https://doi.org/10.63181/ujcvs.2025.33(3).122-129
УДК 616.12-089-036.8:616.98:578.834

Dmytro M. Kosovan, PhD Student, Cardiac Surgeon, https://orcid.org/0009-0009-4623-9846
Amosov National Institute of Cardiovascular Surgery, Kyiv, Ukraine

Surgical Outcomes in the Era of COVID-19: A Stratified Analysis Based on Infection Chronology

Abstract

Aim. To investigate the impact of COVID-19 infection timing on postoperative outcomes in patients undergoing surgical treatment for acquired valvular heart disease.

Materials and Methods. This single-center retrospective study included 96 patients with confirmed COVID-19 who underwent valvular heart surgery. Patients were stratified into two groups based on the timing of SARS-CoV-2 infection: those infected prior to hospital admission (preoperative COVID-19; n=69) and those infected during hospitalization after surgery (postoperative/hospital-acquired COVID-19; n=27). Clinical, laboratory, and perioperative data were analyzed, including length of hospitalization and ICU stay, duration of mechanical ventilation, use of inotropes/vasopressors, and mortality. Statistical analyses included t-tests, Mann–Whitney U tests, Pearson's correlation, and chi-square tests.

Results. Postoperative COVID-19 infection was associated with significantly worse outcomes, including longer hospital and ICU stays, prolonged mechanical ventilation (169.7 ± 140.8 h vs. 18.1 ± 35.3 h; $p<0.001$), increased need for inotropic and vasopressor support (dobutamine use: r=0.84; norepinephrine use: r=0.76), and higher mortality. Laboratory findings revealed more profound thrombocytopenia, hyperglycemia, and renal dysfunction in the postoperative group. In contrast, patients with preoperative COVID-19 demonstrated more favorable outcomes, provided that a sufficient interval (≥6–7 weeks) had elapsed between infection and surgery. Strong correlations were observed between mortality and indicators of multiorgan dysfunction, particularly respiratory failure, cardiac decompensation, and pneumonia.

Conclusions. The timing of COVID-19 infection is a critical determinant of postoperative prognosis in patients undergoing valvular surgery. Hospital-acquired infection is an independent predictor of adverse outcomes. Elective surgery should be deferred for at least 6–7 weeks after COVID-19 recovery, with individualized risk assessment. Preventing early nosocomial infection in the postoperative period is essential to reduce mortality in cardiac surgery patients.

Keywords: *COVID-19, valvular heart disease, cardiac surgery, infection timing, postoperative outcomes, mortality, inotropes, respiratory failure.*

Introduction. The COVID-19 pandemic has profoundly transformed patient management strategies in surgical specialties, particularly in cardiac surgery, where perioperative risks of complications and mortality have increased against the backdrop of systemic inflammation, endothelial dysfunction, and immunosuppression induced by SARS-CoV-2 [1,9,10]. The presence of active or recently resolved coronavirus infection affects all stages of the therapeutic process – from surgical planning to postoperative monitoring. Of particular concern are cases in which infection coincides with the timing of surgery or develops in the early postoperative period, both scenarios being associated with a high incidence of severe complications and mortality [1,6,7].

Early meta-analyses, notably the COVIDSurg Collaborative, reported that 30-day postoperative mortality among patients with perioperative COVID-19 reached 23 % [1,3]. These findings substantiated recommendations to postpone elective surgeries for at least 6-7 weeks after infection, even in asymptomatic cases [3,4]. However, excessive delays in patients with severe valvular heart disease may lead to decompensation, sudden death, or irreversible damage to target organs [2]. This has created a clinical dilemma – how to balance safe surgical timing after COVID-19 with the necessity for timely operative intervention.

Hospital-acquired (nosocomial) COVID-19 in patients after cardiac surgery poses an additional threat. Multicenter studies have shown that SARS-CoV-2 infection within the first 5-7 days postoperatively is associated with a mortality rate exceeding 15 % and a requirement for mechanical ventilation in over 20 % of cases, whereas later infections tend to follow a less aggressive course [7]. This heightened vulnerability is explained by the superimposition of the virus-induced systemic inflammatory response on an already activated postoperative SIRS,

coupled with postoperative immunosuppression, which increases the risk of pneumonia, sepsis, and multiple organ failure [1,10].

In patients who had COVID-19 prior to hospital admission, clinical risk varies considerably depending on the interval before surgery, the severity of infection, and the degree of recovery of target organs. Current guidelines emphasize an individualized approach to determining surgical timing, taking into account residual symptoms, oxygen saturation, CT findings, coagulation markers, respiratory function, and immune status [4]. Real-world cohort data indicate that when a sufficient interval has elapsed after COVID-19 –particularly in cases of mild or asymptomatic disease, surgical outcomes may not differ from those in COVID-negative patients [2,5].

In this context, comparing postoperative outcomes between patients who had COVID-19 before hospitalization and those who acquired the infection postoperatively is of particular scientific and practical relevance. Evaluating the dynamics of laboratory and clinical parameters, duration of intensive care, complication rates, and mortality in these groups provides insights into the pathophysiological mechanisms of SARS-CoV-2's impact on postoperative recovery and facilitates the development of practical algorithms for risk stratification and surgical planning.

Aim. The aim of this clinical and analytical study was to investigate the impact of COVID-19 infection timing on the outcomes of surgical treatment for acquired valvular heart disease. In particular, the analysis focused on postoperative outcomes in patients who underwent valvular surgery, stratified according to whether SARS-CoV-2 infection occurred prior to hospital admission (preoperative COVID-19) or during the postoperative period (hospital-acquired COVID-19).

The study objectives include:

- Comparing clinical and laboratory parameters, postoperative course, and mortality between the two stratified groups.
- Identifying the most significant prognostic markers of an unfavorable clinical course.
- Determining the potential relationship between the timing of COVID-19 infection and the length of hospitalization, duration of ventilatory support, need for vasopressors, severity of organ dysfunction, and mortality.
- Formulating clinical recommendations on the optimal timing for performing valvular interventions in the context of prior or concurrent COVID-19 infection.

Materials and Methods. This single-center retrospective study included 96 patients with a confirmed diagnosis of COVID-19 who underwent valvular surgery for acquired heart valve disease. Participants were stratified into two groups according to the timing of SARS-CoV-2 infection:

- **Group 1 (n=69):** Patients who contracted COVID-19 prior to hospital admission, with an interval of 1 to 24 months before surgery.
- **Group 2 (n=27):** Patients who developed COVID-19 postoperatively during inpatient care, within a time frame of 3 to 21 days after surgery.

Inclusion criteria:

- Confirmed diagnosis of COVID-19 based on PCR, ELISA, or a combination of both methods.
- Surgical treatment for valvular pathology.
- Complete clinical, laboratory, and anesthesiological records.
- Documented final treatment outcome.

Clinical parameters. The analysis included both descriptive characteristics (age, sex, anthropometric data, length of hospital stay, duration of ICU stay, mechanical ventilation time, and dependence on dobutamine/norepinephrine) and dynamic biochemical, hematological, and blood gas parameters (preoperative and on postoperative days 1 and 2). Additional variables analyzed were infusion therapy volume, blood product transfusions, cardiopulmonary bypass duration, and aortic cross-clamp time.

Statistical analysis

1. Intergroup comparisons were performed using:
   - Independent samples *t*-test for normally distributed data.
   - Mann–Whitney U test for non-normally distributed data.
2. Correlation analysis was conducted using Pearson's method to assess associations between key clinical parameters and mortality.
3. Associations between categorical variables and treatment outcomes (survival/mortality) were evaluated using the Chi-square (χ^2) test.
4. A p-value <0.05 was considered statistically significant.
5. Data processing and visualization were performed using MS Excel, JASP, and SPSS software.

Results. The study included 96 patients with confirmed COVID-19 who underwent valvular surgery for acquired heart valve disease. The mean age of the cohort was 56.8 ± 14.2 years (median: 60 years, interquartile range [IQR]: 48.5–67.5), indicating a predominance of older patients without extreme age outliers. Anthropometric characteristics were as follows: mean body weight, 80.7 ± 15.3 kg; mean height, 170.8 ± 9.2 cm. Based on these data, the mean body surface area, calculated using the DuBois formula, was 1.92 ± 0.19 m^2. Patients were stratified into two groups according to the timing of SARS-CoV-2 infection: preoperative COVID-19 (n=69) and postoperative COVID-19 (n=27). In the preoperative group, the mean age was 54.4 ± 14.8 years (median: 57 years), with a predominance of males (71.0 %). In contrast, patients in the postoperative COVID-19 group were significantly older, 62.3 ± 11.8 years (median: 64 years; p<0.05), and the proportion of females was higher (70.4 %), demonstrating the opposite gender distribution compared with the preoperative group.

Comparison of clinical parameters revealed substantial differences between the groups. The mean length

of hospital stay was significantly longer in the postoperative COVID-19 group, 28.07 ± 18.4 days, versus 17.90 ± 10.8 days in the preoperative group (p<0.001). A similar trend was observed for ICU length of stay: 17.11 ± 13.8 days versus 5.14 ± 4.9 days, respectively (p<0.001). Mechanical ventilation duration was markedly prolonged in the postoperative group (169.7 ± 140.8 h vs. 18.1 ± 35.3 h; p<0.001), indicating severe respiratory compromise in this cohort.

A comparison of in-hospital mortality between the groups revealed a highly significant difference. Among patients who had recovered from COVID-19 prior to hospital admission (n=69), mortality was 4.4 % (3 cases), whereas in the group with hospital-acquired (postoperative) infection (n=27), this rate reached 59.3 % (16 cases). Statistical analysis confirmed the robustness of these differences (χ^2=33.48; p<0.000001; Fisher's exact p=1.25×10^{-8}), and the calculated odds ratio (OR=0.031) indicated an approximately 32-fold increase in the risk of death in the latter group. These findings strongly suggest that the development of COVID-19 in the postoperative period is a critical predictor of poor outcomes.

In our study, among patients who had recovered from COVID-19 prior to hospitalization (n=69), the distribution by severity of infection was as follows: mild course in 41 patients (59.4 %), with a postoperative mortality rate of 4.9 % (2 cases); moderate course in 25 patients (36.2 %), with no deaths recorded; and severe course in 3 patients (4.3 %), with a mortality rate of 33.3 % (1 case). Among the three deceased patients in this group, two had active infective endocarditis and died due to sepsis with multiorgan failure, while one patient died from acute myocardial infarction in the postoperative period.

In the group of patients who acquired COVID-19 postoperatively (n=27), a mild course was observed in 4 patients (14.8 %), all of whom survived, and a moderate course in 7 patients (25.9 %), also without fatal outcomes. Importantly, all cases of mild and moderate COVID-19 occurred within the first postoperative week. Severe disease was diagnosed in 16 patients (59.3 %), with a mortality rate of 100 %. Among these, 12 patients became infected after the 10th postoperative day, while 4 developed the infection between postoperative days 1 and 10.

Additional analysis showed that COVID-19 severity was significantly associated with mortality both in the preoperative infection group (p=0.027) and, even more strongly, in the postoperative infection group (p<0.001).

No statistically significant differences were found between groups regarding cardiopulmonary bypass duration, aortic cross-clamp time, or transfusion volumes. Although cardiopulmonary bypass duration was longer in the postoperative COVID-19 group (201.6 ± 14.6 min) compared to the preoperative group (177.0 ± 7.26 min; p=0.099), this difference did not reach statistical significance. Similarly, mean aortic cross-clamp time was comparable between groups (127.0 ± 8.44 min vs. 122.9 ± 5.22 min; p=0.677). The volumes of transfused red blood cells, plasma, and platelets also showed no significant differences (p>0.05 for all), although the postoperative COVID-19 group tended to receive larger platelet transfusions (277.3 ± 56.6 mL vs. 225.0 ± 29.8 mL). These findings suggest that cardiopulmonary bypass duration, aortic cross-clamp time, and transfusion volumes are unlikely to be the primary determinants of outcome differences between the two subgroups. Among patients with preoperative COVID-19, 11 underwent emergency surgery, including 7 with active infective endocarditis and the remainder with decompensated non-infective acquired valvular disease. In the postoperative COVID-19 group, 6 emergency procedures were performed, of which 2 patients had active infective endocarditis. Statistical analysis revealed no significant differences in the proportion of emergency cases between groups, and this factor did not influence overall treatment outcomes.

Patients with hospital-acquired COVID-19 demonstrated a substantially greater need for inotropic and vasopressor support. The duration of dobutamine infusion was markedly prolonged, 125.19 ± 88.4 hours, compared with 21.74 ± 37.2 hours in patients with preoperative COVID-19 (p<0.001), while norepinephrine administration lasted an average of 73.67 ± 65.2 hours versus 7.19 ± 15.6 hours, respectively (p<0.001). These differences indicate more profound hemodynamic instability and a more severe postoperative course in patients with postoperative infection.

Laboratory analysis revealed a statistically significant decrease in platelet count in the postoperative COVID-19 group both preoperatively (205.5 ± 83.4 ×10^9/L vs. 254.2 ± 98.6 ×10^9/L, p<0.01) and at 24 hours post-surgery (125.9 ± 72.1 ×10^9/L vs. 178.4 ± 80.7 ×10^9/L, p<0.001). This group also exhibited lower hematocrit levels during cardiopulmonary bypass (0.303 ± 0.05 vs. 0.340 ± 0.06, p<0.01) and on postoperative day 1 (0.305 ± 0.07 vs. 0.335 ± 0.06, p<0.001). Acid–base imbalance was noted, with an elevated venous pH (7.44 ± 0.07 vs. 7.42 ± 0.05, p<0.05), suggesting activation of compensatory metabolic processes or the presence of respiratory alkalosis.

Patients who developed postoperative (hospital-acquired) COVID-19 were also found to have significantly higher blood glucose levels both at 24 hours (10.40 ± 3.5 mmol/L vs. 8.75 ± 2.6 mmol/L, p<0.01) and on postoperative day 2 (8.42 ± 3.2 mmol/L vs. 6.96 ± 2.4 mmol/L, p<0.01). Indicators of renal dysfunction in this group included increased urea levels (14.74 ± 6.3 mmol/L vs. 11.47 ± 5.1 mmol/L, p<0.05) and reduced urine output on day 1 (1312 ± 901 mL vs. 2028 ± 1078 mL, p<0.001). Elevated central venous pressure on postoperative day 2 (96 ± 20 mmH_2O vs. 76 ± 18 mmH_2O, p<0.001) further confirmed the presence of more severe hemodynamic compromise.

The volume of transfused red blood cells in the ICU was nearly threefold higher in the postoperative COVID-19 group (1168 ± 545 mL vs. 449 ± 384 mL, p<0.05), indicating more frequent hemorrhagic complications or anemia related to coagulopathy. Collectively, these findings

reflect more profound disturbances in homeostasis during the postoperative period in the setting of COVID-19.

An association analysis between clinical and historical variables and treatment outcomes (survival vs. death) revealed statistically significant links between several factors and adverse outcomes. The strongest association was observed between respiratory failure and mortality ($p<0.001$), followed by the need for dobutamine ($p<0.01$) and norepinephrine ($p<0.01$) support. Acute heart failure was also identified as a predictor of mortality ($p<0.05$). In contrast, arterial hypertension, heart rhythm, sex, and microbiological findings had no statistically significant effect on final outcomes.

Within the study, a correlation analysis was performed between key clinical variables and treatment outcomes (death or survival) in the cohort of patients who developed COVID-19 during hospitalization after undergoing cardiac surgery. The dependent variable was the binary outcome "Survival", coded as 1 for death and 0 for survival. Pearson's correlation coefficients were used to assess the direction and strength of associations between individual parameters and the risk of death (Figure 1).

The strongest positive correlation with mortality was observed for dobutamine administration, with a correlation coefficient of $r=0.84$. This finding reflects the critical degree of hemodynamic destabilization in patients requiring inotropic support. Clinically, this implies that patients with profound impairment of cardiac pump function, regardless of etiology, had the poorest prognosis, underscoring the importance of early recognition of heart failure and timely initiation of medical support.

A slightly lower but still strong positive correlation was found between mortality and norepinephrine use ($r=0.76$), indicating the frequent need for vasopressor support in severe patients with septic and/or cardiogenic shock. The strong intercorrelation between dobutamine and norepinephrine ($r=0.81$) points to a shared pathophysiological basis and concurrent need for vasoactive therapy in the setting of multiple organ failure.

Acute heart failure showed a correlation with mortality of $r=0.53$, reaffirming the prognostic relevance of cardiovascular status. In the context of COVID-19, the myocardium is vulnerable to infectious, hypoxic, and thrombotic injury, as well as the excessive postoperative stress

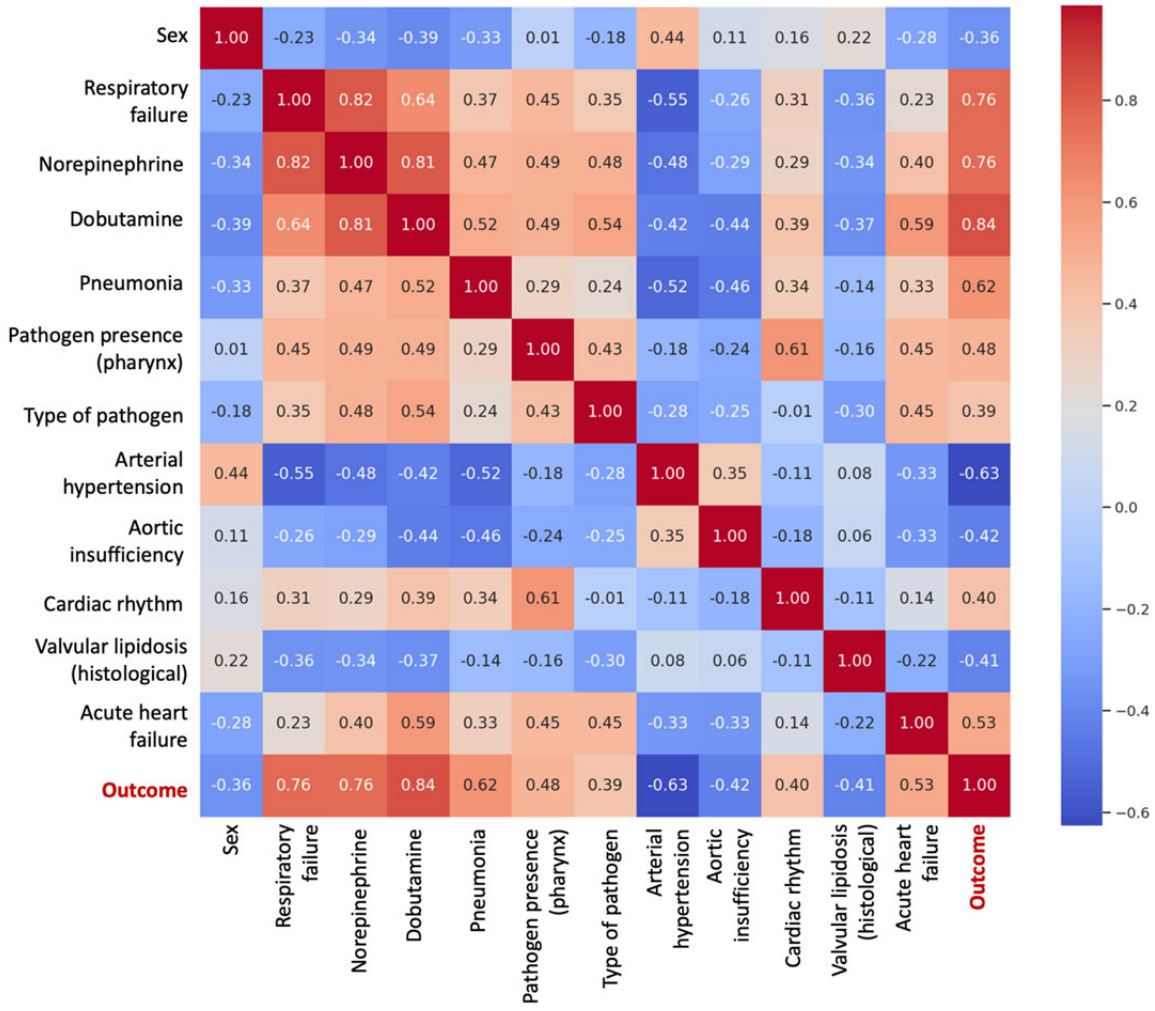

Figure 1. *Correlation matrix of clinical variables and mortality*

response, all of which can cause both direct cardiac dysfunction and the need for vasoactive/inotropic support.

Postoperative respiratory failure, most often manifesting as pneumonia with progression to acute respiratory distress syndrome (ARDS), was also strongly correlated with mortality (r=0.76). The presence of ventilator-dependent respiratory dysfunction was a hallmark of severe COVID-19 in post-surgical patients. This factor is pathophysiologically justified, as respiratory failure is a key component of ARDS development, hypoxemia, hypercapnia, and, in severe cases, multiple organ failure.

A history of pneumonia in either group showed a notable positive correlation with mortality (r=0.62). This suggests that both pre-existing pneumonia and nosocomial lung infections significantly impair prognosis. Lung injury reduces respiratory reserve, promotes systemic hypoxia, and contributes to secondary organ dysfunction. Pneumonia also correlated with dobutamine use (r=0.52), further supporting its systemic impact through hypoxic–ischemic myocardial dysfunction. Other variables had clinically relevant, although less pronounced, associations with mortality. Heart rhythm demonstrated a weak positive correlation (r=0.40), which may reflect the detrimental effect of arrhythmias—particularly atrial fibrillation—on adaptive capacity. Right ventricular hypertrophy showed a negative correlation (r=-0.42), which may indicate a compensatory, rather than decompensatory, adaptation in some patients with pulmonary hypertension. Histological changes in the valvular apparatus, specifically lipoidosis, also correlated negatively (r=-0.41), though this was likely of limited clinical significance in the acute setting.

Of particular note, the presence of arterial hypertension exhibited a strong negative correlation with mortality (r=-0.63), an unexpected finding. A possible explanation is that patients with chronic hypertension were more likely to be on regular medication (including ACE inhibitors or beta-blockers), which may have provided cardio- or vasoprotective effects during acute decompensation. Moreover, these patients may have been under closer outpatient surveillance and received earlier interventions. Nonetheless, this negative correlation should be interpreted with caution due to potential confounding factors.

Microbiological parameters – specifically, the presence of a pathogen in the oropharyngeal swab and its identification – demonstrated moderate positive correlations with mortality (r=0.48 and r=0.39, respectively). This suggests a potential contribution of bacterial colonization to the development of complications; however, these variables did not emerge as leading risk factors within the analyzed cohort.

Patient sex exhibited a weak negative correlation with mortality (r=-0.36), which may indicate a slightly more favorable prognosis in women; nevertheless, the strength of this association is insufficient to draw definitive clinical conclusions without multifactorial confirmation.

Overall, the results of the correlation analysis indicate the presence of several key variables associated with the risk of death in postoperative patients with COVID-19. The strongest associations were observed for parameters reflecting respiratory and hemodynamic decompensation, pneumonia, and acute heart failure. The variable showing an inverse relationship requires further analysis, taking into account potential multifactorial dependencies.

Discussion. The obtained results confirm the substantial impact of SARS-CoV-2 infection timing on the postoperative course in patients who underwent valvular surgery for infective endocarditis.

Specifically, patients who contracted COVID-19 during the postoperative period experienced a significantly higher rate of critical complications, longer ICU stays, markedly prolonged respiratory and hemodynamic support, worse laboratory indices of pulmonary, renal, and coagulation function, and a higher mortality rate 59.3 % vs. 4.4 %. These findings are consistent with reports from multicenter studies demonstrating that early postoperative COVID-19 infection is an extremely unfavorable prognostic factor [6,7].

In our cohort, the mortality rate among patients with postoperative COVID-19 reached 59.3 % (16/27), which was statistically higher compared to 4.4 % (3/69) in the preoperative group (χ^2*=33.48; p<0.001*). These findings are in line with results by Spadaccio et al. (2024), who reported a 30-day mortality of up to 16.7 % in patients infected within the first postoperative week compared with 3.5 % in those infected later [7].

In our analysis, mechanical ventilation duration, as well as dobutamine and norepinephrine requirements, were significantly greater in the postoperative COVID-19 group, supporting the role of severe respiratory failure and septic or cardiogenic shock as the primary pathophysiological mechanisms of death, as described in earlier studies [1,6].

In contrast, patients who had recovered from COVID-19 prior to admission experienced a significantly milder postoperative course. This aligns with the findings of the large prospective *COVIDSurg Collaborative* study, which demonstrated that postoperative mortality decreases in proportion to the interval between infection and surgery, reaching background population levels only after the 7th week [3]. In our sample, this group had a significantly younger mean age, shorter hospital and ICU stays, and fewer critical complications. Such results reinforce prior recommendations to postpone elective cardiac surgery for at least 6-7 weeks after COVID-19, even in cases with mild or asymptomatic disease [3,4].

Laboratory comparisons between groups provide further insight into the pathophysiological basis of these clinical differences. Postoperative COVID-19 was associated with more severe thrombocytopenia, reduced hematocrit, hyperglycemia, acid-base disturbances (acidosis or alkalosis), hypoalbuminemia, and elevated urea levels – findings consistent with systemic inflammation, coagulation disorders, renal impairment, and metabolic dysregulation. These changes align with literature de-

scribing post-COVID endothelial dysfunction, systemic proinflammatory state, immunosuppression, and residual multi-organ dysfunction [1,9,10]. The combination of cardiac surgical inflammatory stress with residual COVID-19 manifestations appears to produce a synergistic detrimental effect on the lungs, myocardium, kidneys, and coagulation system [1].

It is important to note that even in the literature, predicting individual risk after severe COVID-19 remains challenging, as residual pathophysiological changes may persist for more than 2-3 months [4]. In our study, patients with a mild course of preoperative COVID-19 (59.4 % of this subgroup) demonstrated a low postoperative mortality rate of 4.9 %, while those with moderate disease had no fatal outcomes, suggesting that, in the absence of severe pulmonary or systemic complications, cardiac surgery may be performed with acceptable risk following adequate recovery. Conversely, severe preoperative COVID-19 was associated with a markedly higher mortality (33.3 %), underscoring the need for a cautious, individualized risk–benefit assessment in such cases. These findings are consistent with previously published data, which recommend postponing elective surgery for at least 6-7 weeks after SARS-CoV-2 infection to minimize perioperative complications and mortality [3,4].

However, our results also highlight the particularly unfavorable prognosis of postoperative COVID-19, where severe infection was observed in 59.3 % of patients and was universally associated with mortality (100 %). Importantly, even patients with mild or moderate forms of COVID-19 contracted during the early postoperative period required prolonged intensive care, though they ultimately survived. This contrast between pre- and postoperative infection emphasizes the decisive impact of infection timing: while optimized timing of surgery after recovery may mitigate risks in preoperative cases, postoperative COVID-19 represents a critical determinant of adverse outcomes, likely driven by additive effects of surgical stress, cardiopulmonary bypass, and immune dysregulation.

Postoperative infection, however, represents a fundamentally different scenario. As shown in our study, patients with hospital-acquired COVID-19 had markedly higher mortality (59.3 % vs. 4.4 %), longer mechanical ventilation times (169.7 ± 140.8 h vs. 18.1 ± 35.3 h), greater inotropic requirements (dobutamine: 125.2 ± 88.4 h vs. 21.7 ± 37.2 h; norepinephrine: 73.7 ± 65.2 h vs. 7.2 ± 15.6 h), and significantly prolonged ICU stays (17.1 ± 13.8 days vs. 5.1 ± 4.9 days). These findings are consistent with European observational data, where infection within the first 1–5 days after surgery was associated with mortality rates exceeding 15-20 % [7].

A particularly unfavorable scenario was infection occurring within the first postoperative week, with subsequent clinical manifestation of COVID-19 after day 10. In such cases, the disease course was characterized by progressive respiratory failure, the development of septic or cardiogenic shock, prolonged vasopressor and ventilatory support, and a substantially increased risk of death. Thus, the most critical situation remains early hospital-acquired COVID-19 after cardiac surgery, emphasizing the necessity for strict in-hospital infection control and early detection of even minimal symptoms in postoperative patients [6].

In summary, our study supports the key conclusions of current literature regarding the impact of COVID-19 infection timing on postoperative outcomes. Postoperative infection is associated with higher rates of multi-organ failure, prolonged mechanical ventilation, extended vasoactive therapy, and increased mortality. For patients with preoperative COVID-19 and an adequate recovery interval, surgery is potentially safe if clinical recovery and stability are achieved. The identified clinical and analytical associations underscore the importance of considering not only prior COVID-19 history but also the timing of infection relative to surgery when assessing risk, planning procedures, and stratifying prognosis.

Conclusions. The results of this clinical and analytical study indicate that the timing of COVID-19 infection plays a decisive role in determining the postoperative prognosis of patients undergoing valvular surgery for infective endocarditis. Hospital-acquired (postoperative) COVID-19 is associated with a significantly higher incidence of critical complications, including respiratory failure, acute cardiac and renal dysfunction, prolonged inotropic and ventilatory support, elevated glucose and urea levels, and thrombocytopenia. These complications were accompanied by markedly longer durations of mechanical ventilation, ICU stay, and total hospitalization.

Postoperative mortality in this cohort was substantially higher compared with patients who had COVID-19 prior to admission. Conversely, the preoperative COVID-19 group demonstrated more favorable outcomes, provided that there was an adequate interval between recovery from infection and surgery. This finding aligns with global literature identifying a safe interval of approximately 6-7 weeks post-recovery for most patients.

Correlation analysis revealed a strong association between mortality and indicators of multi-organ failure, including prolonged use of dobutamine and norepinephrine, development of respiratory failure, hypovolemia, hypoxia, and metabolic disturbances. Contingency analysis confirmed the statistically significant impact of these variables on treatment outcomes.

Thus, the study establishes that:

1. The timing of SARS-CoV-2 infection is a key predictor of outcomes after valvular surgery. In our cohort, hospital-acquired (postoperative) COVID-19 was associated with significantly higher mortality (59.3 % vs. 4.4 % in patients with preoperative COVID-19), prolonged ICU stay (17.1 ± 13.8 days vs. 5.1 ± 4.9 days), extended mechanical ventilation (169.7 ± 140.8 h vs. 18.1 ± 35.3 h), and greater need for inotropic/vasopressor support.
2. Patients with preoperative COVID-19 demonstrated significantly better outcomes; however, in our cohort

the interval between infection and surgery ranged from 1 to 24 months. This supports the conclusion that delaying surgery after COVID-19 is reasonable. Our findings are consistent with literature reports (*COVIDSurg Collaborative, El-Boghdadly et al.*) indicating that an interval of at least 6–7 weeks is optimal. At the same time, in clinical practice, urgent procedures were unavoidable (11 cases in the preoperative group, 6 in the postoperative group) due to infective endocarditis or decompensated valvular disease.

3. Correlation analysis confirmed strong associations between mortality and indicators of multiorgan dysfunction,particularly the duration of dobutamine infusion (r=0.84), norepinephrine use (r=0.76), postoperative respiratory failure requiring mechanical ventilation (r=0.76), and pneumonia (r=0.62). In contrast, sex, arterial hypertension, and microbiological findings showed no statistically significant impact on outcomes.
4. Practical implications: our findings indicate that the most unfavorable scenario is early hospital-acquired COVID-19 occurring within the first postoperative week, with clinical manifestation after day 10. This underscores the necessity for strict infection control measures in cardiac surgery units, early detection of even minimal COVID-19 symptoms in postoperative patients, and individualized surgical planning in the post-COVID era.

Список використаних джерел
References

1. Widnyana IMG, Senapathi TGA, Cindryani M, Juwita N, Jeanne B. Pulmonary Complications and 30-Day Mortality Rate in COVID-19 Patients Undergoing Surgery: A Systematic Review and Meta-Analysis. Bali J Anesthesiol. 2022 Oct-Dec;6(4):201-9. https://doi.org/10.4103/bjoa.bjoa_182_22
2. Ismail NA, Jaapar AN, Yunus AM, Sanusi AR, Taib ME, et al. (2022) Outcome of adult cardiac surgery following COVID-19 infection in unvaccinated population in a national tertiary centre. PLOS ONE 17(4): e0266056. https://doi.org/10.1371/journal.pone.0266056
3. COVIDSurg Collaborative; GlobalSurg Collaborative. Timing of surgery following SARS-CoV-2 infection: an international prospective cohort study. Anaesthesia. 2021 Jun;76(6):748-758. doi: https://doi.org/10.1111/anae.15458 . Epub 2021 Mar 9.
4. El-Boghdadly K, Cook TM, Goodacre T, Kua J, Denmark S, Mercer N, Moonesinghe SR, Summerton DJ. Timing of elective surgery and risk assessment after SARS-CoV-2 infection: 2023 update: A multidisciplinary consensus statement on behalf of the Association of Anaesthetists, Federation of Surgical Specialty Associations, Royal College of Anaesthetists and Royal College of Surgeons of England. Anaesthesia. 2023 Sep;78(9):1147-1152. doi: https://doi.org/10.1111/anae.16061 . Epub 2023 Jun 19.
5. Codner JA, Archer RH, Lynde GC, Sharma J. Timing is Everything: Surgical Outcomes for SARS-CoV-2 Positive Patients. World J Surg. 2023 Feb;47(2):437-444. doi: https://doi.org/10.1007/s00268-022-06814-4 . Epub 2022 Oct 31.
6. Fattouch K, Corrao S, Augugliaro E, Minacapelli A, Nogara A, Zambelli G, Argano C, Moscarelli M. Cardiac surgery outcomes in patients with coronavirus disease 2019 (COVID-19): A case-series report. J Thorac Cardiovasc Surg. 2022 Mar;163(3):1085-1092.e3. doi: https://doi.org/10.1016/j.jtcvs.2020.09.138 . Epub 2020 Oct 22.
7. Spadaccio C, Rose D, Candura D, Lopez Marco A, Cerillo A, Stefano P, Nasso G, Ramoni E, Fattouch K, Minacapelli A, Oo AY, Speziale G, Shelton K, Berra L, Bose A, Moscarelli M. Effect of Hospital-associated SARS-CoV-2 Infections in Cardiac Surgery: A Multicenter Study. Ann Thorac Surg. 2024 Jan;117(1):213-219. doi: https://doi.org/10.1016/j.athoracsur.2022.05.034 . Epub 2022 Jun 8.
8. Miceli A. Commentary: When a cardiac surgeon takes care of patients with Coronavirus Disease 2019: It's gonna be ok! J Thorac Cardiovasc Surg. 2020 Aug;160(2):e43-e44. doi: https://doi.org/10.1016/j.jtcvs.2020.04.027. Epub 2020 Apr 15.
9. Van Beusekom M. Delaying surgery after COVID may lower risk of cardiac complications. Minneapolis (MN): CIDRAP; 2025 Jun 26. Available from: https://www.cidrap.umn.edu/covid-19/delaying-surgery-after-covid-may-lower-risk-cardiac-complications
10. Song CY, Xu J, He JQ, et al. Immune dysfunction following COVID-19, especially in severe patients. Sci Rep. 2020;10:15838. https://doi.org/10.1038/s41598-020-72718-9

Результати хірургічного лікування клапанних вад серця в епоху COVID-19: стратифікований аналіз залежно від часу інфікування

Косован Д. М.

ДУ «Національний інститут серцево-судинної хірургії ім. М.М. Амосова НАМН України», м. Київ, Україна

Резюме

Актуальність. Пандемія COVID-19 створила безпрецедентні виклики в кардіохірургії, зокрема щодо вибору оптимального часу для втручань у пацієнтів із перенесеним SARS-CoV-2. Вплив часових інтервалів між інфікуванням і хірургією на результати лікування залишається недостатньо вивченим.

Мета. Оцінити вплив термінів інфікування COVID-19 – до госпіталізації або післяопераційного (нозокоміального) зараження – на госпітальні результати хірургічного лікування набутих вад серця.

Матеріали та методи. Ретроспективне клініко-аналітичне дослідження включало 96 пацієнтів, яким у 2021-2024 рр. виконано хірургічну корекцію набутих вад серця. Пацієнтів стратифіковано за часом виникнення COVID-19 щодо операції: (1) до госпіталізації, (2) після операції, (3) обидва випадки. Аналізувалися строки інфікування, тяжкість перебігу, супутні захворювання, хірургічні параметри, госпітальна летальність і ускладнення.

Результати. Показники летальності та ускладнень істотно відрізнялися залежно від часу інфікування. Найвищу смертність (33,3 %) та тривале перебування в реанімації зафіксовано у пацієнтів із нозокоміальним COVID-19. Натомість пацієнти з віддаленим перенесеним COVID-19 (>3 міс) мали зіставні результати з COVID-негативними. Незалежними факторами ризику виявлено багатоклапанне втручання, недавнє інфікування (≤30 діб) та післяопераційний COVID-19 ($p<0,05$).

Висновки. Час інфікування COVID-19 суттєво впливає на результати клапанної хірургії. За можливості планові втручання слід відтерміновувати щонайменше на 6–7 тижнів після перенесеного COVID-19. Профілактика госпітального інфікування залишається критично важливою з огляду на його значний вплив на летальність.

***Ключові слова**: COVID-19, кардіохірургія, набуті вади серця, післяопераційні ускладнення, госпітальна летальність, час інфікування.*

Стаття надійшла в редакцію / Received: 31.07.2025
Після доопрацювання / Revised: 21.08.2025
Прийнято до друку / Accepted:29.08.2025