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Original Research Article

Clinical and laboratory features of patients with postoperative pneumonia after cardiac surgery

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ABSTRACT

Background: Following cardiothoracic surgery, postoperative pneumonia (POP) represents a major clinical sequela. This study compared clinical and diagnostic features among the patients who developed POP and those with an uncomplicated recovery.
Methods: In a retrospective single-centre analysis, 95 patients who had open-heart surgery were divided into group 1 (POP, n=53) and group 2 (no major complications, n=42). We analysed demographics, comorbidities, laboratory results, and echocardiographic data.
Results: While baseline characteristics were similar, group 1 had a significantly higher prevalence of anaemia (98% versus 70%, p<0.001) and postoperative pneumothorax (17% versus 2%, p=0.020). Laboratory findings showed markedly elevated inflammatory biomarkers, including C-reactive protein and procalcitonin (p<0.001), in group 1. Echocardiography revealed a lower left ventricular ejection fraction (57.3% versus 62.8%, p=0.037) and more frequent pericardial effusion (p=0.026) in these patients.
Conclusions: POP is associated with a distinct profile featuring significant anaemia, elevated specific infection biomarkers, slightly impaired left ventricular function, and increased pulmonary complications. The study thus underscores potential focuses for improving both patient assessment before surgery and diagnosis after the procedure.

Keywords: Procalcitonin, Cardiothoracic surgery, Echocardiography, Postoperative pneumonia, Anaemia, Antibiotic therapy

INTRODUCTION

Cardiac surgery represents one of the most formidable achievements of modern medicine, offering definitive treatment for complex valvular heart disease, advanced coronary artery disease, and congenital cardiac anomalies. Over the past five decades, refinements in surgical technique, myocardial protection, anesthetic management, and perioperative care have progressively reduced intraoperative mortality, transforming procedures like coronary artery bypass grafting (CABG) from high-risk ventures into routine, albeit complex, interventions.[1] This success, however, has shifted the clinical and research focus toward the substantial burden of postoperative morbidity, which continues to challenge recovery pathways, prolong hospitalization, escalate healthcare costs, and impair long-term quality of life. Among these complications, infectious sequelae occupy a prominent position, with postoperative pneumonia (POP) consistently emerging as the most prevalent and consequential major infection following cardiac procedures, significantly undermining the benefits of technically successful operations.[2]

The clinical and economic ramifications of POP in this population are profound and well- documented. Epidemiological data reveal incidence rates ranging from

2% to 22%, with variability attributable to differences in diagnostic criteria, surgical populations, and institutional protocols.[3] Beyond its frequency, POP exerts a devastating impact on patient trajectories: it is independently associated with a three-to-fivefold increase in mortality, with attributable mortality rates estimated between 20% and 30% in various series.[4]

Survivors endure significantly prolonged mechanical ventilation (often extended by 5-7 days), longer intensive care unit (ICU) stays (by 4-6 days), and increased overall hospitalization (by 8-15 days) compared to patients without this complication.[5] The financial repercussions are staggering, with POP elevating direct hospitalization costs by 40-60%, translating to tens of thousands of dollars in added expenditure per case and imposing a heavy burden on healthcare systems.[6] Furthermore, long-term sequelae extend beyond the initial hospitalization, with survivors facing higher risks of functional decline, recurrent hospital admission, and diminished health-related quality of life.[6]

The development of POP after cardiac surgery is a multifactorial process, arising from a complex interplay of patient susceptibility, the profound physiological insult of the operation, and postoperative management factors. Recognized patient-related risk factors include advanced age (>70 years), chronic lung disease, diabetes, heart failure, and poor nutritional status.[7] The surgical procedure itself introduces unavoidable insults. Median sternotomy and pleural entry disrupt chest wall mechanics; cardiopulmonary bypass (CPB) triggers a systemic inflammatory response syndrome (SIRS) involving complement activation, cytokine release, and neutrophil-mediated lung injury; and phrenic nerve dysfunction or hypothermia can impair diaphragmatic function.[8,9] Postoperatively, factors like prolonged intubation with micro aspiration, sedation, opioid-related respiratory depression, and immobilization leading to atelectasis create an environment where the lower airways become vulnerable to colonization by nosocomial pathogens, most commonly *Staphylococcus aureus*, *Pseudomonas aeruginosa*, and Enterobacteriaceae.[10]

Despite the implementation of comprehensive preventive strategies—including preoperative optimization, standardized antibiotic prophylaxis, and ventilator care bundles—POP remains a persistent and formidable challenge.[11] This resilience underscores significant gaps in both the understanding of its nuanced pathophysiology and the capacity for accurate, timely diagnosis. Current diagnostic criteria, largely adapted from general critical care guidelines, rely on clinical signs (fever, purulent sputum), new radiographic infiltrates, and laboratory markers like leucocytosis.[12] These indicators suffer from poor specificity in the post-cardiotomy patient, where universal SIRS from surgical trauma causes fever and leucocytosis, and conditions like atelectasis or pleural effusion frequently cloud chest X-ray interpretation. This diagnostic uncertainty creates a clinical dilemma: excessive antibiotic use drives resistance and side effects, while delayed treatment increases mortality risk.[13] There is, therefore, an urgent need for more discriminative tools to distinguish infectious pneumonia from sterile post-surgical inflammation.

In this diagnostic quest, biomarkers have garnered significant interest. While C-reactive protein (CRP) is a sensitive marker of inflammation, its elevation is nonspecific. Procalcitonin (PCT) has shown superior specificity for bacterial infections, as its release is strongly stimulated by bacterial endotoxins and specific cytokines (IL-1β, TNF-α).[14] Evidence supports its role in diagnosing lower respiratory tract infections and guiding antibiotic therapy in critical care.[15] However, its precise investigative utility and optimal cutoff values within the intense inflammatory context distinctive to post-cardiac surgery patients—where SIRS is universal—remain inadequately defined, representing a key knowledge gap.[16] The likelihood of other emerging biomarkers also warrants research.[17]

Beyond diagnostics, the comprehensive clinical profile, or "phenotype," of the cardiac surgery patient who develops POP is incompletely detailed. Specific areas require focused inquiry. First, the role of pervasive perioperative anaemia—common due to chronic disease, CPB haemodilution, and surgical blood loss—needs clarification. Anaemia may impair oxygen delivery and immune function, but its specific link to POP risk is inconsistently reported.[18] Second, the bidirectional relationship relating cardiac function with POP is clinically significant but understudied; pneumonia can strain the heart, while pre-existing ventricular dysfunction may reduce physiological reserve.[19] Few studies have systematically compared detailed echocardiographic indicators between groups with and without POP. Third, the relevance of common postoperative findings like pericardial effusions to POP risk remains ambiguous.[20]

Much of the existing literature has focused on either preoperative risk prediction or analysis of broad outcomes. A holistic, integrative analysis that simultaneously examines detailed clinical profiles, comprehensive laboratory parameters (including advanced biomarkers), and echocardiographic features within a contemporary cohort is lacking. Such an integrated approach is necessary to move beyond isolated risk factors toward a cohesive understanding of the susceptible patient phenotype.

To address these evidence gaps, we conducted this retrospective cohort study. Our primary objective was to perform a detailed comparative analysis of patients undergoing open-heart surgery who developed POP versus those with an uncomplicated recovery. We aimed to evaluate and contrast their: clinical and comorbidity profiles; laboratory parameters, with a dedicated focus on the infection biomarkers PCT and CRP; and echocardiographic indicators of cardiac structure and function.

METHODS

This retrospective analysis examined 95 patients undergoing open heart surgery at the Grodno Regional Clinical Cardiological Centre from January to December 2024.

Inclusion criteria

Inclusion criteria includes patients who underwent open heart surgery during the study period, had complete medical records available in the hospital's digital system, and provided written informed consent. The cohort was divided into two groups: group 1 consisted of 53 patients (55.7%) who developed postoperative pneumonia (POP), and group 2 comprised 42 patients (44.3%) who did not experience this complication.

Pneumonia was diagnosed based on established American guidelines. A clinical diagnosis required the presence of new or progressive pulmonary infiltrates on chest imaging, alongside two or more supporting criteria: fever exceeding 38°C without another cause, leucocytosis (above 12×10^9/l) or leukopenia (below 4×10^9/l), and the presence of purulent respiratory secretions.

Exclusion criteria

Exclusion criteria from the study were: infarction pneumonia, acquired pneumonia within 1 month prior to surgical operation, oncological diseases and severe concomitant extracardiac pathology.

Anamnestic, laboratory, and clinical information was collected from the hospital's digital health records management system (4D Client).

Statistical analysis

Statistical analysis was performed using the STATISTICA 12.0 computer software. Data distribution was assessed for normality via histogram analysis. Sets of quantitative indicators whose distribution deviated from normal were described using median (Me) values and lower and upper quartiles (Q1; Q3). Nominal data were described using absolute values and percentages. Given that most quantitative variables were not normally distributed, non-parametric methods were applied.

Differences between two independent groups were evaluated by Mann-Whitney test, alongside a p value of less than 0.05 considered statistically significant.

The research was conducted following good clinical practice and the ethical guidelines of the Declaration of Helsinki. Every individual provided written informed consent before participating in the study.

RESULTS

Of the operations, 63 (66.3%) were performed for isolated CABG, 9 (9.5%) for isolated valve repair or replacement, 13 (13.7%) for CABG and mixed valve surgery, and 10 (10.5%) for other types.

Among 52 patients with POP, 11 (20.8%) developed right sided and 21 (39.6%) – left sided pneumonia, while 20 (38.5%) patients had bilateral pneumonia.

Clinical characteristics of patients presented in Table 1.

Table 1: Clinical characteristics of patient.

Parameters	Group 1 (n=52), N (%)	Group 2 (n=43) , N (%)	P value
Male gender	33 (63)	32 (74)	0.268
Age, years, (Me [Q1; Q3])	64.8 [61.7; 70.5]	63.9 [58.5; 70.3]	0.456
Obesity	17 (32)	15 (35)	0.822
Overweight	15 (29)	6 (14)	0.078
Hypertension	50 (96)	39 (91)	0.391
Stage 1	5 (10)	10 (23)	0.045
Stage 2	40 (77)	28 (65)	0.201
Stage 3	5 (10)	0	0.031
Myocardial infarction history	25 (48)	23 (53)	0.602
Diabetes mellitus	8 (15)	12 (28)	0.135
Atrial fibrillation	27 (52)	20 (47)	0.602
Chronic kidney disease stage 3-5	3 (6)	13 (30)	0.001
COPD	5 (10)	1 (2)	0.218
Bronchial asthma	2 (4)	0	0.501
Chronic sinusitis	4 (8)	1 (2)	0.378
COVID-19	2 (4)	6 (14)	0.072
Postoperative complications			
Pneumothorax	9 (17)	1 (2)	0.020
Postoperative anemia	51 (98)	30 (70)	<0.001

Continued.

Parameters	Group 1 (n=52), N (%)	Group 2 (n=43) , N (%)	P value
Postoperative bleeding	16 (31)	7 (16.2)	0.098
Infective endocarditis	1 (2)	-	0.913

Me – median value; Q1 – lower quartile; Q3 – upper quartile

Analysis of baseline characteristics revealed that the groups were comparable in age and gender, with predominance of male patients in both groups (63% versus 74%, p>0.05). There were no differences in major cardiac and extracardiac comorbidities including diabetes, obesity, COPD, bronchial asthma and atrial fibrillation (p>0.05). While hypertension was present almost in all the study participants (96% versus 91%, p>0.05), the severity of it differed significantly between the cohorts, with Stage 3 being significantly more common in POP patients (p=0.031). It is interesting to add that patients didn't have differences in history of MI (48% versus 53%, p>0.05), while patients of Group 2 had a greater burden of chronic kidney disease (30% versus 6%, p=0.001). Also, significant differences were observed in specific postoperative complications. Patients who developed POP had a substantially higher prevalence of postoperative anaemia (98% versus 70%, p<0.001) and pneumothorax (17% versus 2%, p=0.020). Also, we observed a tendency for higher rate of postoperative bleeding in POP group (31% versus 16%, p=0.098) which didn't reach margin of statistical significance.

Table 2 presents the antibiotic regimen profile for group 1 (n=53).

The most frequently administered antibiotic classes were fluoroquinolones (20 patients, 37.7%) and cephalosporins (18 patients, 34.0%), followed by carbapenems (11 patients, 20.8%).

The total percentage exceeds 100% as patients could receive more than one antibiotic agent during the study period.

Laboratory parameters of patients are presented in Tables 3 and 4.

Table 2: Postoperative antibiotic therapy.

Parameters	Group 1 (n=53), N (%)
Penicillin	4
Cephalosporins	18
Fluoroquinolones	20
Carbapenems	11
Vancomycin	4
Gentamycin	1
Another antibiotic	19

The biochemical profile revealed that while most metabolic and organ function parameters were comparable among groups, there was a stark and highly significant difference in systemic inflammation and infection biomarkers, as evidenced by significantly elevated levels of both CRP (p=0.001) and PCT (p<0.001). The extremely low p value for PCT underscores a powerful statistical distinction, strongly suggesting a more severe degree of systemic inflammatory response in patients with POP.

The values of echocardiographic parameters recorded in patients of both groups presented in Table 5.

Table 3: Complete blood count parameters of patients.

Parameters	Group 1 (Me [Q1; Q3])	Group 2 (Me [Q1; Q3])	P value
RBC, 10^{12}/l	3.5 [3.2; 3.7]	3.8 [3.4; 4.18]	0.008
Hemoglobin, g/l	105.6 [95.8;114.5]	118.4 [102.5; 129]	0.001
WBC, 10^9/l	9.9 [6.7; 11.5]	10.6 [7.8; 12.9]	>0.05
ESR, mm/hour	24.0 [11.5; 34.2]	17.1 [8; 20]	0.017
Platelets, 10^3/l	341 [238; 386]	269 [198; 331]	0.027
Segmented neutrophils (%)	75 [59.2; 73]	68.8 [60.4; 77.5]	>0.05
Band neutrophils (%)	6.07 [2 7]	3.55 [1; 6]	0.048
Lymphocytes (%)	22 [11.3; 24.9]	19.1 [9; 27]	>0.05
Eosinophils (%)	2.55 [0.7; 3.9]	2.0 [1; 2.8]	>0.05
Basophils (%)	0.4 [0.1; 0.5]	0.26 [0.1; 0.3]	>0.05
Monocytes (%)	7.4 [5.8;9.2]	7.1 [5.1; 9]	>0.05

Me – median value; Q1 – lower quartile; Q3 – upper quartile; RBC – red blood cells; WBC – white blood cells; ESR – erythrocyte sedimentation rate

Table 4: Biochemical blood analysis parameters of patients.

Parameters	Group 1 (Me [Q1; Q3])	Group 2 (Me [Q1; Q3])	P value
Total protein, mg/l	60.8 [57.8; 70]	63 [58; 67.8]	>0.05
Urea, mmol/l	10.1 [5.2;12.0]	10.4 [5.9; 10.1]	>0.05

Continued.

Parameters	Group 1 (Me [Q1; Q3])	Group 2 (Me [Q1; Q3])	P value
Creatinine, µmol/l	111.7 [88.5;118.5]	111.3 [85;127]	>0.05
eGFR, ml/min/1.73m^2	57.9 [45.6;71.4]	60.5 [44;77.4]	>0.05
AST, IU/l	40 [16.6; 33]	30.8 [16.8;35]	>0.05
ALT, IU/l	38.5[17.5; 40]	28.0 [19.1; 31.7]	>0.05
Glucose, mmol/l	6.0 [5.2; 6.2]	5.9 [5.1; 6.2]	>0.05
Sodium, mEq/l	137.9 [135;141.3]	138.1 [136;140]	>0.05
Potassium, mEq/l	4.7 [4.3; 5.0]	4.6 [4.3;4.8]	>0.05
C-reactive protein, mg/l	69.5 [17.9; 71.1]	47.8 [21.7;71.9]	0.001
Procalcitonin, ng/ml	0.63 [0.05; 0.35]	0.17 [0.14; 0.22]	<0.001

Me – median value; Q1 – lower quartile; Q3 – upper quartile; eGFR – estimated glomerular filtration rate; AST – aspartate aminotransferase; ALT – alanine aminotransferase

Table 5: Echocardiographic parameters of patients.

Parameter	Group 1 (Me [Q1; Q3])	Group 2 (Me [Q1; Q3])	P value
LA diameter (2 chamber), mm	38.4 [36; 39.75]	37.9 [34.7; 38.3]	>0.05
LA diameter (medial to lateral), mm	38.7 [36; 42.5]	38.5 [33.2; 42]	>0.05
LA diameter (front to back), mm	53.2 [50; 55.5]	54.3 [50.2; 55.7]	>0.05
RA diameter (medial to lateral), mm	36.8 [32.5; 40]	35.1 [31.2; 37]	>0.05
RA diameter (front to back), mm	50.5 [46.5; 54]	49.4 [46; 51.5]	>0.05
LV ESD, mm	36.3 [32.5; 39]	33.4 [30; 35.7]	>0.05
LV EDD, mm	52 [50; 55.5]	51.3 [48; 53]	>0.05
LV ESV, ml	59.4 [51.5; 67.5]	48.5 [35.5; 52.5]	0.04
LV EDV, ml	137.8 [121; 155]	128.5 [116; 140]	>0.05
LVEF, %	57.3[54.7; 60]	62.8 [59; 68]	0.037
Right ventricle diameter, mm	23.6 [19; 26]	25.0 [21; 26.5]	>0.05
Pericardial effusion, n (%)	11 (20.7)	2 (4.7)	0.026
Pleural effusion, n (%)	28 (53.8)	21 (48.8)	>0.05

Me – median value; Q1 – lower quartile; Q3 – upper quartile; LA – left atrium; RA – right atrium; LV – left ventricle; ESD – end-systolic diameter; EDD – end-diastolic diameter; ESV – end-systolic volume; EDV – end-diastolic volume; LVEF – left ventricular ejection fraction

While most baseline cardiac dimensions, including left and right atrial sizes as well as left ventricular end-diastolic dimensions, were comparable between the groups (p>0.05), key parameters of systolic function and pathology were considerably lower in patients with POP. This group exhibited a notable impairment in systolic function of left ventricle, characterized by a statistically significant reduction in LVEF (57.3% versus 62.8%, p=0.037). Furthermore, group 1 demonstrated a significantly higher incidence of pericardial effusion (20.7% versus 4.7%, p=0.026), indicating a greater prevalence of this comorbidity. The prevalence of pleural effusion and the remaining structural measurements did not differ significantly. These findings point towards a more compromised cardiac systolic function and a higher burden of pericardial involvement in patients with POP.

DISCUSSION

This retrospective study delineates a distinct clinical phenotype associated with POP after cardiac surgery, characterized by significant hematological, inflammatory, and cardiopulmonary disturbances. The cohorts were well-matched for demographics and major comorbidities, strengthening the attribution of observed differences to POP's pathophysiology.

The most salient clinical finding was the near-universal anemia in the POP group (98% versus 70%), with significantly lower postoperative hemoglobin. This corroborates preoperative anemia as a potent risk factor for postoperative infection.[18,21] Impaired oxygen delivery compromises immune cell function and tissue repair, underscoring the importance of preoperative anemia management as a modifiable target within optimization bundles.[22]

The higher incidence of postoperative pneumothorax (17% versus 2%) represents another critical factor. As a complication from pleural entry or central line placement, pneumothorax promotes atelectasis and creates a nidus infection. This finding reinforces the need for meticulous surgical technique, ultrasound-guided vascular access, and lung-protective ventilation to mitigate this risk.[23]

Antibiotic use in the POP group reflected the severity of healthcare-associated infections, dominated by broad-spectrum agents like fluoroquinolones and carbapenems, aligning with guidelines for empiric coverage of multidrug-resistant organisms.[12,24] This pattern, while clinically appropriate, highlights the necessity for rapid

microbiological diagnostics to enable early de-escalation and combat antimicrobial resistance.[25]

The laboratory profile provided key diagnostic insights. While elevated ESR indicated inflammation, the biomarker findings were most revealing. The marked elevation of procalcitonin (PCT) in the POP group is particularly significant. PCT demonstrates higher specificity for bacterial infection than CRP, as its release is stimulated by bacterial endotoxins.[14, 15] In the invariably inflammatory post-cardiac surgery setting, our data support PCT's utility as a discriminatory tool to help confirm bacterial pneumonia and guide antibiotic initiation, potentially reducing unnecessary antimicrobial exposure.[26] This strengthens the argument for incorporating PCT into postoperative stewardship protocols.[27]

Echocardiographic data introduced a crucial cardiopulmonary dimension. The lower left ventricular ejection fraction (LVEF) in the pneumonia group suggests either pre-existing subclinical dysfunction reducing physiological reserve or the development of sepsis-induced cardiomyopathy (SICM).[19,20] SICM, a reversible contractility of depression driven by inflammatory cytokines, requires integrated management where hemodynamic support is as vital as antimicrobial therapy.[28] The higher incidence of pericardial effusion further indicates a pronounced local inflammatory response, related to post-pericardiotomy syndrome.[29]

An unexpected finding was the lower prevalence of advanced chronic kidney disease (CKD) in the pneumonia group. This contrasts with typical associations between CKD and infection risk.[30] Potential explanations include more intensive perioperative renal protection strategies in CKD patients, indirectly reducing pulmonary complications, or selection bias in surgical planning and antibiotic prophylaxis. This counterintuitive result merits further investigation, suggesting POP's risk profile may differ from other postoperative sequelae.

Limitations and strengths

This study has limitations inherent to its retrospective, single-center design, which may introduce unmeasured confounding and limit causal inference. The sample size, though adequate for primary comparisons, may be underpowered for subgroup analyses. The lack of detailed microbiological data restricts pathophysiological context, and generalizability may be limited to similar clinical settings.

Notable strengths include the integrated, simultaneous analysis of clinical, laboratory, and imaging domains, providing a holistic view of the POP phenotype. The use of guideline-based diagnostic criteria enhances validity, and the application of specific biomarkers like PCT in this unique surgical context adds valuable data to an evolving field. The well-matched control group strengthens comparative analysis.

Future research directions

To verify these outcomes, prospective findings involving multiple research sites are necessary. Upcoming research ought to focus on: serial measurement of PCT and novel biomarkers (e.g., pre-sepsin) to establish kinetic profiles for improved diagnosis; detailed microbiological analyses correlating pathogens with clinical and biomarker phenotypes; interventional trials assessing whether preoperative anemia correction or strict PCT-guided antibiotic algorithms improve outcomes; and mechanistic investigations into the links between subclinical cardiac dysfunction and pulmonary infection susceptibility.

CONCLUSION

This analysis demonstrates that POP following cardiac surgery presents as a distinct clinical syndrome, characterized by significant perioperative anaemia, a high incidence of pneumothorax, markedly elevated procalcitonin levels, and concurrent evidence of impaired left ventricular function with pericardial effusion.

These findings highlight key clinical priorities: optimizing anaemia management, vigilant monitoring of patients with pneumothorax, and incorporating procalcitonin testing to improve diagnostic accuracy and antibiotic stewardship. Ultimately, an integrated cardiopulmonary treatment strategy is essential, recognizing that supporting cardiac function is integral to managing pulmonary infection.

Understanding this phenotype enhances risk prediction, enables earlier diagnosis, and informs more effective treatment. Future prospective studies should validate these findings and develop targeted interventions to reduce the substantial burden of this complication.

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