surgery on the left knee under general anesthesia. Following a single administration of saphenous nerve block and sciatic nerve block (20 mL each of 0.2% ropivacaine), the anesthesia induction, maintenance, and airway management strategies were consistent with those used in Case 1. The surgical procedure lasted 1 h and 35 min. Neuromuscular blockade was reversed with neostigmine and atropine at the conclusion of the surgery. After the removal of the LMA, the patient exhibited snoring in the PACU; however, SpO_2 was maintained at 98% with supplemental mask oxygen, and he did not complain of discomfort. Approximately 40 min post-extubation, he developed dyspnea and produced pink, frothy sputum, leading to a drop in SpO_2 to 86%. A nasopharyngeal airway was promptly inserted, and oxygen was administered via mask. SpO_2 slightly improved to 90%, and 40 mg of methylprednisolone was administered. The patient was subsequently transferred to the ICU with ongoing mask oxygen therapy. During this period, SpO_2 was maintained between 86 and 90%, and the patient was positioned in a prone orientation while receiving oxygen therapy. One day later, the patient was transitioned to nasal cannula oxygen at a flow rate of 3–5 L/min, resulting in SpO_2 stabilization at 98%. The patient was then transferred back to the ward and discharged 7 days later with no unusual complications noted at a one-month follow-up. Chest imaging is displayed in Fig. 1D.

Case 5

A 50-year-old male underwent debridement and tendon anastomosis for an open wound in the right calf under general anesthesia. The patient had a history of habitual snoring. A single femoral and sciatic nerve block was

administered, utilizing 30 mL of 0.2% ropivacaine for the femoral nerve and 20 mL for the sciatic nerve. Following the nerve block, anesthesia was induced with propofol, sufentanil, and rocuronium. An ID 7.5 endotracheal tube was successfully inserted with the assistance of a video laryngoscope, and anesthesia was maintained with sevoflurane and propofol. The surgical procedure lasted 1 h and 45 min. Sugammadex sodium was administered, resulting in successful extubation of the patient. Two minutes post-extubation, the patient exhibited signs of tongue drop and experienced difficulty in ventilation. Oxygen was delivered via mask with assisted ventilation using mandibular support, maintaining SpO_2 levels above 80%. The patient became irritable and coughed up pink, frothy sputum for over 10 min. In response, intravenous methylprednisolone (40 mg) was administered, which improved SpO_2 levels to above 90%. The patient was subsequently admitted to the ICU. In the ICU, nasal catheter oxygen was provided, maintaining SpO_2 at 96%. Twenty minutes later, he developed further ventilation difficulties and was immediately placed on non-invasive ventilator-assisted ventilation. Intravenous furosemide (20 mg) was administered, and dexmedetomidine was given for sedation. After one day, the patient's symptoms improved, leading to a transfer out of the ICU. Six days later, he was discharged from the hospital, and a follow-up visit one-month post-discharge revealed no remarkable findings. Chest imaging is displayed in Fig. 1E.

Discussion and conclusions

NPPE is an important clinical complication with a low incidence. Reports indicate that the incidence of NPPE in patients undergoing general anesthesia ranges from 0.1% to 11% [4, 5], with variability depending on specific clinical settings. Although rare, NPPE poses risks such as hypoxemia, cardiac failure, and shock [4]. It may manifest as a serious complication during the recovery phase from anesthesia, particularly following upper airway obstruction due to laryngospasm during extubation [1]. This complication is noted in certain high-risk surgeries like rhinoplasty or spinal surgery and may result in prolonged recovery and extended hospitalization, or even reoperation [5]. The severity of NPPE is underscored by its potential to cause additional complications, such as Takotsubo cardiomyopathy, which may worsen the patient's condition and affect multiple organs [6]. These factors highlight the importance of early identification and intervention for NPPE during anesthesia recovery.

NPPE can be classified into two types [7]. Type I occurs when a patient generates high negative intrathoracic pressure by forceful inspiration against a closed glottis. This negative pressure may reach -50 cmH_2O or higher [2, 7] and can be transmitted to the pulmonary capillaries, resulting in hydrostatic imbalance and subsequent capillary fluid leakage [7]. Type II is more common in children with chronic upper airway obstruction, who develop increased positive end-expiratory pressures over time to maintain airway patency. When the obstruction is abruptly relieved, intrathoracic pressure drops sharply, creating negative pressure that is transmitted to the pulmonary capillaries, leading to fluid extravasation into the lung interstitium [7]. The primary mechanism of NPPE involves forceful inspiratory efforts against an obstructed airway, commonly due to laryngospasm or obstruction post-extubation [1]. These efforts produce marked negative intrathoracic pressures, which increase venous return, elevate pulmonary blood volume, and raise pulmonary capillary pressure, potentially resulting in capillary rupture and alveolar fluid leakage. Additionally, stress failure of capillary walls can increase pulmonary vascular permeability, further aggravating pulmonary edema [8].

In recent years, NPPE has garnered increased attention due to its potential severity and recognition as a perioperative complication. Within the past five years, 17 cases of NPPE associated with general anesthesia have been documented, as presented in Table 2. Studies suggest that younger patients, particularly healthy males, are at a higher risk of developing NPPE post-extubation, often occurring within minutes to a few hours. This heightened risk may be linked to their capacity to generate greater negative intrathoracic pressures [2]. NPPE typically presents with symptoms such as dyspnea, agitation, pink frothy sputum, and decreased oxygen saturation. Management protocols generally encompass the prompt re-establishment of a patent airway, administration of supplemental oxygen, positive pressure ventilation, and diuretics [9]. Recent case studies have explored alternative treatment modalities for NPPE, such as extracorporeal membrane oxygenation, which has demonstrated effectiveness in severe cases unresponsive to conventional therapies [10]. This approach may be beneficial in cases complicated by refractory hypoxemia. Although NPPE remains uncommon in patients undergoing general anesthesia, its potential severity and diverse clinical manifestations necessitate heightened vigilance from anesthesiologists [11].

This report presents five cases that examine the predisposing factors, clinical manifestations, management strategies, and outcomes of NPPE. Key predisposing factors were identified within this case series. In Case 3, strong inspiratory efforts due to the displacement of the laryngeal mask, which subsequently resulted in the development of NPPE. In the remaining four cases, glossoptosis emerged as the primary triggering factor, underscoring the significant role of airway obstruction

Table 2 Summary of NPPE case characteristics in the last five years

Year	Age (years)	Sex	Type of surgery	Type of airway ventilation	Time window	Therapeutic intervention	Refs
2024	24	F	Laparoscopic cholecystectomy	ETI	50 min after surgery	Diuretic, methylprednisolone	[12]
2024	28	F	Axillary lymph node dissection	LMA	20 min after surgery	Mask-delivered positive pressure, high-flow oxygen therapy	[13]
2024	12	M	Tonsillectomy	ETI	PACU	Restricted fluid therapy, diuretic, high-flow oxygen therapy	[14]
2024	55	F	Tonsillectomy	ETI	PACU	Oxygen therapy, steroid	[15]
2023	28	M	Dental caries treatment	ETI	No mention	Mask-delivered positive pressure	[16]
2023	27	F	Laparoscopic sleeve gastrectomy	ETI	Shortly before extubation	Diuretic, epinephrine, continuous suctioning of airway secretions	[17]
2023	2 months	M	Pyloromyotomy	ETI	Shortly after extubation	Dexamethasone, high-flow oxygen therapy	[18]
2023	65	M	Cervical corpectomy	ETI	Immediately after extubation	Mask-delivered positive pressure, high-flow oxygen therapy	[19]
2022	25	F	Breast reconstruction surgery	ETI	After extubation	Continuous positive airway pressure, diuretic	[20]
2022	35	M	Laparoscopic hernia repair	ETI	After anesthesia induction	Diuretic, celiprolol sodium hydrate	[21]
2022	44	M	Robot-assisted laparoscopic bladder augmentation	ETI	20 min after extubation	Re-tubation with positive pressure ventilation	[22]
2021	21	M	Open rhinoplasty	ETI	3 min after extubation	Re-tubation with positive pressure ventilation	[23]
2021	78	M	Laminectomy	ETI	PACU	Diuretic, methylprednisolone	[24]
2021	68	F	Lumbar vertebroplasty	ETI	Before extubation	Mask-delivered positive pressure	[25]
2020	28	F	Laparoscopic exploration	ETI	Immediately after extubation	Re-tubation, diuretic, methylprednisolone	[26]
2020	36	/	Septoplasty	ETI	3 h after extubation	Diuretic, continuous positive airway pressure	[27]
2020	37	F	Vocal cord polypectomy	ETI	3.5 h after extubation	Cardiopulmonary resuscitation, veno-arterial extracorporeal membrane oxygenation	[28]

Abbreviations: *F* Female, *M* Male, *ETI* Endotracheal intubation, *LMA* Laryngeal mask airway, *Refs*. References, *PACU* Post-anesthesia care unit

in the pathogenesis of NPPE. This observation differs somewhat from previous literature, which suggests that laryngospasm is the most likely inducer of NPPE, thereby aiding anesthesiologists in identifying high-risk groups and facilitating preventive measures.

Each case exhibited characteristic NPPE clinical features. In Case 1, delayed recovery of consciousness and postoperative hypoxemia were noted, both characteristic of NPPE onset. All cases showed pink, frothy sputum, wet rales visible on auscultation and hypoxemia requiring at least supplemental oxygen via a face mask. Imaging findings revealed bilateral extensive infiltrates and ground-glass opacities, which contributed to a definitive diagnosis of NPPE. Imaging demonstrated bilateral infiltrates and ground-glass opacities, supporting an NPPE diagnosis. Treatment across cases followed a structured approach, including oxygen therapy, corticosteroids, and a restrictive fluid strategy with intravenous furosemide. In our study, all five patients experienced significant hypoxemia during the early postoperative period. Evidence suggests that the prone position can effectively enhance oxygenation in patients with acute respiratory distress syndrome, especially in cases where there is atelectasis in the gravity-dependent areas of the lungs [29, 30]. The application of the prone position for two patients of this study was based on the analysis of pulmonary imaging and achieved marked improvement in oxygenation without notable complications.

While previous cases have been documented, the early recognition and diagnosis of NPPE remain challenging. Based on cases from our institution and literature reports, several key factors can help in confirming the diagnosis of NPPE. First, potential triggers should be considered, including foreign body aspiration, tracheal mucus secretion, hiccups, difficult airway intubation, upper airway tumors, mediastinal tumors, compression of the thyroid isthmus, pharyngeal surgeries, glossoptosis during recovery from anesthesia, and severe patient-ventilator dyssynchrony. Second, the primary symptoms of NPPE are generally acute dyspnea, cough with pink,

frothy sputum, hypoxemia, and chest pain. Patients frequently report shortness of breath, rapidly declining oxygen saturation, and may also describe chest tightness or discomfort [31]. Clinically, imaging studies can be useful in confirming the diagnosis. Chest X-rays may reveal diffuse infiltrative shadows in both lungs, typically in a butterfly distribution, consistent with nonspecific pulmonary interstitial edema. Computed tomography (CT) scans offer greater detail, showing bilateral diffuse ground-glass opacities and alveolar infiltrates. However, diagnosis during the perioperative period remains challenging [32]. These imaging findings resemble those seen in other forms of noncardiogenic pulmonary edema and should be interpreted alongside clinical symptoms for an accurate diagnosis. Additionally, the patient's recovery trajectory can aid in diagnosis. NPPE is generally self-limiting, and with appropriate treatment, symptoms often improve significantly within 48 h, with most patients fully recovering within 72 h. In severe cases, recovery may take longer, but the prognosis is usually favorable [31].

Previous studies have demonstrated the efficacy of several therapeutic interventions, including: (1) Airway obstruction relief: This is the critical first step, which may require reintubation or other methods to maintain airway patency. (2) Oxygen therapy: High-flow oxygen or noninvasive positive pressure ventilation are commonly used to manage hypoxemia. Mechanical ventilation may be necessary in severe cases. (3) Diuretics: Furosemide can alleviate pulmonary fluid accumulation, but must be used cautiously to avoid precipitating hypovolemic shock. (4) Sedatives: When necessary, sedatives may be used to manage anxiety and control breathing, thus reducing the risk of further pulmonary injury. (5) Supportive therapy: This includes prone position, fluid management, hemodynamic monitoring, and, if required, vasoactive agents to maintain hemodynamic stability.

Based on the findings of this study, the prevention of NPPE during anesthesia recovery primarily relies on effective airway management to prevent upper airway obstruction. Key strategies include ensuring complete reversal of neuromuscular blockade before extubation, maintaining `an adequate depth of anesthesia during airway manipulation, and avoiding premature extubation to reduce the risk of laryngospasm and glossoptosis. Vigilant monitoring of high-risk patients, particularly those undergoing surgeries associated with airway edema, is essential. Early signs of airway obstruction, such as stridor and paradoxical breathing, should prompt immediate intervention. The use of airway adjuncts, like oropharyngeal or nasopharyngeal airways, can help maintain airway patency in vulnerable individuals. Additionally, applying continuous positive airway pressure or non-invasive ventilation in cases of partial obstruction can prevent the generation of excessive negative intrathoracic pressures. Timely recognition and prompt relief of airway obstruction, along with supplemental oxygen administration, are critical in mitigating the risk of NPPE. Educating perioperative teams on NPPE risk factors and management protocols further enhances prevention efforts.

This study has several limitations. First, the sample size was small, consisting of only five cases, which restricts the generalizability of the findings. Second, the data were collected from a single institution, which may introduce selection bias, and patient characteristics may vary across different institutions, potentially influencing treatment approaches. The short follow-up period, primarily focused on the acute recovery phase, limited the assessment of the long-term effects of NPPE. Additionally, while several therapeutic interventions were summarized, the variation in treatment strategies across cases may have affected the results. Retrospective studies involving multiple centers and longer follow-up periods can be conducted in the future to further validate these findings.

This case series highlights the importance of prevention and early identification of NPPE during anesthesia recovery, especially in patients with laryngeal mask displacement during recovery as well as in those with glossoptosis after extubation. Anesthesiologists should allocate greater attention to identifying and managing these airway-related events.

Abbreviations

BP	Blood pressure
BMI	Body mass index
CS	Corticosteroids
CT	Computed tomography
ETI	Endotracheal intubation
F	Female
ICU	Intensive Care Unit
LMA	Laryngeal mask airway
M	Male
NPPE	Negative pressure pulmonary edema
PACU	Post-Anesthesia Care Unit
Refs.	References
SpO2	Peripheral oxygen saturation

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Authors' contributions
Meng Luo: conception, design, data acquisition, formal analysis, writing-original draft. Man Li: data curation, visualization, software, validation, writing review and editing. Zhijun Qin: resources, supervision, conceptualization, project administration, methodology, funding acquisition, writing review and editing. All authors have read and approved the manuscript.

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Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate
This study was approved by the Ethics Committee of Sichuan Province Orthopedic Hospital in accordance with the Declaration of Helsinki (no. KY2024-061–01). Written informed consent was obtained from all patients.

Consent for publication
Written informed consent for publication was obtained from every patient for the publication of the date, images, etc.

Competing interests
The authors declare no competing interests.

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