Therapeutics and Clinical Risk Management

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ORIGINAL RESEARCH

Incidence of and Risk Factors for Laryngopharyngeal Reflux in Patients Undergoing Modified Electroconvulsive Therapy: A Prospective Observational Study

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Background: Laryngopharyngeal reflux (LPR) is among the most common complications associated with modified electroconvulsive therapy (MECT). The purpose of this study was to assess the prevalence of LPR among patients undergoing MECT and to identify risk factors for LPR within this population.

Methods: This observational prospective study enrolled 107 consecutive patients who underwent MECT at the Third Affiliated Hospital of Sun Yat-sen University. Data regarding potential risk factors for LPR in patients undergoing MECT were collected. The salivary pepsin test was used to diagnose LPR.

Results: The incidence of LPR was 39.3% in this study. On univariate analysis, height ($p = 0.040$), history of acid regurgitation ($p = 0.19$), number of MECT session number ($p = 0.014$), succinylcholine dose ($p = 0.032$), and oral secretion volume ($p = 0.01$) were significantly associated with LPR. Outcomes from the multivariate analysis are shown as odds ratio (OR [95% confidence interval (CI)]), >3 MECT sessions (3.02 [1.20–7.58]), history of acid regurgitation (3.90 [1.20–12.70]), succinylcholine dose > 50 mg (2.54 [1.04–6.22]), oral secretion volume > 3 mL (3.66 [1.50–8.97]) were significantly and independently associated with the development of LPR.

Conclusion: A history of acid regurgitation, >3 MECT sessions, succinylcholine dose > 50 mg, oral secretion volume > 3 mL was significantly associated with an increased risk of LPR in patients undergoing MECT.

Keywords: LPR, MECT, risk factors, salivary pepsin test

Introduction

In modified electroconvulsive therapy (MECT), a grand mal seizure is induced by directly applying electrical stimulation to the scalp under general anaesthesia.[1] Encouragingly, advancements in anaesthesia and technology have facilitated MECT as a comparatively safe and efficacious therapeutic approach for psychiatric diseases such as major depressive disorder, bipolar disorder, schizophrenia, schizoaffective disorders, and catatonia.[2,3] Nevertheless, headaches, myalgia, nausea, cardiovascular changes, and respiratory and neurological complications are some of the side effects associated with MECT,[4] among which laryngopharyngeal reflux (LPR) is often underreported.

LPR is a clinical syndrome characterised by the dysregulation of gastric contents that leads to various laryngeal symptoms such as throat clearing, hoarseness, pain, a globus sensation, coughing, excessive throat mucus, and voice disorders.[5,6] A study examining patients with laryngeal and voice disorders found that 50% were diagnosed with LPR based on 24-h double-probe pH monitoring.[7] About 10–30% of consultations in otolaryngology are due to LPR.[8] From 1990 to 2019, the overall burden of gastroesophageal reflux disease (GERD) increased by 77.53%, with prevalent cases rising from 441.57 million to

Received: 28 July 2025
Accepted: 13 November 2025
Published: 12 December 2025

783.95 million.[9] Importantly, GERD after MECT is independently associated with a 30-day mortality rate of 1.13%.[10] Compared to those without LPR, GERD patients with LPR symptoms have a poorer quality of life, a lower satisfaction rate, and higher medical expenses.[11] However, the occurrence of LPR in patients undergoing MECT is uncertain. Due to anesthesia, temporary relaxation of the lower esophageal sphincter, and cardiovascular and respiratory factors, patients undergoing MECT may be vulnerable to LPR. It is essential to identify risk factors that can be controlled and take preventive measures early to reduce their occurrence and prevent further complications like LPR and aspiration.

To our knowledge, the predictive factors associated with LPR in patients undergoing MECT treatment have not been reported. The study's objective was to examine the occurrence of LPR in patients receiving MECT and identify possible risk factors for LPR in this group to facilitate improved management and targeted therapeutic interventions, ultimately enhancing patient outcomes.

Materials and Methods

This prospective observational study complied with the Declaration of Helsinki and received approval from the Research Ethics Committee at the Third Affiliated Hospital of Sun Yat-sen University (no. 2022–086-01). All participants provided informed consent.

Study Population

The inclusion criteria were >14 years and <75 years and having undergone MECT in the daytime operating room of our centre between June 2022 and July 2022. The exclusion criteria were MECT contraindications, severe cardiovascular and cerebrovascular diseases, uncontrolled diabetes, acute and uncured respiratory inflammation within 2 weeks, positive pepsin detection before MECT, and noncooperation.

Data Collection

Based on previous sources,[12–14] the clinical data related to demographics, vital signs, comprehensive medical history, and perioperative factors linked to LPR were collected from the electronic health records system and a questionnaire. Demographic characteristics included sex, age, height, weight, body mass index (BMI), and relevant pre-existing medical conditions, such as a history of alcohol consumption, smoking, acid regurgitation, gastroesophageal diseases, and prior gastroesophageal surgery and a previous use of gastric protective medications. Perioperative variables included Mallampati airway classification, fasting duration, American Society of Anesthesiologists (ASA) score, number of MECT sessions, seizure duration, and oral secretion volume. Perioperative medications included psychiatric medications, penehyclidine hydrochloride, propofol, and succinylcholine. Perioperative adverse events were also collected, including acute cough, nausea and vomiting, delirium, airway spasms, body movements, delayed recovery, fever, and pneumonia.

Prior to the surgical procedure, a comprehensive medical history and physical examination were conducted of all patients accompanied by the routine restrictions on drinking and fasting. Specially trained nurses documented essential patient information using a research-specific questionnaire. Written informed consent was obtained from all participants before the evaluation. Deep oral secretions were collected before the use of anticholinergic drugs and immediately after the electrical stimulation. Following the guidelines provided by the Peptest™ (RD Biomed Limited, NY, United Kingdom), the patients were instructed to produce a minimum of 1 mL of saliva by expectorating into 0.5-mL tubes containing citrate. Before the induction of anaesthesia, the standard ASA monitoring, which included electrocardiography, oxygen saturation, blood pressure, heart rate, and respiratory rate, was implemented. Anesthetic drugs were administered at the standard dose before and during MECT.[3,15] Within 15 to 30 minutes before induction, 0.007 mg/kg atropine was administered to reduce saliva secretion. General anaesthesia was then induced intravenously using 2 mg/kg propofol. Succinylcholine 0.5–1.5 mg/kg was intravenously administered after a bite block was placed, and ventilation was achievable using the transparent facemask held in place by head straps. After the clinical cessation of a tender reflex, the first MECT stimulus was administered. The MECT dose and seizure duration were determined based on patients' age, weight, MECT frequency, psychiatric medications, and symptoms. The ideal seizure time is 25 seconds. To estimate the seizure duration, the time interval in seconds between seizure onset and offset points was visually identified on the

EEG.[16] Deep oral secretions were collected by suction immediately after the MECT procedure. When spontaneous respiration resumed, the patients were transferred to the post-anaesthesia care unit (PACU).

Saliva Collection and Laboratory Procedures

A total of 103 saliva samples were collected. The volume of saliva in each container was recorded. Samples were refrigerated prior to being tested for pepsin using Peptest™. The collection tubes underwent centrifugation at 4000 rpm for a duration of 5 minutes. A volume of 80 μL from the top layer of the centrifuged sample was transferred to a microtube with 240 μL of migration buffer, and the mixture was vortexed for 10 seconds. Afterward, 80 μL of the mixed sample was transferred to the circular well of the lateral flow device (LFD). The LFD includes two distinct human monoclonal antibodies against pepsin: one for capturing and another for detecting pepsin. The pepsin test cube reader automatically converts the intensity of the pepsin test line on the LFD window into the concentration of pepsin measured in ng/mL. A pepsin concentration of over 75 ng/mL is indicative of LPR.[17]

Endpoints

The primary endpoint was the incidence of LPR. The patient's risk factors for LPR were investigated during the preoperative visit. The secondary endpoint was the incidence of perioperative adverse events.

Sample Size

Based on the findings of the pilot study, the incidence of LPR was 50%. To ensure adequate statistical power, a sample size calculation was performed with an alpha value of 0.05 and a power of 0.8, resulting in a required sample size of 97 patients. Accounting for a projected dropout rate of 10%, the minimum total sample size was adjusted to 107 patients.

Statistical Analysis

The statistical analysis was conducted using SPSS (version 25.0; IBM Corp., NY, USA). Continuous variables were tested for normality using the Shapiro–Wilk test and homogeneity of variances using Levene's test. Normally distributed data were reported as mean ± standard deviation, and the median (interquartile ranges [IQR]) was used for non-normally distributed data. Categorical variables are presented as frequencies (%). For statistical comparisons, Student's *t*-test was used for variables with normal distribution and homogeneous variances, and the Mann–Whitney *U*-test for variables with non-normal distribution or heterogeneous variances, and categorical variables were analysed by the chi-squared test or Fisher's exact test. All factors with values of $P < 0.05$ in the univariate analysis were included in a multivariate logistic regression model. All tests were two-sided at a significance level of 0.05.

Results

A total of 129 patients undergoing MECT were included in the eligibility review. A total of 125 patients were included in the study after excluding those who did not meet the eligibility criteria. Eighteen patients were lost to follow-up, most commonly because of failing to detect pepsin and language problems. In the final cohort, 107 patients were analysed (Figure 1).

Cohort Characteristics

The median patient age was 25 (15–31) years. Overall, 67.3% of the patients were female. LPR occurred in 42/107 (39.3%) of them (Table 1).

Risk Factors for LPR

During the perioperative visit, patients' risk factors for LPR included height ($p = 0.04$), number of MECT session, history of acid regurgitation, succinylcholine dose, and oral secretion volume. There was no significant difference in the consumption of penehyclidine hydrochloride ($p = 0.242$) and propofol ($p = 0.091$) between patients with and without LPR (Table 1). However, the consumption of intraoperative succinylcholine was significantly higher in patients with LPR (54.8% vs 33.8%, $p = 0.032$; Table 2). Furthermore, patients with LPR were more likely to have a history of acid regurgitation (28.6% vs 10.8%, $p = 0.019$), a greater oral secretion volume (57.1% vs 26.2%, $p = 0.001$), and

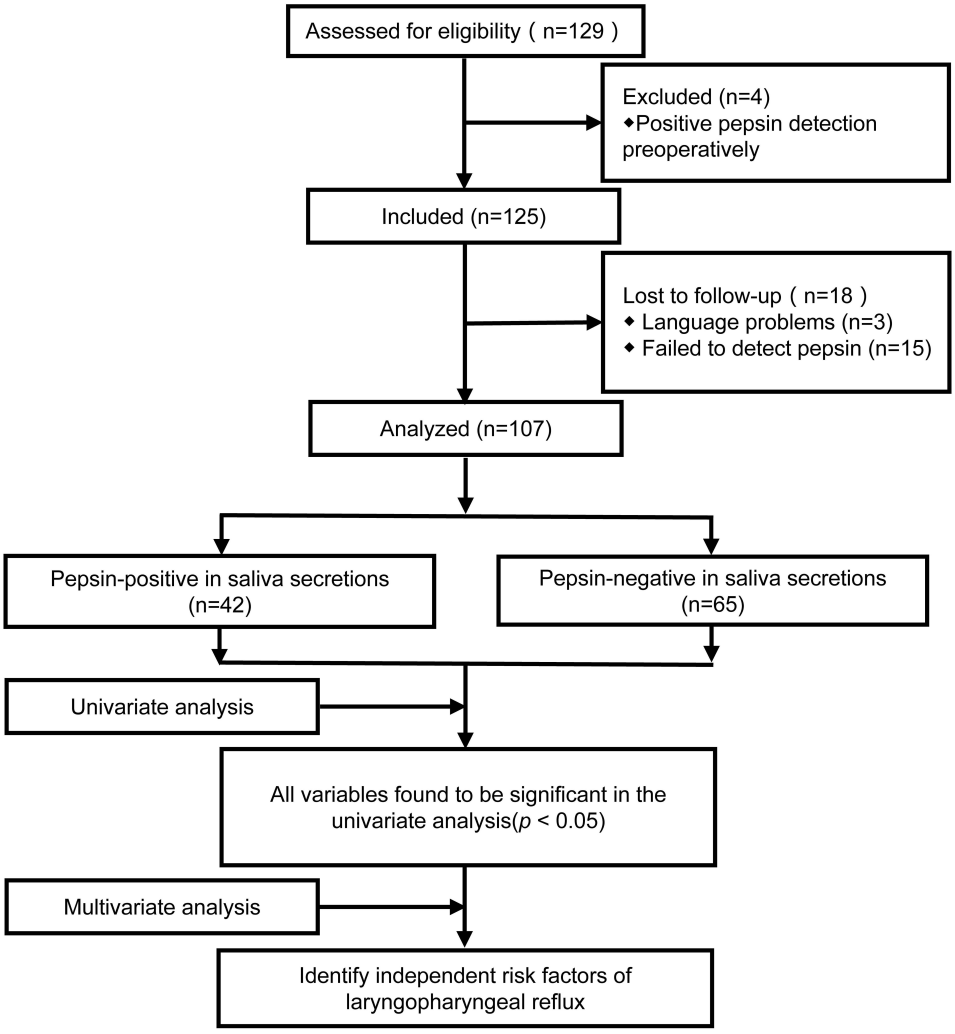

Figure 1 Flowchart of patient selection process.

undergone more MECT sessions (47.6% vs 24.6%, $p = 0.014$). A diagnosis of psychiatric disease did not significantly impact the risk of LPR, nor did the preoperative use of psychiatric medications ($p = 0.119$; Table 1). No significant differences were found between the LPR and no LPR groups in sex ($p = 0.072$), age ($p = 0.522$), weight ($p = 0.153$), BMI

Table 1 Baseline Characteristics of Patients with versus Without LPR

Variable	Total (N=107)	Pepsin Status		*P* value
		Positive (n=42)	Negative (n=65)	
	107	42 (39.3%)	65 (60.7%)	
Sex				0.072
Male	35 (32.7%)	18 (42.9%)	17 (26.2%)	
Female	72 (67.3%)	24 (57.1%)	48 (73.8%)	
Age, years	25 (15–31)	22 (16–33)	18 (14–31)	0.522
Height, cm		165 (160–173)	163 (159–168)	0.04
Weight, kg		63 ± 13	59 ± 14	0.153

(*Continued*)

Table 1 (Continued).

Variable	Total (N=107)	Pepsin Status		*P* value
		Positive (n=42)	Negative (n=65)	
BMI ($kg.m^{-2}$)				0.515
Underweight	24 (22.4%)	9 (21.4%)	15 (23.1%)	
Normal weight	49 (45.8%)	17 (40.5%)	32 (49.2%)	
Overweight	34 (31.8%)	16 (38.1%)	18 (27.7%)	
Diagnosis				0.435
Bipolar disorder	48 (44.9%)	20(47.6%)	28 (43.1%)	
Depression	21 (19.6%)	5 (11.9%)	16 (24.6%)	
Mood disorder	14 (13.1%)	6 (14.3%)	8 (12.3%)	
Schizophrenia	24 (22.4%)	11 (26.2%)	13 (20.0%)	
Psychiatric meditations				0.119
Olanzapine	13 (4.7%)	5 (11.9%)	8 (12.3%)	
Quetiapine fumarate	29 (27.1%)	10 (23.8%)	19 (29.2%)	
Sodium valproate	16 (15.0%)	6(5.6%)	10 (9.3%)	
Aripiprazole	2 (1.9%)	0 (0)	2 (3.1%)	
Lamotrigine	3 (2.8%)	0 (0)	3 (4.6%)	
Clozapine	5 (4.7%)	4 (9.5%)	1 (1.5%)	
Oxcarbazepine	8 (7.5%)	2 (4.8%)	6 (9.2%)	
Paliperidone	1 (0.9%)	0 (0)	1 (1.5%)	
Amisulpride	4 (3.7%)	3 (7.1%)	1 (1.5%)	
Lithium carbonate	18 (16.8%)	8 (19.0%)	10 (15.4%)	
Risperidone	6 (5.6%)	2 (4.8%)	4 (6.2%)	
Sertraline	2 (1.9%)	2 (4.8%)	0 (0)	
Smoking				0.736
Yes	9 (8.4%)	4 (9.5%)	5 (7.7%)	
No	98 (91.6%)	38 (90.5%)	60 (92.3%)	
Drinking				1
Yes	2 (1.9%)	1 (2.4%)	1 (1.5%)	
No	105 (98.1%)	41 (97.6%)	64 (98.5%)	
History of acid regurgitation				0.019
Yes	19 (17.8%)	12 (28.6%)	7 (10.8%)	
No	88 (82.2%)	30 (71.4%)	58 (89.2%)	
History of gastroesophageal surgery				0.152
Yes	2 (1.9%)	2 (1.9%)	0 (0)	
No	105 (98.1%)	40 (98.1%)	65 (100%)	
Gastroesophageal disease				0.141
Yes	14 (13.1%)	8 (19.0%)	6 (9.2%)	
No	93 (86.9%)	34 (81.0%)	59 (90.8%)	
Other comorbidities				0.208
Yes	6 (5.6%)	4 (9.5%)	2 (3.1%)	
No	101 (94.4%)	38 (90.5%)	63 (96.9%)	
Preoperative use of gastric protective drugs				0.258
Yes	8 (7.5%)	5 (11.9%)	3 (4.6%)	
No	99 (92.5%)	37 (88.1%)	62 (95.4%)	
ASA classification				1
I	103 (96.3%)	41 (97.6%)	62 (95.4%)	
II	4 (3.7%)	1 (2.4%)	3 (4.6%)	

(*Continued*)

https://doi.org/10.2147/TCRM.S549412

Table 1 (Continued).

Variable	Total (N=107)	Pepsin Status		*P* value
		Positive (n=42)	Negative (n=65)	
Mallampati classification				0.208
I	90 (84.1%)	33 (78.6%)	57 (87.7%)	
II	17 (15.9%)	9 (21.4%)	8 (12.3%)	
Fasting duration				0.899
Number of MECT sessions				0.014
≤3	71 (66.4%)	22 (52.4%)	49 (75.4%)	
>3	36 (33.6%)	20 (47.6%)	16 (24.6%)	
Drugs used in MECT				
Penehyclidine hydrochloride (mg)		0.4 (0.4–0.5)	0.4 (0.3–0.5)	0.242
Propofol (mg)				0.091
≤100	69 (64.5%)	23 (54.8%)	46 (70.8%)	
>100	38 (35.5%)	19 (45.2%)	19 (29.2%)	
Succinylcholine (mg)				0.032
≤50	62 (57.9%)	19 (45.2%)	43 (66.2%)	
>50	45 (42.1%)	23 (54.8%)	22 (33.8%)	
Seizure duration (s)		5 (3–8)	4 (0–6)	0.387
Oral secretion volume (mL)				0.001
≤3	66 (61.7%)	18 (42.9%)	48 (73.8%)	
>3	41 (38.3%)	24 (57.1%)	17 (26.2%)	

Notes: Data are presented as mean ± standard deviation, median (IQR), or n (%). Data are shown as median (IQR) or n (%).
Abbreviations: BMI, body mass index; LPR, laryngopharyngeal reflux; LPR, laryngopharyngeal reflux; MECT, modified electroconvulsive therapy.

(p = 0.515), smoking history (p = 0.736), alcohol history (p = 1), concurrent gastroesophageal diseases (p = 0.141), history of gastroesophageal surgery (p = 0.152), preoperative use of gastric protective drugs (p = 0.258), seizure duration (p = 0.387), ASA classification (p = 1), Mallampati classification (p = 0.208), or fasting duration (p = 0.899; Table 1).

Regression Analysis of Risk Factors for LPR

On univariate analysis, height, history of acid regurgitation, greater oral secretion volume, and more MECT sessions were significantly associated with LPR (Table 2). These variables were included in the final multivariate analysis. In the

Table 2 Risk Factors for LPR on Univariate Analyses

Factor	Total	Pepsin Status		*P* value
		Positive	Negative	
Height				0.04
History of acid regurgitation				0.019
Yes	19 (17.8%)	12 (28.6%)	7 (10.8%)	
No	88 (82.2%)	30 (71.4%)	58 (89.2%)	
Number of MECT sessions				0.014
≤3	71 (66.4%)	22 (52.4%)	49 (75.4%)	
>3	36 (33.6%)	20 (47.6%)	16 (24.6%)	
Succinylcholine (mg)				0.032
≤50	62 (57.9%)	19 (45.2%)	43 (66.2%)	
>50	45 (42.1%)	23 (54.8%)	22 (33.8%)	

(*Continued*)

Table 2 (Continued).

Factor	Total	Pepsin Status		*P* value
		Positive	Negative	
Oral secretion volume (mL)				0.001
≤3	66 (61.7%)	18 (42.9%)	48 (73.8%)	
>3	41 (38.3%)	24 (57.1%)	17 (26.2%)	

Notes: Data are presented as n (%).
Abbreviations: LPR, laryngopharyngeal reflux; MECT, modified electroconvulsive therapy.

Table 3 Risk Factors for LPR on Multivariate Analysis

Subgroup	OR (95% CI)	*P* value
History of acid regurgitation		
No	1 (1–1)	
Yes	3.90 (1.20–12.70)	0.024
Number of MECT sessions		
≤3	1 (1–1)	
>3	3.02 (1.20–7.58)	0.018
Succinylcholine (mg)		
≤50	1 (1–1)	
>50	2.54 (1.04–6.22)	0.042
Oral secretion volume (mL)		
≤3	1 (1–1)	
>3	3.66 (1.50–8.97)	0.004

Abbreviations: CI, confidence interval; LPR, laryngopharyngeal reflux; OR, odds ratio.

multivariate analysis, >3 MECT sessions (3.02 [1.20–7.58]), history of acid regurgitation (3.90 [1.20–12.70]), dose of succinylcholine > 50 mg (2.54 [1.04–6.22]), and oral secretion volume > 3 mL (3.66 [1.50–8.97]) were significantly and independently associated with the development of LPR (Table 3). Figure 2 shows the forest plot of factors analysed to determine association with incidence of laryngopharyngeal reflux on multivariate logistic regression.

Vital signs such as heart rate, blood pressure, and blood oxygen saturation did not differ significantly between groups. Only one patient reported having postoperative complications like fever. The incidence of adverse events like nausea, vomiting, delirium, and delayed recovery in the PACU did not differ significantly between groups (Table 4).

Discussion

Our research indicates that the incidence of LPR reached 39.3% among patients who underwent MECT. Having more than three MECT sessions, a history of acid regurgitation, succinylcholine doses over 50 mg, and oral secretion volumes above 3 mL were found to be significant and independent factors associated with the development of LPR. To our knowledge, this was the first study to determine the incidence of LPR in patients undergoing MECT and identify risk factors for LPR by detecting salivary pepsin levels.

Determining the incidence and prevalence of LPR is difficult because there is no definitive diagnostic test. In recent years, several diagnostic tools and biomarker expressions have been reportedly associated with LPR, including laryngoscopy, reflux monitoring, motility testing, inflammatory cytokines, carbonic anhydrase, and E-cadherin.[18,19] However, there is no consistently simple non-invasive and easily repeatable diagnostic tool, and these biomarkers are mainly associated with the pathophysiological changes associated with LPR, which are unknown.[19] Pepsin normally exists only in the stomach, but studies have confirmed its presence in the laryngeal mucosal epithelium of patients with LPR,[20] and that salivary pepsin values were significantly higher in the LPR versus control group.[21] Compared with oropharyngeal

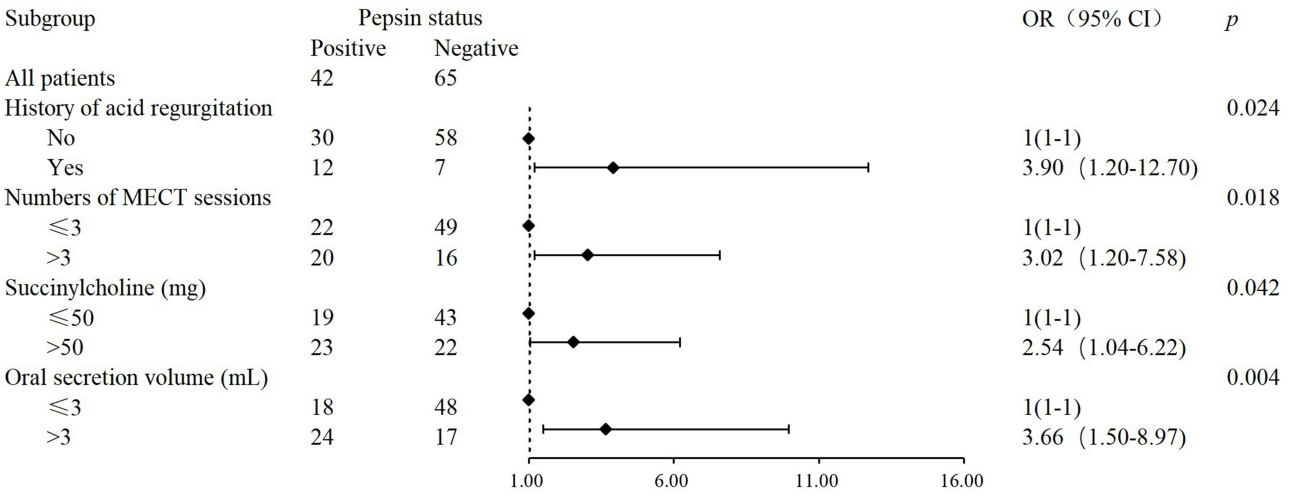

Figure 2 Forest plot of factors analysed to determine association with incidence of laryngopharyngeal reflux on multivariate logistic regression.
Abbreviations: CI, confidence interval; MECT, modified electroconvulsive therapy; OR, odds ratio.

pH monitoring, salivary pepsin could be an alternative tool to aid the diagnosis of LPR with a specificity of 86.2% and sensitivity of 41.5%.[22] Currently, dual pH monitoring and hypopharyngeal multichannel intraluminal impedance-pH (HMII-pH) monitoring can be used to diagnose LPR. In contrast to the salivary pepsin test, these methods provide dynamic reflux monitoring. However, these procedures are invasive, expensive, very unpleasant to patients, and may overlook cases with variable progression.[23] Thus, the salivary pepsin test is a simple, affordable, non-invasive, and easily repeatable tool with certain diagnostic value for LPR disease.[24] Additionally, the salivary pepsin test may be important during the perioperative period, as it helps identify patients at high risk for reflux aspiration, allowing for preventive measures to minimize the risk of reflux aspiration and postoperative pneumonia.

Table 4 Vital Signs and Adverse Events

Characteristics	Total	Pepsin		*P* value
		Positive	Negative	
	107	42(39.3%)	65(60.7%)	
Preoperative vital signs				
Heart rate (bpm)		81±15	84±15	0.333
DBP (mmHg)		66±99	68±12	0.315
SBP (mmHg)		110 (100–118)	107 (101–118)	0.762
MAP (mmHg)		80 (73–86)	80 (73–91)	0.266
SpO2 (%)		99 (98–99)	99 (97–99)	0.787
Vital signs after electrical stimulation				
Heart rate		115±22	118±21	0.465
DBP (mmHg)		129 (112–145)	125 (115–143)	0.594
SBP (mmHg)		85±15	83±13	0.426
MAP (mmHg)		97±17	96±16	0.626
SpO2 (%)		100 (100–100)	100 (100–100)	0.215
Side effects				
Fever				0.393
Yes	1 (0.9%)	1 (2.4%)	0 (0)	
No	106 (99.1%)	41 (97.6%)	65 (100%)	

Notes: Data are presented by mean±standard deviation, median (IQR) or n (%).
Abbreviations: SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure.

First, we confirmed that the history of acid regurgitation is associated with pepsin-positive saliva and that aspiration risk is high from a medical condition like LPR. Our present outcomes are consistent with the previous research that reflux disease increases the risk of aspiration of gastric contents during general anaesthesia. However, a previous study reported that most patients with mild or well-managed GERD do not have a higher risk of aspiration during MECT.[25] Gastric hypomotility can be induced by psychiatric illnesses like depression or schizophrenia as well as by the antipsychotic agents prescribed to treat these conditions.[26,27] However, psychiatric illnesses and antipsychotic medication use was not associated with an increased incidence of LPR in our study.

Second, a dose of succinylcholine > 50 mg seemed to pose a notable risk for LPR in our study. General anaesthetics and muscle relaxants are typically used during MECT, which can render patients unconscious with muscle paralysis and amnesia. Succinylcholine is used to prevent physical damage linked to motor seizures. However, the use of succinylcholine increases the oral secretions and MECT complications following general anaesthesia. Finding the right dosage is challenging since a low dose may not be effective, but a high dose could lengthen the apnoeic period and heighten the

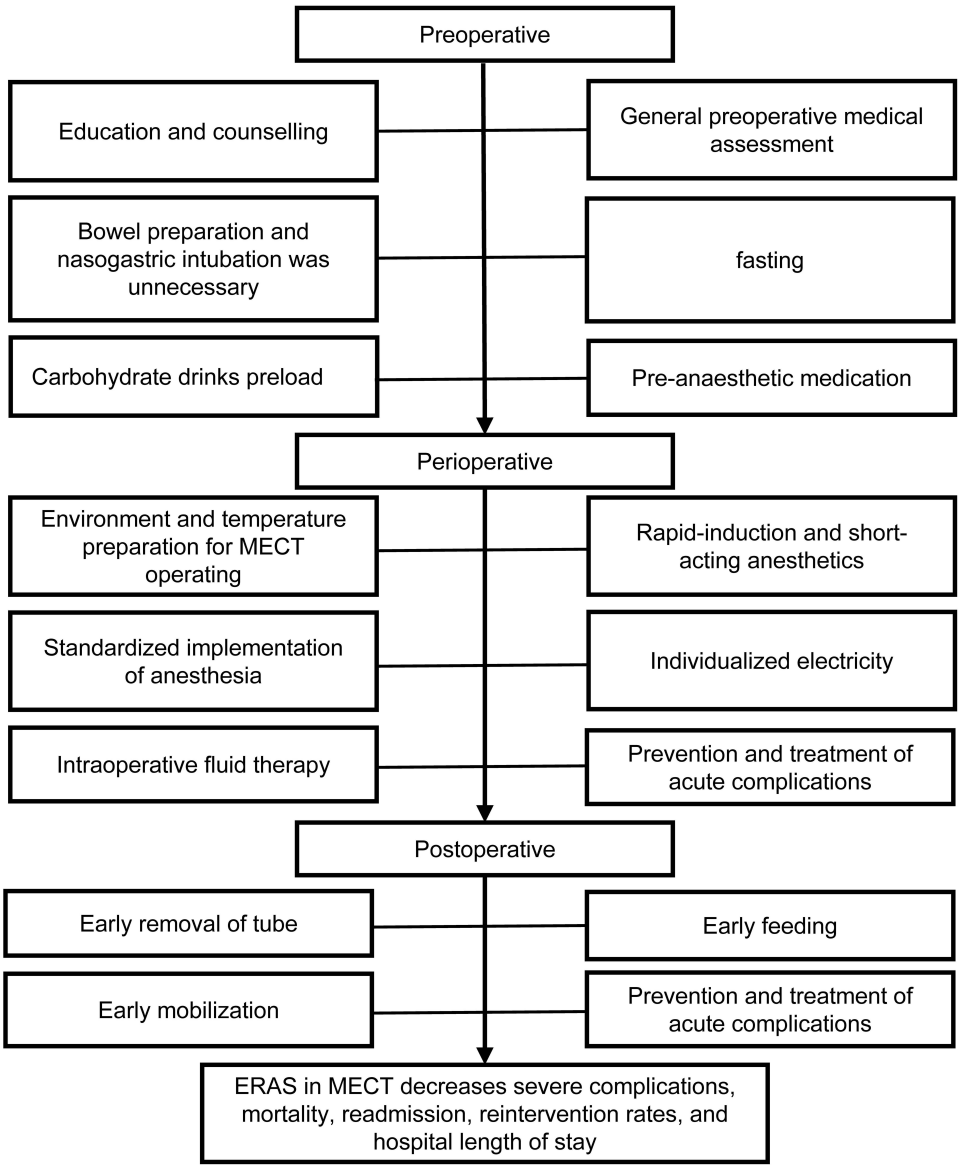

Figure 3 Flowchart of study treatment process.
Abbreviations: ERAS, enhanced recovery after surgery; MECT, modified electroconvulsive therapy.

https://doi.org/10.2147/TCRM.S549412

risk of complications. The mean (SD) appropriate dose of succinylcholine was found to be 0.96 (0.26) mg/kg in a retrospective analysis of charts from 500 patients,[28] which is similar to that in our study.

Third, completing >3 MECT sessions was an important factor in the development of LPR. MECT is typically administered 2–3 times weekly for 2–4 weeks, then continued less frequently for maintenance.[29] On the one hand, parasympathetic activation, lasting for a few seconds and characterised by excessive oral secretions, occurs immediately after the MECT stimulus. On the other hand, a subsequent sympathetic surge increases intragastric pressure during the seizure. Thus, a possible explanation for this finding is that the frequent activation of the parasympathetic and sympathetic nervous systems may contribute to the development of LPR.

The 39.3% observed incidence of LPR in our population was markedly higher than that reported by Wong et al (29.8%)[30] and El-Serag et al (18.1–33.1%),[31] but it was lower than that reported by Koufman et al (50%).[7] Aspiration leading to pneumonitis neurogenic pulmonary oedema and pulmonary embolism may rarely complicate MECT according to some case reports.[32,33] With less saliva, less fluid can be aspirated, probably reducing the risk of aspiration or inhalation pneumonia. In our medical centre, we are focused on established a unified and coordinated enhanced recovery after surgery (ERAS) management system.[34] We also conduct ERAS procedures during MECT (Figure 3). Penehyclidine hydrochloride medication 15–20 min before induction typically decreases saliva production. We also frequently suctioned the secretions during the perioperative period including deep suction (immediate, end of stimulation) and oral suction (upon occurrence of spontaneous breathing). We previously observed that patients have copious secretions requiring suctioning during treatment associated with postoperative respiratory complications. Fortunately, only one of our patients suffered from postoperative fever. Thus, frequent suctioning improved safety and patient comfort by preventing postoperative respiratory adverse events such as fever and pneumonia.

Our study has some limitations. With a larger sample, more independent predictors may have been identified among the investigated variables. Increasing the frequency of sampling could enhance the sensitivity of pepsin as a marker for aspiration during treatment. Due to the intermittent nature of aspiration episodes, sampling tracheal secretions from each patient more often might have improved our identification of pepsin-positive aspirates. Second, we did not study whether the administration of promotility agents or gastric acid–neutralising agents can reduce the frequency of gastroesophageal reflux. Despite the infrequent respiratory complications such as aspiration pneumonitis associated with MECT, it is recommended to pre-treat with medications aimed at reducing LPR effects, even for patients diagnosed with LPR.

Conclusions

In conclusion, we demonstrated the incidence of LPR reached 39.3% among patients who underwent MECT. A history of acid regurgitation, having completed >3 MECT sessions, doses of succinylcholine > 50 mg, and oral secretion volume > 3 mL were significantly associated with an increased risk of LPR among patients undergoing MECT.

Data Sharing Statement

The raw data supporting the conclusions of this article will be made available by the corresponding authors via email.

Ethics Approval and Informed Consent

This research complied with the Declaration of Helsinki and received approval from the Institutional Ethics Committee of the Third Affiliated Hospital of Sun Yat-sen University in Guangzhou, China (no. 2022-086-01). Informed consent was obtained from all participants.

Acknowledgments

The authors thank Xiangyang Zang, Jingting Zhang, and Chulian Gong for their help with collecting data and providing care for study patients.

Author Contributions

All authors made a significant contribution to the work reported, encompassing areas such as conception, study design, execution, data acquisition, analysis, and interpretation, or a combination thereof. All authors participated in drafting,

revising, or critically reviewing the manuscript, provided final approval for the version to be published, consented to the journal submission, and accepted accountability for all aspects of the work.

Funding

Project supported by the Special Support Project of Guangdong Province (Grant No.0720240209), Science and Technology Program of Guangzhou City (No.2024A04J4246), Joint Funds of the National Natural Science Foundation of China (No. U22A20276), Science and Technology Planning Project of Guangdong Province-Regional Innovation Capacity and Support System Construction (No. 2023B110006), and the "Five and fve" Project of the Third Afliated Hospital of Sun Yat-Sen University (No.2023WW501).

Disclosure

No conflicts of interest are declared by the authors.

References

1. Soehle M, Bochem J. Anesthesia for electroconvulsive therapy. *Curr Opin Anaesthesiol*. 2018;31(5):501–505. doi:10.1097/ACO.0000000000000624
2. Lisanby SH. Electroconvulsive therapy for depression. *N Engl J Med*. 2007;357(19):1939–1945. doi:10.1056/NEJMct075234
3. Lava-Parmele S, Lava C, Parmele JB. The historical struggles of modified electroconvulsive therapy: how anesthesia came to the rescue. *J Anesth Hist*. 2021;7(2):17–25. doi:10.1016/j.janh.2021.03.001
4. Andrade C, Arumugham SS, Thirthalli J. Adverse effects of electroconvulsive therapy. *Psychiatr Clin North Am*. 2016;39(3):513–530. doi:10.1016/j.psc.2016.04.004
5. Vakil N, van Zanten SV, Kahrilas P, Dent J, Jones R, Global Consensus Group. The montreal definition and classification of gastroesophageal reflux disease: a global evidence-based consensus. *Am J Gastroenterol*. 2006;101(8):1900–1920. doi:10.1111/j.1572-0241.2006.00630.x
6. Lechien JR, Akst LM, Hamdan AL, et al. Evaluation and management of laryngopharyngeal reflux disease: state of the art review. *Otolaryngol Head Neck Surg*. 2019;160(5):762–782. doi:10.1177/0194599819827488
7. Koufman JA, Amin MR, Panetti M. Prevalence of reflux in 113 consecutive patients with laryngeal and voice disorders. *Otolaryngol Head Neck Surg*. 2000;123(4):385–388. doi:10.1067/mhn.2000.109935
8. Lechien JR, Mouawad F, Bobin F, Bartaire E, Crevier-Buchman L, Saussez S. Review of management of laryngopharyngeal reflux disease. *Eur Ann Otorhinolaryngol Head Neck Dis*. 2021;138(4):257–267. doi:10.1016/j.anorl.2020.11.002
9. Li N, Yang WL, Cai MH, et al. Burden of gastroesophageal reflux disease in 204 countries and territories, 1990–2019: a systematic analysis for the Global Burden of disease study 2019. *BMC Public Health*. 2023;23(1):582. doi:10.1186/s12889-023-15272-z
10. Lindblad L, Nordenskjöld A, Otterbeck A, Nordenskjöld AM. Risk factors for mortality of medical causes within 30 days of electroconvulsive therapy. *J Affect Disord*. 2023;320:527–533. doi:10.1016/j.jad.2022.10.008
11. Gong EJ, Choi KD, Jung HK, et al. Quality of life, patient satisfaction, and disease burden in patients with gastroesophageal reflux disease with or without laryngopharyngeal reflux symptoms. *J Gastroenterol Hepatol*. 2017;32(7):1336–1340. doi:10.1111/jgh.13716
12. O'Shea R, Gaffney M, Kaare M, Fenton JE. Laryngopharyngeal reflux induced sleep-related laryngospasm. *Ir J Med Sci*. 2023;192(1):335–340. doi:10.1007/s11845-022-02934-x
13. Wang Y, Liang Q, Luo J, Miao H, Qin G, Bao Y. Laryngopharyngeal reflux in otolaryngology-head and neck surgery clinic: an epidemiological survey. *Ear Nose Throat J*. 2025;104(8):NP510–NP517. doi:10.1177/01455613241301601
14. Barillari MR, Giordano GM, Costa G, et al. Laryngopharyngeal reflux and psychological distress: a vicious cycle worth investigating. *Eur Arch Otorhinolaryngol*. 2025;282(6):3103–3113. doi:10.1007/s00405-025-09313-z
15. Lee K, Jenkins KD, Sparkle T. A narrative overview of current anesthetic drugs in electroconvulsive therapy. *Life*. 2021;11(9):981. doi:10.3390/life11090981
16. Pottkämper JC, Verdijk JP, Stuiver S, et al. Seizure duration predicts postictal electroencephalographic recovery after electroconvulsive therapy-induced seizures. *Clin Neurophysiol*. 2023;148:1–8. doi:10.1016/j.clinph.2023.01.008
17. Johnston N, Dettmar PW, Ondrey FG, Nanchal R, Lee SH, Bock JM. Pepsin: biomarker, mediator, and therapeutic target for reflux and aspiration. *Ann N Y Acad Sci*. 2018;1434(1):282–289. doi:10.1111/nyas.13729
18. Barona-Lleó L, Duval C, Barona-de Guzmán R. Salivary pepsin test: useful and simple tool for the laryngopharyngeal reflux diagnosis. *Acta Otorrinolaringol Esp*. 2018;69(2):80–85. doi:10.1016/j.otorri.2017.03.001
19. Wood JM, Hussey DJ, Woods CM, Watson DI, Carney AS. Biomarkers and laryngopharyngeal reflux. *J Laryngol Otol*. 2011;125(12):1218–1224. doi:10.1017/S0022215111002234
20. Maronian NC, Azadeh H, Waugh P, Hillel A. Association of laryngopharyngeal reflux disease and subglottic stenosis. *Ann Otol Rhinol Laryngol*. 2001;110(7 Pt 1):606–612. doi:10.1177/000348940111000703
21. Lee SH, Samuels T, Bock JM, Blumin JH, Johnston N. Establishment of an immortalized laryngeal posterior commissure cell line as a tool for reflux research. *Laryngoscope*. 2015;125(2):E73–77. doi:10.1002/lary.24952
22. Weitzendorfer M, Antoniou SA, Schredl P, et al. Pepsin and oropharyngeal pH monitoring to diagnose patients with laryngopharyngeal reflux. *Laryngoscope*. 2020;130(7):1780–1786. doi:10.1002/lary.28320
23. Lien HC, Lee PH, Wang CC. Diagnosis of laryngopharyngeal reflux: past, present, and future-a mini-review. *Diagnostics*. 2023;13(9):1643. doi:10.3390/diagnostics13091643

24. Wang J, Zhao Y, Ren J, Xu Y. Pepsin in saliva as a diagnostic biomarker in laryngopharyngeal reflux: a meta-analysis. *Eur Arch Otorhinolaryngol*. 2018;275(3):671–678. doi:10.1007/s00405-017-4845-8
25. Bryson EO, Aloysi AS, Farber KG, Kellner CH. Individualized anesthetic management for patients undergoing electroconvulsive therapy: a review of current practice. *Anesth Analg*. 2017;124(6):1943–1956. doi:10.1213/ANE.0000000000001873
26. Quick C, Kliem A, Berger S, et al. Gastric dysmotility in major depression. *Prog Neuropsychopharmacol Biol Psychiatry*. 2010;34(1):92–97. doi:10.1016/j.pnpbp.2009.10.003
27. Palmer SE, McLean RM, Ellis PM, Harrison-Woolrych M. Life-threatening clozapine-induced gastrointestinal hypomotility: an analysis of 102 cases. *J Clin Psychiatry*. 2008;69(5):759–768. doi:10.4088/jcp.v69n0509
28. Bryson EO, Kellner CH, Li EH, Aloysi AS, Majeske M. Extreme variability in succinylcholine dose for muscle relaxation in electroconvulsive therapy. *Australas Psychiatry*. 2018;26(4):391–393. doi:10.1177/1039856218761301
29. Espinoza RT, Kellner CH. Electroconvulsive Therapy. *N Engl J Med*. 2022;386(7):667–672. doi:10.1056/NEJMra2034954
30. Wong WM, Lai KC, Lam KF, et al. Prevalence, clinical spectrum and health care utilization of gastro-oesophageal reflux disease in a Chinese population: a population-based study. *Aliment Pharmacol Ther*. 2003;18(6):595–604. doi:10.1046/j.1365-2036.2003.01737.x
31. El-Serag HB, Sweet S, Winchester CC, Dent J. Update on the epidemiology of gastro-oesophageal reflux disease: a systematic review. *Gut*. 2014;63(6):871–880. doi:10.1136/gutjnl-2012-304269
32. Kurnutala LN, Kamath S, Koyfman S, Yarmush J, SchianodiCola J. Aspiration during electroconvulsive therapy under general anesthesia. *J ECT*. 2013;29(4):e68. doi:10.1097/YCT.0b013e318290fbd4
33. Zibrak JD, Jensen WA, Bloomingdale K. Aspiration pneumonitis following electroconvulsive therapy in patients with gastroparesis. *Biol Psychiatry*. 1988;24(7):812–814. doi:10.1016/0006-3223(88)90257-0
34. Chen C, Li X, Luo G, Zhou S, Hei Z. Realising the full potential of anaesthesiology to promote enhanced recovery after surgery programmes in China. *Br J Anaesth*. 2021;126(5):e157–e159. doi:10.1016/j.bja.2021.01.016