GBMN GEORGIAN BIOMEDICAL & MEDICAL NEXUS
Former Georgian Biomedical News
VOLUME 4 ISSUE 3. JUL-SEP 2026

# Short-Course Dexamethasone in Acute Pancreatitis: Preliminary Clinical and Biomarker Findings from a Pilot Study

Roberti Avalishvili[1,ID], Mamuka Gurgenidze[1,ID], George Asatiani[1,ID]

## ABSTRACT

**BACKGROUND.** Acute pancreatitis (AP) is a clinically significant condition that can progress to a severe, life-threatening disease with substantial morbidity and mortality. Experimental studies in animal models have demonstrated beneficial effects of corticosteroid therapy on pancreatic inflammation and disease severity. In addition, several clinical studies in humans have reported promising results. However, the available evidence remains limited and inconclusive because of small sample sizes, heterogeneous study designs, and inconsistent outcomes.

**OBJECTIVES.** The study analyzes the effectiveness of dexamethasone therapy in AP and its impact on clinical and laboratory parameters and length of hospital stay. The study aimed to compare these outcomes in patients treated with and without dexamethasone therapy.

**METHODS.** A prospective, single-blind, pilot randomized clinical study was conducted, including 20 patients aged 45–85 years with moderately severe and severe acute pancreatitis treated at The First University Clinic, Simon Khechinashvili University Clinic, and The First Medical Center in Tbilisi, Georgia. Patients were randomly assigned (1:1) using a computer-generated randomization sequence generated with IBM SPSS Statistics version 23 (IBM Corp., Armonk, NY, USA) to receive either standard therapy (control, n=10) or adjunctive dexamethasone (0.5 mg/kg/day for 3 consecutive days) plus standard therapy (n=10). The primary endpoints were changes in serum pancreatitis-associated protein (PAP) and interleukin-6 (IL-6) levels from day 1 to day 5. Clinical outcomes and laboratory parameters, including procalcitonin, serum lipase, leukocyte count, and C-reactive protein (CRP), were also assessed. Statistical analyses included analysis of variance (ANOVA) for continuous variables and Fisher'sFisher's exact test for categorical variables. A two-sided p-value <0.05 was considered statistically significant.

**RESULTS.** Patients in the control group had a longer hospital stay compared with those in the dexamethasone group (15.1±2.60 vs 12.3±1.77 days, respectively). No significant between-group differences were observed in CRP, IL-6, or procalcitonin levels during the early study period. However, by day 5 of hospitalization, the dexamethasone group demonstrated significantly lower CRP, IL-6, and procalcitonin levels compared with the control group. Increased PAP levels were also observed in the dexamethasone group, suggesting a possible effect on pancreatic tissue response and recovery. Serum lipase and leukocyte counts did not differ significantly between the groups.

**CONCLUSIONS.** In this small preliminary pilot study, adjunctive dexamethasone was associated with shorter hospitalization and favorable day-5 trends in selected inflammatory markers. However, these findings should be interpreted with caution because of the small sample size. Larger, adequately powered randomized controlled trials are needed to confirm these preliminary findings and further evaluate the efficacy and safety of adjunctive dexamethasone in acute pancreatitis.

**KEYWORDS.** Acute pancreatitis (AP); C-reactive protein (CRP); Corticosteroid therapy; Dexamethasone; Inflammation; Interleukin-6 (IL-6); Leukocytes; Lipase; Pancreatitis-associated protein (PAP).

DOI. **10.52340/GBMN.2026.01.01.183**

## BACKGROUND

Acute pancreatitis (AP) is a common acute abdominal condition with multiple etiologies. Severe acute pancreatitis (SAP) may be life-threatening and is associated with significant morbidity, mortality, and economic burden. Over the last two decades, the global incidence of this disease has increased steadily and continues to rise. In the UK, approximately 56 out of every 100,000 people are diagnosed with AP, while in the United States, over 220,000 people are hospitalized annually due to this disease. [1] Recent advancements in its management have primarily focused on the development of early-phase medical interventions targeting inflammatory pathways, optimization of supportive treatment (including fluid resuscitation, pain management, and nutritional management), appropriate use of antibiotics, implementation of minimally invasive interventions for infected necrosis, and the necessity of follow-up for long-term complications. These advancements have significantly improved personalized management and overall outcomes of AP. Despite

GBMN GEORGIAN BIOMEDICAL & MEDICAL NEXUS
Former Georgian Biomedical News
VOLUME 4 ISSUE 3. JUL-SEP 2026

these efforts, early-phase medical interventions to mitigate disease progression are still lacking, and substantial variability in disease severity and outcomes persists. [2]

In recent years, there has been ongoing discussion about the role and effectiveness of corticosteroid therapy in the treatment of inflammatory pancreatic diseases. [3,4] Glucocorticoids are successfully used in inflammatory processes like sepsis, pneumonia, burns, and acute respiratory distress syndrome. [5-7] In recent decades, the role of glucocorticoid therapy in treating AP has been under investigation. Corticosteroids are not established routine therapy for general acute pancreatitis, and existing human data remain insufficient for guideline-level recommendation. According to existing literature, this therapy has been shown to attenuate disease progression, reduce pancreatic inflammation, and improve clinical outcomes in animals. [8-10] However, studies in humans are scarce, often equivocal, and require solid scientific evidence. [11-13]

Inflammatory injury induces substantial changes in the pancreatic parenchyma, including a pronounced increase in PAP expression. Free radicals or cytokines upregulate PAP expression, and such upregulation confers cellular resistance to apoptosis. [14]

We hypothesize that dexamethasone may be beneficial in acute pancreatitis by inducing the PAP gene and increasing PAP protein production. Furthermore, attenuation of the inflammatory cascade may help prevent disease progression and reduce the incidence of severe complications. As a non-specific anti-inflammatory agent, dexamethasone can inhibit the production of multiple inflammatory mediators simultaneously. Therefore, suppression of inflammatory mediators may represent an important therapeutic target in the management of acute pancreatitis.

## METHODS

### General data of patients

The study was conducted at the First University Clinic and the Simon Khechinashvili University Clinic. It included 20 patients diagnosed with moderately severe (MSAP) and severe AP. The study comprised 12 male and 8 female participants. The mean age of the patients was 60.5 years (range 45–80 years). The diagnosis, treatment, and assessment of disease severity were based on the definitions and criteria of the Revised Atlanta Classification and the International Consensus Guidelines. [15,16] Ethical approval for the study was granted by the Ethics Commission of Tbilisi State Medical University (Approval No. 5-2023/106), in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from all patients before participation in the study.

### Inclusion and exclusion criteria

Patients aged 18 years or older who were diagnosed with MSAP or SAP and agreed to participate in the study were included.

Patients meeting any of the following criteria were excluded from the study: grade III arterial hypertension, peptic ulcer disease of the stomach or duodenum, inflammatory bowel disease (including ulcerative colitis or Crohn'sCrohn's disease), stage IV osteoporosis, liver cirrhosis (Child–Pugh class C), acute or chronic heart failure (NYHA class III–IV), Cushing'sCushing's disease, glaucoma, psychotic disorders, epilepsy, Alzheimer'sAlzheimer's disease, immunosuppression, refusal to participate in the study, and the presence of any malignant disease.

### Randomization and group allocation

Eligible patients were randomly assigned in a 1:1 ratio using a computer-generated randomization sequence generated with IBM SPSS software. Ten patients were allocated to the experimental group and ten to the control group. The study was conducted as a single-blind randomized trial. Patients, nursing staff, and laboratory personnel performing biomarker analyses were blinded to treatment allocation, whereas treating physicians were aware of group assignment. All randomized patients completed the study and were included in the final analysis.

### Diagnosis

The patients experienced epigastric pain of visceral origin, often radiating to the back. This pain was persistent and, in some cases, poorly localized. Additional clinical symptoms included fever, nausea,

vomiting, paralytic ileus, abdominal distention, and elevated serum lipase levels at least 3 times higher than reference values. Ultrasound examinations revealed pancreatic edema and, in some cases, peripancreatic and intra-abdominal fluid accumulation. Computed tomography (CT), including contrast-enhanced dynamic CT, showed pancreatic enlargement, peripancreatic fluid accumulation, intra-abdominal fluid collection, necrotic changes within the pancreatic parenchyma, and edema of adjacent organs.

### Treatment

After a diagnosis of MSAP and SAP, patients received standard supportive management according to the Revised Atlanta Classification and current international guidelines for acute pancreatitis15,16. Supportive therapy was provided according to current international guidelines and included intravenous fluid resuscitation with Lactated Ringer'sRinger's solution. A moderate infusion rate (approximately 1.5 mL/kg/h) was used and adjusted according to the patient's hemodynamic status, urine output, hematocrit, blood urea nitrogen, and other clinical indicators of fluid balance. Additional fluid boluses were administered in patients presenting with hypovolemia or hypotension. Analgesia with opioid or non-opioid agents was administered according to pain severity. Early oral or enteral nutrition was initiated as tolerated. Antibiotics were not routinely administered and were reserved for patients with suspected or confirmed infection. Thromboprophylaxis, ICU admission, ERCP, and other invasive interventions were performed according to established clinical indications. Glycemic monitoring was performed throughout hospitalization, particularly in patients receiving corticosteroid therapy. The same supportive treatment protocol was applied across all participating centers. Treatment protocols were consistent between participating centers.

In the experimental group, in addition to the standard treatment regimen mentioned above, patients received intravenous dexamethasone at a dose of 0.5 mg/kg/day for 3 consecutive days. The daily dose was divided into three intravenous administrations and was calculated according to actual body weight. Treatment was initiated within the first 24 hours after symptom onset, immediately following hospital admission and confirmation of the diagnosis of acute pancreatitis. No tapering was required after completion of the short-course dexamethasone regimen. All patients received the same treatment duration and dosing protocol, based on previously published clinical studies. Steroid-related adverse events were monitored throughout the treatment period, including hyperglycemia, infectious complications, gastrointestinal bleeding, delirium, and other clinically relevant adverse effects.

Patients were discharged according to predefined clinical criteria based on current international guidelines16. Discharge criteria included tolerance of oral nutrition, improvement of inflammatory markers (CRP and/or leukocyte count), absence of persistent fever, adequate pain control with no or minimal analgesic requirements, and no further need for inpatient management.

### Pancreatitis-associated protein (PAP), cytokine and other laboratory parameters

PAP and IL-6 levels were measured at 24 hours, on day 3, and on day 5 after treatment initiation using the enzyme-linked immunosorbent assay (ELISA) method with a DIA Reader analyzer in the Ivane Beritashvili Center of Experimental Biomedicine.

Other laboratory parameters, including serum lipase, complete blood count (CBC), CRP, and procalcitonin levels, were also measured at the same time intervals in the clinical laboratory.

### Study endpoints

The primary endpoints of the study were the changes in serum PAP and IL-6 levels between day 1 and day 5. Secondary endpoints included changes in C-reactive protein (CRP), procalcitonin, serum lipase, and leukocyte count, as well as clinical outcomes, including length of hospital stay and treatment-related adverse events.

### Statistical analysis

Statistical analyses were performed using IBM SPSS Statistics version 23 (IBM Corp., Armonk, NY, USA).


ISSN (Online): 2720-8796 ISSN (Print): 2720-7994

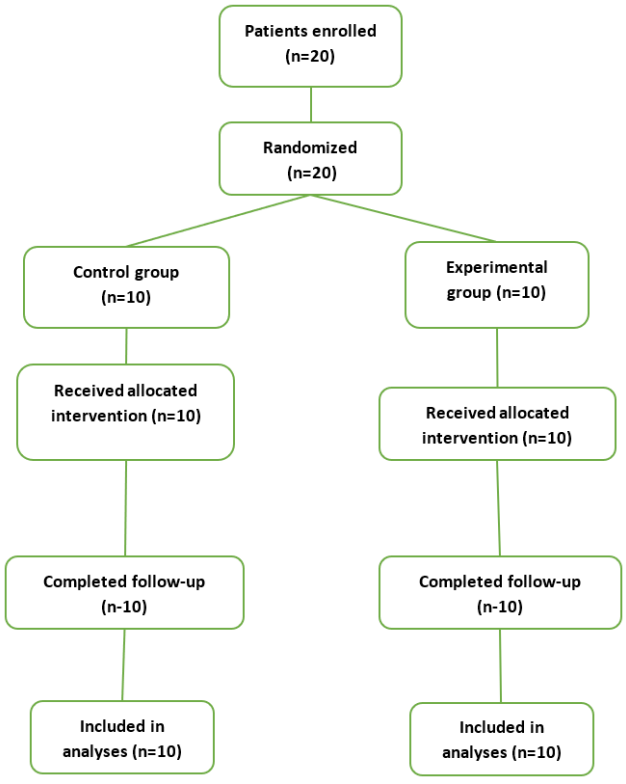
GBMN GEORGIAN BIOMEDICAL & MEDICAL NEXUS
Former Georgian Biomedical News
VOLUME 4 ISSUE 3. JUL-SEP 2026

Continuous variables were compared using analysis of variance (ANOVA), whereas categorical variables were compared using Fisher's exact test. Correlations between variables were assessed using Pearson's or Spearman'sSpearman's correlation coefficients, as appropriate according to data distribution. A two-sided $p<0.05$ was considered statistically significant.

## RESULTS

The flow of participants throughout the study is shown in **FIGURE 1**. All 20 randomized patients completed the study and were included in the final analysis.

**FIGURE 1**. Flow diagram of patient enrollment, randomization, follow-up, and analysis




The baseline demographic characteristics of the study population are presented in **TABLE 1**. The results of our statistical analysis are shown in **TABLE 2**.

Patients in the experimental group had a significantly shorter hospital stay than those in the control group (12.3±1.77 vs 15.1±2.60 days, respectively; p=0.011). The duration of hospital stay ranged from 11 to 19 days in the control group and from 10 to 15 days in the experimental group.

**TABLE 1**. Baseline characteristics of the study population

| Variable | Control (n=10) | Dexamethasone (n=10) | P value |
|---|---|---|---|
| Age, mean±SD | 66.2±8.4 | 71.6±7.1 | 0.139 |
| Male sex, n (%) | 6 (60%) | 7 (70%) | 0.639 |
| Female sex, n (%) | 4 (40%) | 3 (30%) | 0.639 |
| Etiology, n (%) | | | |
| Biliary | 6 (60%) | 5 (50%) | 0.524 |
| Alcoholic | 3(30%) | 2 (20%) | |
| Idiopathic | 1 (10%) | 3 (30%) | |
| Time from symptom onset to admission, n (%) | | | |
| ≤24 h | 9 (90%) | 7 (70%) | 0.582 |
| >24 h | 1 (10%) | 3 (30%) | |
| SIRS, n (%) | | | |
| Yes | 5 (50%) | 7 (70%) | 0.650 |
| No | 5 (50%) | 3 (30%) | |
| Disease severity (Atlanta classification), n (%) | | | |
| SAP | 1 (10%) | 2 (20%) | 1.000 |
| MSAP | 9 (90%) | 8 (80%) | |
| Oral nutrition (p.o) | 10 (100%) | 10 (100%) | 1.000 |
| Antibiotic use | | | |
| Yes | 1 (10%) | 2 (20%) | 1.000 |
| No | 9 (90%) | 8 (80%) | |
| ICU admission, n (%) | | | |
| Yes | 1 (10%) | 2 (20%) | 1.000 |
| No | 9 (90%) | 8 (80%) | |
| CRP (mg/L), mean±SD | 226.90 | 173.10 | 0.043 |
| IL-6 (pg/L), mean±SD | 84.2 | 87.1 | 0.549 |
| PAP (ng/mL), mean±SD | 75.5 | 93.7 | 0.044 |
| Lipase (U/L), mean ± SD | 727.9 | 887.5 | 0.062 |
| Procalcitonin (ng/mL), mean±SD | 2.4 | 2.69 | 0.226 |

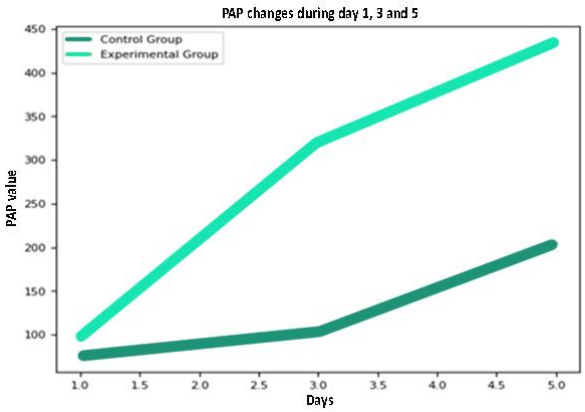
GBMN GEORGIAN BIOMEDICAL & MEDICAL NEXUS
Former Georgian Biomedical News
VOLUME 4 ISSUE 3. JUL-SEP 2026

**TABLE 2**. Length of hospitalization and laboratory parameters measured on days 1, 3, and 5 in the control and dexamethasone groups

| Parameter | Control Group (mean) | Experimental group (mean) | P value (day 1) | P value (day 3) | P value (day 5) | P value (mean days 1–5) |
|---|---|---|---|---|---|---|
| Length of hospitalization, days | 15.1±2.60 | 12.3±1.77 | - | - | - | 0.011 |
| PAP, ng/mL | 127.33 | 283.13 | 0.044 | 0.001 | 0.046 | 0.001 |
| IL 6, pg/L | 91.83 | 80.57 | 0.549 | 0.227 | 0.017 | 0.12 |
| Lipase, U/L | 418.50 | 436 | 0.062 | 0.833 | 0.484 | 0.83 |
| CRP, mg/L | 152.00 | 124.57 | 0.043 | 0.904 | 0.011 | 0.11 |
| leukocyte, $\times 10^9$/L | 12.27 | 12.73 | 0.538 | 0.216 | 0.900 | 0.61 |
| Procalcitonin, ng/mL | 1.77 | 1.70 | 0.226 | 0.328 | 0.040 | 0.74 |

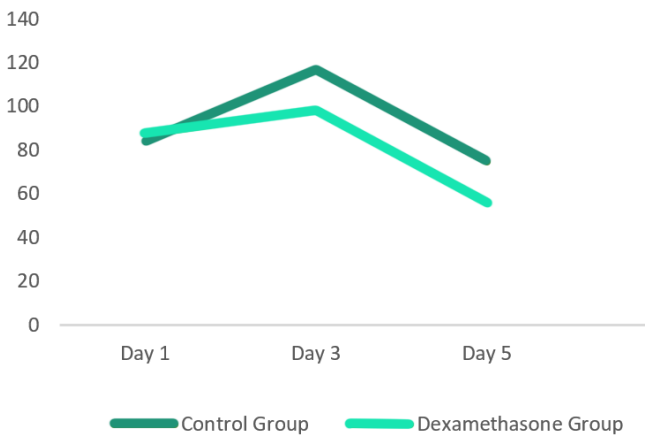
On day 1, PAP levels were 75.5 ng/mL in the control group and 93.7 ng/mL in the experimental group (p=0.044). On day 3, PAP levels were 102 ng/mL in the control group and 211 ng/mL in the experimental group (p=0.001). On day 5, PAP levels were 204 ng/mL in the control group and 435 ng/mL in the experimental group (p=0.046) (**FIG.2**). The increase in PAP levels observed in the experimental group may be associated with dexamethasone administration and its influence on acute-phase protein expression.

**FIGURE 2**. Dynamics of PAP levels on days 1, 3 and 5 in the experimental and control groups




As shown in **FIGURE 3**, baseline IL-6 levels were comparable between the control and experimental groups (84 pg/L vs. 87 pg/L; p=0.549). On day 3, no statistically significant difference was observed between the groups (116.6 pg/L vs. 98.3 pg/L; p=0.227). By day 5, IL-6 levels were significantly lower in the experimental group than in the control group (56.0 pg/L vs. 74.7 pg/L; p=0.017). Baseline CRP levels were significantly higher in the control group than in the experimental group (226.6 mg/L vs. 173 mg/L; p=0.043). By day 3, CRP values had declined markedly in both groups, with no statistically significant difference between them (140 mg/L vs. 138 mg/L; p=0.904). By day 5, CRP levels remained lower in both groups but were significantly lower in the experimental group than in the control group (61.8 mg/L vs. 88.3 mg/L; p=0.011). Procalcitonin showed a similar pattern. No significant differences were observed at baseline (2.4 ng/mL vs. 2.69 ng/mL; p=0.226) or on day 3 (1.88 ng/mL vs. 1.73 ng/mL; p=0.328). However, by day 5, procalcitonin levels were significantly lower in the experimental group than in the control group (0.68 ng/mL vs. 1.02 ng/mL; p=0.040).

**FIGURE 3**. Changes in serum IL-6 levels on days 1, 3, and 5 in the control and experimental groups




Georgian Biomedical and Medical Nexus (Former Georgian Biomedical News)
ISSN (Online): 2720-8796 ISSN (Print): 2720-7994

GBMN GEORGIAN BIOMEDICAL & MEDICAL NEXUS
Former Georgian Biomedical News
VOLUME 4 ISSUE 3. JUL-SEP 2026

Leukocyte counts and serum lipase levels showed no significant differences between the control and Experimental groups at baseline, day 3, or day 5 (all p>0.05).

### Clinical outcomes

During hospitalization, ERCP was performed in 4 of the 20 patients. Following recovery from acute pancreatitis, 5 patients underwent cholecystectomy. ICU admission was required in one patient in the control group and two patients in the dexamethasone group. These patients developed infected pancreatic necrosis and received antibiotic therapy according to clinical indications. None of the patients required necrosectomy or drainage procedures. No readmissions or mortality were recorded in either group.

### Safety outcomes

No serious adverse events related to dexamethasone therapy were observed during the study period. No patients developed gastrointestinal bleeding, delirium/psychosis, fungal infections, or transient hyperglycemia requiring insulin therapy. In the dexamethasone group, transient elevation of blood pressure was recorded in two patients, with maximum values of 160/100 mmHg and 155/95 mmHg, respectively. These episodes were managed conservatively and did not require pharmacological intervention or discontinuation of dexamethasone therapy.

## DISCUSSION

Although the risks associated with AP are well known, current treatment strategies for AP are supportive and include fluid resuscitation, pain management, and nutritional support. Current treatment paradigms do not target the underlying pathophysiological mechanisms of this disease. [17]

A better understanding of the pathophysiological mechanisms underlying AP is essential, particularly regarding why only a subset of patients progresses from localized pancreatic inflammation to SIRS. Although most patients experience a mild and self-limiting course, approximately 20% develop severe disease characterized by SIRS, followed by shock and multi-organ failure (MOF). [18] The clinical course of SAP is generally divided into two phases. The early phase refers to the first 7–14 days after the onset of acute pancreatitis and is characterized by SIRS and MOF resulting from the release of large amounts of cytokines. In the late phase (14–28 days after the onset of acute pancreatitis), the disease may be complicated by infected pancreatic necrosis and secondary MOF, as described by Fu et al. [19]

Interleukins (IL), as signaling molecules, play a major role in modulating and regulating the inflammatory response in the pancreas. They can either promote inflammation (i.e., pro-inflammatory ILs) or suppress it (i.e., anti-inflammatory ILs), depending on the specific IL involved. During AP, the pancreas can suffer significant tissue damage due to inflammation and the immune response. ILs are responsible for recruiting and activating immune cells (i.e., macrophages and neutrophils), which can lead to tissue injury. Understanding the role of specific ILs can help in developing strategies to minimize tissue damage. [20]

Under normal physiological conditions, serum PAP is virtually undetectable. However, its concentration increases markedly within six hours after the onset of acute pancreatitis and reaches peak levels approximately 48 hours later. Several biological functions have been attributed to PAP, including antibacterial, antiapoptotic, and mitogenic activities. PAP proteins serve a protective role in AP, and this protective effect may be via downregulation of acute-phase cytokine gene expression in the peripheral immune system. [14]

According to Closa et al., PAP can bind and aggregate several bacterial strains from the intestinal flora, including Gram-positive and Gram-negative, aerobic and anaerobic bacteria, although without inhibiting their growth. These findings suggest that PAP may act as an endogenous antibacterial agent and could play a protective role in preventing infectious complications of acute AP. [21]

In the present study, dexamethasone administration attenuated the inflammatory response, particularly by reducing serum IL-6 levels. Notably, IL-6 concentrations on the fifth day of treatment were significantly lower in the experimental group than in the control group. Lower


ISSN (Online): 2720-8796 ISSN (Print): 2720-7994

levels of procalcitonin and C-reactive protein (CRP), especially on the fifth treatment day, may also indicate attenuation of systemic inflammatory activity and a potentially milder disease course. Furthermore, patients receiving dexamethasone had a significantly shorter duration of hospitalization. Dexamethasone administration was associated with higher serum PAP levels together with lower inflammatory marker levels compared with the control group. However, no statistically significant differences were observed between the experimental and control groups with respect to leukocyte counts and serum lipase levels.

Although baseline CRP and PAP levels differed between the study groups, patients receiving dexamethasone demonstrated more favorable temporal trends throughout the study. The dexamethasone group showed a progressive increase in serum PAP levels together with a more pronounced decline in CRP, IL-6, and procalcitonin by day 5. In contrast, no significant differences were observed in leukocyte count or serum lipase levels. Future adequately powered multicenter randomized controlled trials are warranted to validate these preliminary findings and further evaluate the efficacy and safety of adjunctive dexamethasone therapy in acute pancreatitis.

## CONCLUSIONS

In this small preliminary pilot study, adjunctive dexamethasone was associated with shorter hospitalization, higher serum PAP levels, and favorable day-5 trends in selected inflammatory markers. However, baseline imbalances between the study groups, the small sample size, and the preliminary nature of these findings preclude definitive conclusions regarding treatment efficacy. Larger randomized controlled trials with standardized treatment protocols, severity stratification, and comprehensive safety assessment are required to confirm these findings.

## AUTHOR AFFILIATION

1. Department of Surgery, Tbilisi State Medical University, Tbilisi, Georgia

## ACKNOWLEDGEMENTS

The Ethics Committee of Tbilisi State Medical University approved the study, and it was conducted in accordance with the principles of the Declaration of Helsinki. A legally certified bilateral agreement was established between Tbilisi State Medical University, the First University Clinic, and Simon Khechinashvili University Clinic for this research. All participants provided informed consent and voluntarily agreed to participate in the study.

The authors express their sincere gratitude to the above-mentioned institutions. Vano Kechakmadze for his support in the statistical analysis, and Davit Dzneladze and the Ivane Beritashvili Center of Experimental Biomedicine for providing the material and technical support essential for conducting this research.

## REFERENCES

1. Shah A, Mourad MM, Bramhall S. Acute pancreatitis: current perspectives on diagnosis and management. J Inflamm Res. 2018; 11:77- 85.
2. Beij A, Verdonk RC, et al. Acute Pancreatitis: An Update of Evidence-Based Management and Recent Trends in Treatment Strategies. United European Gastroenterol J. 2025;13(1):97–106.
3. Wang M, Jiang Z, Liang H. Glucocorticoids in acute pancreatitis: a propensity scores matching analysis. BMC Gastroenterol. 2021; 21:331.
4. Wang ZF, Liu C, Lu Y et al. Dexamethasone and dextran 40 treatment of 32 patients with severe acute pancreatitis. World J Gastroenterol. 2004; 10(9):1333–1336.
5. Huang J, Guo J, Li H, Huang W, Zhang T. Efficacy and safety of adjunctive corticosteroids therapy for patients with severe community-acquired pneumonia: A systematic review and meta-analysis. Medicine (Baltimore). 2019; 98(13): e14636.
6. Meduri G, Bridges L, Shih M, et al. Prolonged glucocorticoid treatment is associated with improved ARDS outcomes: analysis of individual patients' data from four randomized trials and trial-level meta-analysis of the updated literature Intensive Care Med. 2016;42(5):829-840.
7. Chan EYL, Chan MMK, et al. Use of glucocorticoids in the critical care setting: Science and clinical evidence Pharmacol Ther. 2020; 206:107428.
8. Pezzilli R, Fantini L. Glucocorticoids and acute pancreatitis: suggestions for future clinical trials. Journal of Pancreas. 2006;7(2):249-251.
9. Shimosegawa T, Are glucocorticoids really useful for the treatment of acute pancreatitis. J Gastroenterol. 2002;37(7):580–581.

10. Yang X, Yao L, et al. Experimental Acute Pancreatitis Models: History, Current Status, and Role in Translational Research. Front Physiol. 2020; 11:614591
11. Wang M, Jiang Z, Liang H. Glucocorticoids in acute pancreatitis: a propensity scores matching analysis. BMC Gastroenterol. 2021; 21:331.
12. Wang ZF, Liu C, Lu Y et al. Dexamethasone and dextran 40 treatment of 32 patients with severe acute pancreatitis. World J Gastroenterol. 2004; 10(9):1333–1336.
13. Ataalah B, Abdulrahman M, AI- Zakhari R, et al. Steroid-Induced Pancreatitis: A Challenging Diagnosis. Cureus. 2020;12(7): e8939.
14. Kandil E, Zhang H, Levi G, Lin YY, et al. Dexamethasone mediates protection against acute pancreatitis via upregulation of pancreatitis-associated proteins. World J Gastroenterol. 2006; 12(42):6806–11.
15. Banks PA, Bollen TL, Dervenis C, et al. Classification of acute pancreatitis – 2012: revision of the Atlanta classification and definitions by international consensus. Gut. 2013; 62:102–111.
16. IAP/APA/EPC/IPC/JPS Working Group. International Association of Pancreatology Revised Guidelines on Acute Pancreatitis 2025: Supported and Endorsed by the American Pancreatic Association, European Pancreatic Club, Indian Pancreas Club, and Japan Pancreas Society. Pancreatology. 2025;25(6):770-814.
17. Zaman S, Gorelick F. Acute pancreatitis: pathogenesis and emerging therapies. J Pancreatol. 2024 Jan 2;7(1):10–20.
18. Dhong LH, Liu ZM, Wang SJ, et al. Corticosteroid therapy for severe acute pancreatitis: a meta-analysis of randomized, controlled trials. Int J Clin Exp Pathol. 2015; 8(7):7654–7660.
19. Fu CY, Yeh CN, Hsu JT, et al. Timing of mortality in severe acute pancreatitis: Experience from 643 patients World J Gastroenterol. 2007; 13(13):1966–1969.
20. Rafaqat S, Patoulias D, Behnoush A. Interleukins: pathophysiological role in acute pancreatitis. Arch Med Sci. 2024; 20(1):138.
21. Closa D, Motoo Y, Iovanna JL. Pancreatitis-associated protein: From a lectin to an anti-inflammatory cytokine. World J Gastroenterol 2007;13(2):170–174.