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Research article

Association of point-of-care lung ultrasound findings with 30-day pulmonary complications after cardiac surgery: A prospective cohort study

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ABSTRACT

Background: Several studies have shown that bedside lung ultrasound findings in postanaesthesia care units (PACUs) and intensive care units (ICUs) correlate with postoperative pulmonary complications(PPCs) after noncardiac major surgery. However, it remains unclear whether lung ultrasound findings can be used as early predictors of PPCs in patients undergoing cardiac surgery. The main aim of our study was to evaluate the relationship between early postoperative point-of-care lung ultrasound findings and PPCs after cardiac surgery.

Methods: Two board-certified physicians performed a point-of-care pulmonary ultrasound on cardiac surgery patients approximately 2 h after the patient was admitted to the ICU. Pulmonary complications occurring within 30 days postoperatively were recorded. Logistic regression modeling was used to analyze the relationship between lung ultrasound findings and PPCs.

Results: PPCs occurred in 61 (30.9 %) of the 197 patients. Lung ultrasound scores(LUS), number of lung consolidation(NLC), and depth of pleural effusion(DPE) were more significant in patients who developed PPCs ($P < 0.001$). According to the multivariate analysis, NLC≥3(aOR 2.71,95% CI 1.14–6.44; $p = 0.024$)and DPE >0.95(aOR 3.79,95%CI 1.60–8.99; $p = 0.002$) were found to be independently associated with PPCs during this study.

Conclusions: Our study demonstrated that DPE >0.95 and NLC ≥3 were associated with PPCs after cardiac surgery based on bedside lung ultrasound findings in the ICU. When these signs manifest perioperatively, the surgeon should be alerted and the necessary steps should be taken, especially if they present simultaneously.

1. Introduction

The incidence of postoperative pulmonary complications (PPCs) in patients who undergo cardiac surgery is as high as 30 % and is

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associated with prolonged postoperative hospital stays, morbidity, and mortality. Postoperative pulmonary complications include hypoxemia, pulmonary atelectasis, pulmonary infection, pleural effusion, and prolonged postoperative ventilatory support [1–3]. Recent advances in cardiac surgery techniques, perioperative anaesthesia management, and postoperative critical care have made it possible to manage postoperative pulmonary complications. Perioperative risk assessment is necessary because early identification of patients at risk for PPCs allows for earlier intervention. Despite clinical monitoring with bedside chest radiography, there is still a lack of effective indicators for early risk prediction of pulmonary complications after cardiac surgery.

Bedside lung ultrasound, as a tool for repeated noninvasive monitoring of ventilation status, has become an essential method for screening for perioperative pleural effusion, pneumothorax, pulmonary oedema, and alveolar consolidation, with higher sensitivity and specificity than bedside chest radiography and a more complete set of theoretical and diagnostic guidelines [4–6]. Lung ultrasound is performed by transthoracic emission of ultrasound waves that interact between air, pleura, and fluid and produce signs associated with various pathophysiologic processes [7]. Some studies have shown that bedside pulmonary ultrasound findings in the post-anaesthesia care unit(PACU) and intensive care unit(ICU) correlate with pulmonary complications after major noncardiac surgery [8–10]. Nevertheless, it remains unclear whether lung ultrasound findings can be used as early predictors of PPCs in cardiac surgery patients.

The main aim of our study was to evaluate the relationship between early bedside lung ultrasound findings in the ICU(ICU-LUS) after cardiac surgery and PPCs, which may aid in early detection and treatment.

2. Materials and methods

2.1. Study design and study population

The Medical Ethics Committee of the Affiliated Hospital of Xuzhou Medical University approved this prospective observational study on November 04, 2022 (XYFY2022-KL367-01). Before patient recruitment, this study was registered with the Chinese Clinical Trials Registry (ChiCTR2200066690, registration date: December 14, 2022). Written informed consent was obtained from all patients prior to enrollment. This manuscript adheres to the STROBE statement guidelines [11].

A prospective cohort of 197 adult patients having elective open heart surgery between December 2022 and September 2023 were included in this study. The exclusion criteria were as follows: body mass index (BMI) $\geq$ 40 kg/m2; preoperative CT examination of the chest showing abnormalities (pulmonary atelectasis, lung infection, pneumothorax, pleural effusion, etc.); history of open thoracic surgery; preoperative comorbidities of severe liver and renal dysfunction; unavailable operator; refused to participate in the present study or experienced difficulty communicating preoperatively; underwent a second surgery postoperatively; poor quality of lung ultrasound images; and inability to complete the postoperative examination because of haemodynamic instability.

2.2. Study protocol

Chest CT was performed 30 days before surgery to rule out pre-existing lung pathologies, including pulmonary atelectasis. The anesthetic protocol was performed according to our standard procedures. After tracheal intubation and connection to the anaesthesia machine (Dräger, Germany), we performed lung-protective ventilation during surgery. The respiratory mode was volume-controlled ventilation with a fresh gas flow of 2 L/min,an inspiratory-to-expiratory ratio of 1:2, a tidal volume of 6–8 ml/kg of the ideal body weight, a PEEP of 5 cmH2O, and an inhaled oxygen concentration of 40–60 %. The respiratory rate (RR) was set to 12 breaths/minute, and the end-tidal carbon dioxide pressure was maintained between 33 and 45 mmHg. During cardiopulmonary bypass, mechanical ventilation was suspended. The same analgesic strategy was used for all patients after surgery. The same pulmonary ventilation strategy was continued after the patient was transferred from the operating room to the ICU.

Data collected preoperatively: age, gender, smoking and alcohol consumption, body mass index (BMI), comorbidities (hypertension, diabetes mellitus, myocardial infarction, and sleep apnea syndrome, etc.), serum hemoglobin, serum albumin, serum creatinine, left ventricular ejection fraction(LVEF), pulmonary function markers (FVC, FEV1, and FEV1/FVC), and the relevant perioperative risk assessment tools [European System for Cardiac Operative Risk Evaluation II(EuroSCORE II) [12], Clinical Frailty Scale(CFS) [13], American Society of Anesthesiologists class(ASA class) and New York Heart Association grade(NYHA grade)].

Intraoperative data included type of procedure, bypass time, cross-clamp time and duration of surgery (from incision to end of surgery), blood product transfusion and blood loss, urine output, and total intraoperative fluid intake and output.

The following vital signs were collected in the ICU: mean arterial pressure (MAP), heart rate (HR), pulmonary compliance, hourly urine output, lactate level, oxygenation index (OI), and delay between ICU admission and the practice of ultrasound. All these variables were collected at the beginning of the lung ultrasound examination.

2.3. Lung ultrasonography

After the patient was admitted to the ICU and stabilized for approximately 2 h, an anaesthesiologist with two years of experience in lung ultrasound and an ICU physician with more than five years of experience in lung ultrasound performed an ultrasound on the patient's lungs. A Philips CX-50 ultrasound machine was used, and all measurements were performed with the patient in the supine position, at which time the patient remained in the unawakened state under general anaesthesia, and the patient was admitted to the ICU under sedation and analgesia with propofol and remifentanil, respectively. The patient remained under complete ventilatory support, and there was no spontaneous respiration. Each of the two physicians who performed the ultrasound recorded and interpreted

the condition of all the lung segments. After discussion between the two physicians, a final result of the lung ultrasound examination was obtained. They were blinded to the results of the postoperative follow-up. The ICU physicians at our study centre determined the patients' lung ventilation status by bedside chest radiographs or chest CT, and performed lung ultrasound at bilateral diaphragmatic points only when the patients left the ICU to test for pleural effusions that required management.

The lungs were scanned transversely between the intercostal spaces using a 2–6 MHz convex array probe to obtain the appropriate lung ultrasound signs. For each region, the most severe finding was scored. In the supine position, the paraxillary, anterior and posterior axillary lines (vertical) and the nipple and diaphragm lines (horizontal) were used as markers; one side of the lung was divided into 6 regions, with a total of 12 regions in both lungs, similar to a previous study [7]. The 12 lung regions were scanned sequentially from right to left and from head to foot, and we scanned the posterior region behind the posterior axillary line rather than the paravertebral region so as to avoid rotating the patient, a minimum of 2 clips were stored for each lung examination region [7].

The LUS was calculated as the sum of the integral values for each scan site [0 = normal scan(Fig. 1a), 1 = moderate interstitial syndrome(Fig. 1b), 2 = severe interstitial syndrome (multiple or combined B-lines,Fig. 1c), 3 = alveolar consolidation(Fig. 1d/e/f)] [14], Fig. 1. A score ranging from 0 to 36 was then calculated. Two examiners examined each lung field for evidence of alveolar consolidation, interstitial syndrome, pneumothorax, pleural effusion, or pleural irregularities and simultaneously measured and calculated the total number of lung consolidations(NLC), B-line score, and depth of pleural effusion(DPE). All counts of the number of lung ultrasound signs were based on bilateral lung.

In this case, unlike the B-line scoring rules in the LUS system, the B-line score was based on a study by Enghard [15] et al. (Supplementary Table 1). The depth of pleural effusion(DPE) was measured as follows [16]: the patient was placed in the supine position and the trunk slightly elevated by 15°. The probe was moved upwards along the posterior axillary line to obtain a cross-section parallel to the intercostal space to visualize the pleural separation at the base of the lung. The maximum distance between the wall and the visceral pleura at the end of expiration was recorded(Fig. 1f).

2.4. Primary outcome

The diagnostic criteria for PPCs were based on a study by Kroenke et al. [17], Supplementary Table 2 in which pulmonary complications, mainly including pulmonary infection, reintubation, prolonged mechanical ventilation (>24 h), thoracentesis drainage, and ARDS were classified as grade 2 or higher complications, which mainly included pulmonary infection, reintubation, prolonged mechanical ventilation (>24 h), thoracentesis drainage, and ARDS. During the 30-day postoperative follow-up (telephone follow-up after discharge), an anaesthesiologist, without knowing the results of the lung ultrasound examination, visited the patients in the ward or ICU every day to collect information on whether the patients had chest tightness, breathlessness, dyspnoea, abnormal breath sounds on auscultation of the lungs bilaterally, whether the oxygen saturation level had decreased to less than 90 % (with an oxygen flow rate of 4–6 L/min), the characteristics and volume of sputum, chest imaging data (X-ray and chest CT), laboratory test results (inflammatory markers and sputum culture results), and clinical medication. Finally, two chief physicians of the Department of Critical Care

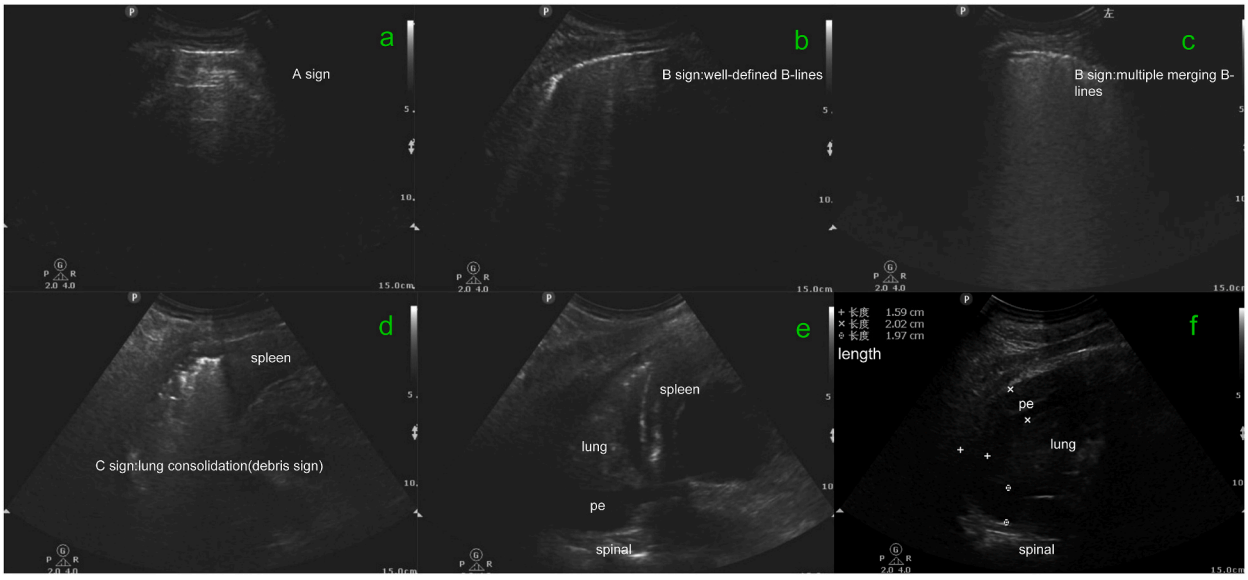

Fig. 1. (a) Parallel A-lines or fewer than two isolated B-lines, (b) well-defined B-lines, (c) multiple merged B-lines; (d) C sign (lung consolidation): Debris sign (slight lung atrophy without large pleural effusion); (e) C sign (lung consolidation) - Compressive atelectasis formed by large amounts of pleural effusion (with compressed lung tissue in the centre), pe: pleural effusion; (f) Ultrasound probe scanning of the axillary line behind the transverse section above the base of the lung. After cardiac surgery, the patient had consolidation of the lower lobes of the lung, and the depth of pleural effusion was 1.97 cm paravertebrally, 1.59 cm dorsally, and 2.02 cm laterally, so the final maximum depth of pleural effusion was 2.02 cm.

Medicine synthesized and analysed all these clinical data and determined whether the patients met the criteria for PPCs.

2.5. Secondary outcomes

We also collected information on acute kidney injury(AKI) status, length of ICU stay, postoperative length of hospital stay, readmission within 30 days of surgery, and disability or death after 30 days (obtained by telephone follow-up after discharge). At the 30-day postoperative follow-up, patient survival status was assessed using the validated World Health Organization Disability Assessment Scale (WHODAS) 2.0. A score ≥12 was defined as disability [18]. According to the Kidney Disease Improvement Global Prognosis Organization (KDIGO) guidelines [19], acute kidney injury was defined as an increase in blood creatinine of at least 26.5 μmol/L within 48 h; an increase in blood creatinine of at least 1.5 times the basal value over a 7-d period; or a urine output of less than 0.5 ml/(h-kg) over a 6-h period.

2.6. Statistical analysis

Data normality was tested by visual inspection of histograms and Shapiro-Wilk's W test. All normally distributed and skewed continuous variables were expressed as mean(SD)or median (interquartile range [IQR]). Categorical variables were indicated as frequencies (%). Comparison of continuous variables among groups was performed with the use of the Student's t-test or Mann–Whitney *U* test, depending on the normality of the distribution. In contrast, the Fisher's Exact test was used to compare categorical variables. One-way ANOVA or Chi-square test was used when three groups were compared. We randomly selected 30 patients' LUS scores for consistency analysis, and the Kendall consistency coefficient was used to analyze inter-observer consistency(These randomly selected data are undiscussed lung ultrasound findings).

A least absolute shrinkage and selection operator regression analysis was conducted with statistically significant risk factors included in the univariable study to remove nonzero characteristic components. Afterwards, multivariate logistic regression analysis(a stepwise regression method) was used to identify the risk variables for PPCs. Internal validation was performed using the bootstrap self-sampling approach (1000 bootstrap samples repeated sampled), and the relatively corrected C-index (concordance index) was generated to test the model's discrimination ablility. The calibration curve was plotted to assess the model's consistency. The area under the receiver operating characteristic (ROC) curve (AUC) was used to evaluate the predictive validity. An AUC between 0.5 and 0.7 indicates poor prediction performance, while an AUC between 0.7 and 0.9 indicates good prediction performance. We compared event rates according to the presence of DPE >0.95 or (and) NLC≥3. We used Kaplan-Meier curves to characterize their cumulative incidence of PPCs and weaning from mechanical ventilation, and we used the log-rank test-to compare differences.

Because the percentage of missing data was small(0%–5%), no imputation was performed. P-value <0.05 (two-sided) was considered statistically significant. R4.1.2 and SPSS 26. statistical software was used for analysis.

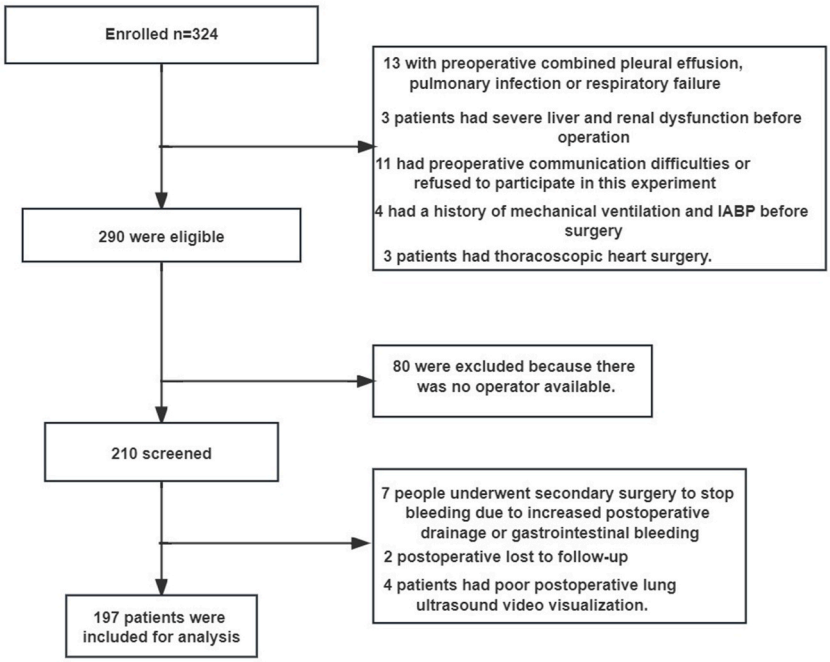

Fig. 2. Flow chart of patients. IABP intra aortic balloon pump.

2.7. Sample size calculating

Based on the results of the pilot study, the incidence of pulmonary complications after cardiac surgery in our study center was approximately 30 %. According to the 10EPV principle, the multivariate analysis included a total of 5 variables, resulting in 10*5/0.3 = 167, and considering a drop-out rate of 15 %, 196 patients will be included in this study.

3. Results

3.1. Population characteristics

Between December 2022 and September 2023, a total of 324 patients were admitted to the ICU after cardiac surgery, 197 of whom were eligible for enrolment (Fig. 2). The baseline and perioperative characteristics are shown in Tables 1 and 2. The patients in the PPCs group had higher preoperative age, EuroSCORE II, ASA class, NYHA grade, frailty rate, and lower lung function (FVC, FEV1, and FEV1/FVC%) than did those in the non-PPCs group ($P < 0.05$). Between-group differences at the intraoperative level indicated that

Table 1
Baseline and surgical characteristics of the study cohort.

Variables	Total (n = 197)	Non-PPCs (n = 136)	PPCs (n = 61)	p
Age, years	62.9 ± 9.0	61.4 ± 9.3	66.0 ± 7.7	<0.001
Sex, female	65 (33.0)	41 (30.1)	24 (39.3)	0.204
BMI, kg/m^2	24.6 ± 3.6	24.6 ± 3.1	24.5 ± 4.6	0.841
Alcohol	56 (28.4)	42 (30.9)	14 (23)	0.254
Smoke	73 (37.1)	53 (39)	20 (32.8)	0.406
LVEF	58.4 ± 8.0	59.0 ± 7.9	57.0 ± 8.1	0.099
FEV_1, L	2.4 ± 0.8	2.5 ± 0.7	2.0 ± 0.8	<0.001
FVC, L	2.6 ± 1.0	2.8 ± 1.0	2.3 ± 0.9	0.001
FEV_1/FVC, %	89.8 ± 13.3	91.6 ± 8.3	86.0 ± 19.8	0.008
Type of surgery				0.111
CABG	95 (48.2)	70 (51.5)	25 (41)	
Valve	88 (44.7)	60 (44.1)	28 (45.9)	
CABG + Valve	10 (5.1)	4 (2.9)	6 (9.8)	
Aortic	4 (2.0)	2 (1.5)	2 (3.3)	
Comorbidity				
PAH	51 (25.9)	31 (22.8)	20 (32.8)	0.139
MI	20 (10.2)	14 (10.3)	6 (9.8)	0.922
Stroke/TIA	58 (29.4)	36 (26.5)	22 (36.1)	0.172
Diabetes	34 (17.3)	24 (17.6)	10 (16.4)	0.83
Hypertension	102 (51.8)	67 (49.3)	35 (57.4)	0.292
Cough and sputum	43 (21.8)	30 (22.1)	13 (21.3)	0.907
Sleep apnoea	20 (10.2)	11 (8.1)	9 (14.8)	0.152
Bronchial disease	13 (6.6)	6 (4.4)	7 (11.5)	0.116
Haemoglobin, g/L	135.7 ± 16.9	136.3 ± 16.3	134.3 ± 18.1	0.425
Albumin, g/L	43.8 ± 26.6	45.0 ± 32.0	41.4 ± 3.8	0.386
Creatinine, μmol/L	64.8 ± 18.6	63.4 ± 18.2	67.7 ± 19.3	0.133
Risk assessment tool				
EuroSCORE II,%	1.3 ± 1.0	1.1 ± 0.7	1.8 ± 1.3	<0.001
CFS				<0.001
1-3	151 (76.6)	116 (85.3)	35 (57.4)	
≥4	46 (23.4)	20 (14.7)	26 (42.6)	
NYHA				<0.001
I-II	141 (71.6)	108 (79.4)	33 (54.1)	
III-IV	56 (28.4)	28 (20.6)	28 (45.9)	
ASA Class				0.002
2–3	175 (88.8)	127 (93.4)	48 (78.7)	
4	22 (11.2)	9 (6.6)	13 (21.3)	
Off-pump surgery	73 (37.1)	55 (40.4)	18 (29.5)	0.142
Bypass time, min	88.0 (0.0, 129.0)	71.5 (0.0, 118.2)	118.0(0.0,181.0)	<0.001
Cross-clamp time,min	60.0 (0.0, 89.0)	46.5 (0.0, 74.2)	86.0 (0.0, 130.0)	<0.001
Time of operation, h	4.8 (4.0, 5.8)	4.7 (4.0, 5.5)	5.2 (4.5, 6.6)	0.004
Urine output, ml	1645.7 ± 828.2	1644.7 ± 836.2	1648.0 ± 816.9	0.979
Blood loss, ml	540.3 ± 384.1	515.4 ± 349.0	595.9 ± 451.1	0.175
Blood transfusion	59 (29.9)	36 (26.5)	23 (37.7)	0.111
Total liquid output, ml	2134.2 ± 863.1	2138.6 ± 841.4	2124.4 ± 916.7	0.916
Total fluid intake, ml	3400.7 ± 1327.2	3335.6 ± 1388.6	3545.8 ± 1176.8	0.305

The data are presented as the means ± standard deviations, absolute rates, and percentages. Statistical significance was defined as a P value < 0.05. BMI body mass index; LVEF left ventricular ejection fraction; FVC forced vital capacity; FEV1 forced expiratory volume in 1 s; CABG coronary artery bypass graft; PAH pulmonary arterial hypertension; MI myocardial infarction; TIA transient ischaemic attacks; EuroSCORE II European System for Cardiac Operative Risk Evaluation II; CFS Clinical Frailty Scale; ASA American Society of Anaesthesiologists; NYHA New York Heart Association.

Table 2
Postoperative lung ultrasound findings and secondary and primary outcome indicators.

Variables	Total (n = 197)	Non-PPCs (n = 136)	PPCs (n = 61)	p
Delay between ICU admission and the practice of ultrasound (h)	2.1 (1.7, 2.4)	2.1 (1.8, 2.4)	2.1 (1.7, 2.3)	0.802
LUS score total	10.4 ± 5.6	9.0 ± 5.0	13.6 ± 5.6	<0.001
LUS score anterior	0.0 (0.0, 2.0)	0.0 (0.0, 2.0)	0.0 (0.0, 2.0)	0.081
LUS score lateral	3.0 (0.0, 5.0)	2.0 (0.0, 4.0)	4.0 (2.0, 6.0)	<0.001
LUS score posterior	6.5 ± 3.1	5.8 ± 2.9	8.1 ± 2.9	<0.001
B-lines score	15.0 (10.0, 24.0)	15.0 (10.0, 23.0)	16.0 (10.0, 24.0)	0.450
NLC	2.0 (1.0, 3.0)	1.0 (0.0, 2.0)	3.0 (2.0, 4.0)	<0.001
NLC≥3	67 (34.0)	30 (22.1)	37 (60.7)	<0.001
DPE,cm	0.5 (0.1, 1.2)	0.4 (0.0, 0.8)	1.2 (0.5, 1.8)	<0.001
DPE>0.95	62 (31.5)	25 (18.4)	37 (60.7)	<0.001
Clinical features in ICU				
Cdyn, ml/cmH_2O	29.9 ± 5.2	30.8 ± 4.9	27.9 ± 5.5	<0.001
MAP, mmHg	84.6 ± 14.5	83.9 ± 14.8	86.0 ± 13.6	0.354
Heart rate, times/minute	84.9 ± 15.3	82.7 ± 15.3	89.8 ± 14.4	0.003
Oxygenation index	287.6 ± 100.0	308.1 ± 100.0	242.2 ± 84.4	<0.001
Urine volume,ml/h	209.9 ± 117.2	221.3 ± 117.6	184.5 ± 113.2	0.041
IVCD,cm	2.1 ± 0.2	2.1 ± 0.2	2.1 ± 0.2	0.202
ICU Stay, h	17.0 (17.0, 18.0)	17.0 (17.0, 18.0)	18.0 (17.0, 65.0)	<0.001
Length of stay, days	9.0 (8.0, 11.0)	8.0 (8.0, 10.0)	12.0 (9.0, 18.0)	<0.001
AKI	28 (14.2)	7 (5.1)	21 (34.4)	<0.001
WHODAS-12	6.0 (4.0, 13.0)	5.0 (3.0, 8.0)	13.0 (5.0, 30.0)	<0.001
Disability or death	56 (28.4)	23 (16.9)	33 (54.1)	<0.001
Mortality within 30	12 (6.1)	1 (0.7)	11 (18)	<0.001
Readmission within 30 days	18 (9.1)	8 (5.9)	10 (16.4)	0.018

P value from unadjusted analysis. Values are expressed as absolute numbers (percentages) or medians [interquartile ranges].LUS Lung ultrasound score; NLC Number of lung consolidation; DPE Depth of pleural effusion; Cdyn dynamic lung compliance; MAP mean arterial pressure; IVCD internal diameter of the inferior vena cava; ICU intensive care unit; AKI acute kidney injury; WHODAS-12 WHO disability assessment schedule 2.0: 12-part questionnaire.

patients who developed PPCs had longer bypass times, cross-clamp times, and operation times ($P < 0.05$). During the postoperative ICU period, the lung ultrasound score, number of lung consolidations (NLC), and depth of pleural effusion(DPE) were significantly greater in patients who developed PPCs ($P < 0.001$), and the length of ICU stay, length of hospital vstay, and duration of mechanical ventilation were significantly longer ($P < 0.001$). In addition, the patients in the PPC group had higher rates of acute kidney injury, readmission, and death or disability ($P < 0.05$).

3.2. Description of early bedside lung ultrasound findings

The LUS was 10.4 ± 5.6 for the patients with PPCs and 9.0 ± 5.0 for the patients without PPCs ($P < 0.001$). To predict PPCs, estimation by maximizing the Youden index using a LUS cutoff value ≥ 10 yielded an area under the curve of 0.69 (95 % CI 0.61–0.77). ROC analysis of the number of lung consolidation(NLC) and depth of pleural effusion(DPE) predicting PPC showed that the unweighted Youden J statistic was maximized when the NLC threshold was 2.5 and the DPE threshold was 0.95, respectively. This value was used to categorize patients into two groups for multivariate logistic regression. In addition, DPE and NLC showed better efficacy in

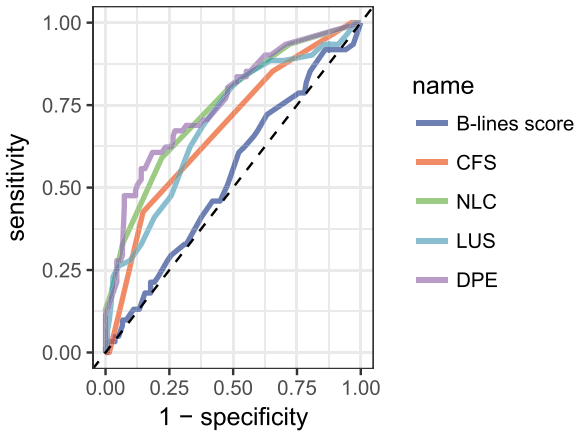

Fig. 3. Receiver operating characteristic curve with various variables predicting PPCs.(NLC Number of lung consolidation; DPE Depth of pleural effusion; LUS Lung ultrasound score; CFS Clinical Frailty Scale.)

predicting PPCs with areas under the curve of 0.76 and 0.74, respectively. B-line scores had poor predictive efficacy for PPCs in our study[area under the curve of 0.53 (95 % CI 0.45–0.62)]. Similarly, a CFS score≥ 4 indicated frailty,(Fig. 3 and Supplementary Table 3.

Lung ultrasound findings revealed an immediate NLC≥3 in 67 (34 %) patients and a DPE >0.95 in 62 (31.5 %) patients. ICU-LUS was performed after ICU admission of 2.1 h (interquartile range [IQR], 1.7–2.4) (Table 2). Kendall's consistency coefficient analysis was applied to the lung ultrasound score data of the two groups to evaluate differences in inter-observer agreement. The results showed that the interobserver agreement was 0.827, indicating robust interobserver agreement.

3.3. Variables independently associated with PPCs(Primary outcome) in multivariable analysis

The incidence of PPCs was 30.96 %. According to the least absolute shrinkage and selection operator regression analysis (Supplementary Fig. 1), we selected five nonzero characteristic variables, namely frailty, NLC≥3, cross-clamp time, AKI, and DPE >0.95 (Supplementary Table 4). The variables associated with PPCs are shown in Table 3. According to the multivariate analysis, frailty(aOR 3.16,95 % CI 1.32–7.59; p = 0.01), NLC≥3(aOR 2.71,95 % CI 1.14–6.44; p = 0.024),cross-clamp time(aOR 1.01,95 % CI 1.00–1.02; p = 0.02), AKI(aOR 6.86,95 % CI 2.25–20.91; p = 0.001), and DPE >0.95(aOR 3.79,95 % CI 1.60–8.99; p = 0.002) were found to be independently associated with PPCs in this study. We used calibration and discrimination with a 1000-sample bootstrapping technique to provide bias-corrected concordance statistics to validate the internal validity of our basic model. Our calibration curve (Supplementary Fig. 2) shows that our model is well-calibrated. The discriminative ability had a C-statistic of 0.84 and an optimism-corrected C-statistic of 0.82, indicating that the model performed well in predicting PPCs.

The population was divided into three groups based on whether the DPE was >0.95 or the NLC was ≥3 (Table 4). Fig. 4 shows the cumulative incidence of PPCs(Fig. 4A) and weaning from mechanical ventilation(Fig. 4B) in the presence of DPE>0.95 or(and) NLC≥3. All patients were weaned from mechanical ventilation except for those who died. Loss of lung ventilation increases the incidence of PPCs and the duration of mechanical ventilation(p < 0.001, Table 4 and Fig. 4).

3.4. Sensitivity analysis

According to the definition of PPCs, we excluded patients who underwent postoperative closed thoracic drainage and developed pulmonary atelectasis, obtaining a new study population (N = 173), for whom the prevalence of PPCs was 21.4 %. Multivariate logistic regression analysis revealed that the NLC was independently associated with PPCs (aOR 3.07,95 % CI 1.08–8.71; p = 0.035), Supplementary Table 5.

3.5. Secondary outcomes

A DPE>0.95 and NLC≥3 were associated with a lower oxygenation index, longer length of hospital stay, longer ICU stay, and higher risk of disability or death (p < 0.05, Table 4).

4. Discussion

We discovered that ICU-LUS results of DPE>0.95 and NLC≥3 may be related to PPCs following cardiac surgery in this single-centre study of LUS performed approximately 2 h after ICU admission. In addition, DPE >0.95 cm combined with NLC≥3 was associated with prolonged postoperative mechanical ventilation, prolonged ICU stay, prolonged length of hospital stay, and short-term postoperative disability or death.

The results of our study are similar to previous findings in that the diagnosis of lung consolidation and pleural effusion using bedside lung ultrasound in the ICU (ICU-LUS) is both rapid and reliable. In a 2018 study, Touw et al. [20] repored that the use of ICU-LUS was earlier and more accurate than bedside X-rays in diagnosing clinically relevant pulmonary complications in patients after adult cardiac surgery. However, that study used the ICU-LUS as a tool to diagnose PPCs. It did not investigate whether the number of lung consolidations and depth of pleural effusion were independent risk factors for PPCs. The follow-up time for postoperative pulmonary complications was only 3 days. In contrast, our study considered the ICU-LUS to be a tool for predicting PPCs and patients were followed up for 30 days to assess the occurrence of PPCs. In addition, in the 2020 study of non-cardiac postoperative patients, Zieleskiewicz et al. [8]. reported that postoperative alveolar consolidation and pleural effusion detected early by PACU(Postanaesthesia care unit)-LUS were associated with pulmonary complications up to 8 days postoperatively. The idea behind our experiment is similar,

Table 3
Variables independently correlated with postoperative pulmonary complications as determined by logistic regression.

Variable	crude.OR_95CI	crude.P_value	adj.OR_95CI	adj.P_value
NLC≥3	5.45 (2.83–10.48)	<0.001	2.71 (1.14–6.44)	0.024
DPE>0.95	6.85 (3.49–13.41)	<0.001	3.79 (1.6–8.99)	0.002
AKI	9.67 (3.83–24.42)	<0.001	6.86 (2.25–20.91)	0.001
Cross-clamp time	1.01 (1.01–1.02)	<0.001	1.01 (1~1.02)	0.02
(Frailty)CFS ≥4	4.31 (2.15–8.63)	<0.001	3.16 (1.32–7.59)	0.01

NLC Number of lung consolidation; DPE Depth of pleural effusion; AKI acute kidney injury; CFS Clinical Frailty Scale.

Table 4
Postoperative clinical outcome was determined by the number of lung consolidations≥3 or(and) the depth of pleural effusion >0.95.

Variables	Normal (n = 112)	DPE>0.95/NLC≥3 (n = 41)	DPE>0.95&NLC≥3 (n = 44)	p
PPCs grade≥2	17 (15.2)	14 (34.1)	30 (68.2)	<0.001
Hypoxemia	6 (5.4)	8 (19.5)	22 (50)	<0.001
Pulmonary infection	12 (10.7)	5 (12.2)	12 (27.3)	0.028
Pleural drainage	3 (2.7)	4 (9.8)	12 (27.3)	<0.001
PMV(>24 h)	5 (4.5)	3 (7.3)	9 (20.5)	0.007
Reintubation	2 (1.8)	1 (2.4)	4 (9.1)	0.11
Atelectasis	1 (0.9)	2 (4.9)	8 (18.2)	0.001
Pneumothorax	0 (0)	2 (4.9)	2 (4.5)	0.037
Bronchospasm	2 (1.8)	3 (7.3)	4 (9.1)	0.052
Level of PPCs				<0.001
0	43 (38.4)	8 (19.5)	2 (4.5)	
1	52 (46.4)	19 (46.3)	12 (27.3)	
2	2 (1.8)	4 (9.8)	7 (15.9)	
3	10 (8.9)	7 (17.1)	16 (36.4)	
4	5 (4.5)	3 (7.3)	7 (15.9)	
MVT,h	9.0 (6.5, 13.0)	11.5 (7.5, 16.0)	14.2 (9.8, 20.4)	<0.001
Oxygenation index	310.6 ± 103.8	279.0 ± 85.4	237.2 ± 83.6	<0.001
ICU Stay,h	17.0 (17.0, 18.0)	17.0 (17.0, 18.0)	18.0 (17.0, 48.0)	0.001
Length of stay, days	8.0 (8.0, 10.2)	9.0 (8.0, 10.0)	11.0 (9.0, 17.2)	<0.001
WHODAS-12	5.0 (3.0, 9.0)	6.0 (4.0, 12.0)	11.0 (5.5, 18.2)	<0.001
Disability or Death	23 (20.5)	11 (26.8)	22 (50)	0.004
Mortality within 30	5 (4.5)	1 (2.4)	6 (13.6)	0.073
Readmission within 30 days	5 (4.5)	9 (22)	4 (9.1)	0.006

NLC Number of lung consolidation; DPE Depth of pleural effusion; PMV prolonged mechanical ventilation; MVT mechanical ventilation time; ICU intensive care unit; WHODAS-12 WHO disability assessment schedule 2.0: 12-part questionnaire.

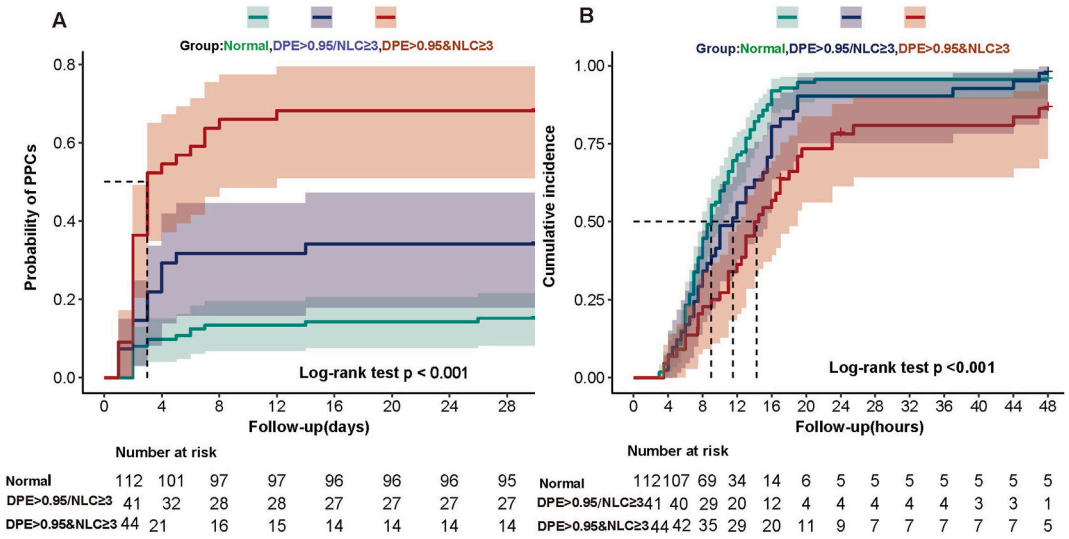

Fig. 4. (A):Kaplan-Meier curve of the probability of PPCs by postoperative day 30.(B):Kaplan-Meier curve of the probability of weaning from mechanical ventilation by postoperative hour 48 h.(DPE depth of pleural effusion; NLC number of lung consolidation)

but unlike in this study, we took NLC≥3 and DPE>0.95 as risk factors to be included in the multivariate logistic regression analysis because postoperative lung injury after cardiac surgery is more serious [21], basically all of them are accompanied by different degrees of alveolar consolidation and pleural effusions. Still, smaller areas of lung atelectasis and a small amount of pleural effusion will not affect the patient's prognosis. Therefore, our study suggested that clinicians should be vigilant when NLC ≥3 and/or DPE >0.95 are present, especially when both are present. Future studies need to measure the impact of prophylactic treatments such as ICU-LUS-guided recruitment manoeuvers, different levels of positive end-expiratory pressure (PEEP), continuous positive airway pressure (CPAP), high-flow nasal oxygen (HFNO), and particular positions on PPCs.

There is a relationship between pleural fluid content and the number of lung consolidation to some extent, as pleural fluid can cause compressive pulmonary atelectasis [22], with the lung ultrasound sign appearing as a quadrilateral or jellyfish sign [23]. However, lung consolidation may not be accompanied by a large amount of pleural effusion in absorptive and obstructive atelectasis. It may be characterized by discontinuity of the pleura or (and) disappearance or diminution of the pleural sliding sign (e.g., debris sign and tissue

sign) on lung ultrasound [24]. Therefore, some patients have an increased number of lung consolidations but do not necessarily have a large amount of pleural effusion. A higher number of lung consolidations combined with a larger amount of pleural effusion, indicates a more severe loss of pulmonary ventilation, and the patient is more likely to have higher risks of PPCs and prolonged mechanical ventilation.

Unlike previous studies [9,10], we focused on the depth of pleural effusion and the number of lung consolidations rather than a comprehensive lung ultrasound score because they are faster to caculate and more predictive of postoperative PPCs than lung ultrasound scores(Fig. 3 and Supplementary Table 3). The present study showed that the loss of lung ventilation was mainly concentrated in the lateral inferior and dorsal inferior portions of the lung ultrasound region, as in previous studies, suggesting that clinicians can prioritize scanning these two areas to quickly assess lung ventilation loss in patients.

In our study, B-line scores were poor predictors of PPCs in all patients, which is inconsistent with the findings of previous studies [25,26]. The fact that B-lines respond to volume overload to some extent in patients has been widely confirmed in previous studies [27]. Therefore, if a patient develops fluid overload (mainly determined by NT-proBNP levels), ICU physicians can treat it promptly (e.g., with diuretics) and prevent further loss of lung ventilation in a timely manner; on the other hand, extracorporeal circulation causes ischemia/reperfusion injury to the lungs and contributes to systemic inflammatory response syndrome (SIRS), which leads to an increase in lung permeability and interstitial oedema [28]. Nevertheless, the routine postoperative use of lung protective drugs (ulinastatin and sivelestat sodium) may be effective [29–31]. Thus, the B-line is more common after cardiac surgery,is reversible in the short-term by drugs, and is not a good predictor of the development of PPCs. In contrast, the complete loss of alveolar ventilation (lung consolidation) is challenging to manage quickly and develops into clinically significant PPCs. In addition, thoracentesis is an invasive procedure, and the pleural effusion volume must be large enough to prevent damage to the pleura during puncture, which results in constant compression of the dorsal lung region by the fluid in patients who do not meet the criteria for thoracentesis.

According to the results of the sensitivity analysis, we found that NLC$\geq$3 was still an independent risk factor for postoperative PPCs after excluding patients who developed postoperative pulmonary atelectasis and underwent thoracentesis drainage, while DPE$>$0.95 was not ($P = 0.055$), possibly because the exclusion of 24 patients decreased the rate of the outcome event; therefore, we believe that this may be due to an insufficient sample size.

Finally, our study also showed that preoperative frailty and postoperative acute kidney injury were independent risk factors for PPCs, similar to previous studies [32,33]. This may be because poorer physiological reserve causes a decrease in respiratory muscle function, resulting in difficulty in coughing up sputum, which obstructs the airways and causes pulmonary atelectasis [32]. In addition, acute kidney injury (AKI) can cause lung injury, probably because AKI exacerbates volume overload and the accumulation of harmful substances in the body, leading to cardiogenic pulmonary oedema and an inflammatory response in the alveolar capillaries [33].

There are several limitations to this study. First, this was a single-centre, small-sample observational study, and multicentre, large-sample studies are needed in the future for external validation. Second, lung ultrasound is commonly performed in the ICU. ICU physicians provide timely interventions for high-risk patients, and these interventions may have an impact on the incidence of PPCs. Third, we did not perform preoperative lung ultrasound, but cardiac surgery patients underwent preoperative chest CT. Fourth, pulmonary complications after cardiac surgery are closely related to cardiac and diaphragmatic function, both of which were not assessed in this study [34,35]. Finally, we did not scan the paravertebral region to avoid turning the patient's body. However, it would have reduced the identification of the number of lung consolidations and the depth of pleural effusion, thus underestimating the severity of the loss of pulmonary ventilation. Nevertheless, the vast majority of cardiac patients experience postoperative gravity-dependent areas of atelectasis, especially in the paravertebral region, so scanning this region may not have the ability to differentiate the loss of pulmonary ventilation.

In conclusion, our single-centre cohort study suggested that DPE$>$0.95 and NLC$\geq$3 in ICU-LUS may be associated with PPCs after cardiac surgery. In addition, DPE$>$0.95 & NLC$\geq$3 were associated with prolonged postoperative mechanical ventilation, prolonged ICU stay, prolonged hospitalization, and short-term disability or death after surgery. Future studies should focus on measures to reduce the number of lung consolidations and the loss of compressed lung ventilation caused by increased pleural effusion.

Ethics statement

The Medical Ethics Committee of the Affiliated Hospital of Xuzhou Medical University approved this prospective observational study on November 04, 2022 (XYFY2022-KL367-01). Before patient recruitment, this study was registered with the Chinese Clinical Trials Registry (ChiCTR2200066690, registration date: December 14, 2022). Written informed consent was obtained from all patients prior to enrollment.

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Data availability statement

Data associated with this study has not been deposited into a publicly available repository. And data of this study will be made available on request.

CRediT authorship contribution statement

Guanglei Fan: Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Software, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Fengran Zhang:** Formal analysis, Data curation, Conceptualization. **Tianchi Shan:** Formal analysis, Data curation, Conceptualization. **Yaning Jiang:** Supervision, Software, Formal analysis, Data curation, Conceptualization. **Mingzhu Zheng:** Methodology, Investigation, Data curation. **Baohe Zang:** Writing – review & editing, Validation, Supervision, Software, Resources, Investigation, Formal analysis, Conceptualization. **Wenjing Zhao:** Writing – review & editing, Writing – original draft, Supervision, Resources, Methodology, Funding acquisition, Formal analysis, Data curation, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

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