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Trends in Immunotherapy

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Immunological and Clinical Implications of Surgical Techniques in Primary Inguinal Hernia Repair: Autoplasty and Alloplasty Compared

Kanat Mamakeev [1], Zhanybek Ashimov [1], Urmat Aitiev [2], Batyrkhan Niyazov [2], Omar Dinlossan [1], Sardar Kayumov [1] and Tugolbai Tagaev [3,*]

[1] Department of Scientific, National Surgical Center Named after M.M. Mamakeev, Ministry of Health of the Kyrgyz Republic, Bishkek 720000, Kyrgyzstan

[2] Department of General Surgery, Kyrgyz State Medical Institute, Retraining/Advanced Training Named after S.B. Daniyarov, Bishkek 720017, Kyrgyzstan

[3] Department of Hospital Internal Medicine, I.K. Akhunbaev Kyrgyz State Medical Academy, Bishkek 720020, Kyrgyzstan

[*] Correspondence: ttagaev22.kg@gmail.com

Received: 31 March 2026; **Revised:** 23 April 2026; **Accepted:** 29 May 2026; **Published:** 11 June 2026

Abstract: Inguinal hernia repair is performed over 20 million times annually, with outcomes reflecting mechanical reconstruction and immune activation. This study compared autoplasty and tension-free alloplasty for primary inguinal hernias, focusing on clinical outcomes and inflammatory responses. This prospective single-center study compared tissue-based autoplasty (Shouldice/Spasokukotsky) with tension-free mesh alloplasty (Lichtenstein) in 118 adults (mean age 46 ± 12.1 years; 93% male; autoplasty, n = 50; alloplasty, n = 68). Pain (verbal descriptor scale), complications, hospital stay, and systemic markers (C-reactive protein, neutrophil-to-lymphocyte ratio, interleukin-6, tumor necrosis factor-alpha, lymphocyte count) were assessed ($p < 0.05$). Alloplasty showed faster operation (48.4 ± 1.5 vs. 59.6 ± 2.6 min), less pain (day 1: 4.5 ± 0.5 vs. 5.7 ± 0.6; day 2: 1.1 ± 0.2 vs. 2.4 ± 0.3; day 5: 0.5 ± 0.1 vs. 1.2 ± 0.2), earlier walking (7.2 ± 2.1 vs. 21.4 ± 3.6 h), fewer complications (8.4% vs. 28.8%), and reduced hospitalization (3.6 ± 0.9 vs. 5.1 ± 1.2 days; all $p < 0.05$). Autoplasty showed higher inflammatory markers (C-reactive protein 32.5 ± 6.8 vs. 21.3 ± 5.4 mg/L; neutrophil-to-lymphocyte ratio 5.8 ± 1.2 vs. 3.9 ± 0.9; interleukin-6 48.7 ± 10.2 vs. 29.4 ± 8.6 pg/mL; tumor necrosis factor-alpha 26.5 ± 7.3 vs. 17.2 ± 5.8 pg/mL; all significant). Operative time was correlated with pain (r = 0.64; $p < 0.01$) and complications (r = 0.48; $p < 0.05$). Tension-free alloplasty improved recovery and reduced immune activation; however, non-randomization, single-center design, and limited follow-up constrain long-term inferences of the study.

Keywords: Inguinal Hernia Repair; Autoplasty; Alloplasty; Immune Activation; Inflammatory Markers; Postoperative Outcomes

1. Introduction

Inguinal hernia repair is among the most common surgeries globally, with over 20 million procedures performed annually, posing challenges to healthcare systems [1, 2]. Despite progress from tissue-based repairs to tension-free mesh techniques, postoperative results vary, with no universally superior method [1]. Research has

shown that recovery and long-term success depend on mechanical support, mesh characteristics, fixation methods, surgical techniques, and patient factors, such as pain sensitivity and hernia features [3–5]. Optimal outcomes require a personalized approach that combines technical accuracy with biological and clinical risk factors [6].

From a modern immunological standpoint, surgery triggers a coordinated innate immune response that extends beyond the correction of structural issues. After the procedure, neutrophils and macrophages are drawn to the site, and mediators initiate the acute-phase inflammatory cascade involving interleukin (IL)-6 and other proinflammatory signals [7,8]. Although this response is crucial for wound repair, excessive activation can increase pain, swelling, and complications, and uncontrolled reactive oxygen species (ROS)-related signaling can cause inflammatory damage [9]. In hernia surgery, postoperative pain remains significant even after successful repair, and modified mesh fixation can reduce chronic pain without eliminating inflammation-related issues [10]. Therefore, postoperative recovery balances beneficial repair inflammation against maladaptive immune activation, rather than being merely a mechanical outcome [7,8].

The inflammatory response after surgery is affected by the surgical approach. In autoplasty, suturing tissues under tension increases mechanical stress, hinders blood flow, and worsens nerve irritation, thereby amplifying inflammation and pain. Tension-free mesh repair aims to reduce strain on groin tissues; however, clinical benefits vary across studies. Research has shown that mesh repair reduces recurrence rates; however, postoperative pain and quality-of-life outcomes often match those for non-mesh repair [11].

The host's response to prosthetic repair depends on the mesh design and implantation. Studies have shown that lighter meshes may reduce inflammatory infiltration compared to heavier meshes [12]. However, lightweight meshes do not consistently reduce pain and may increase recurrence in laparoscopic-endoscopic cases [13]. Mesh porosity matters; small-pored polypropylene causes more chronic groin pain than large-pored mesh [14]. Surgical technique affects inflammation; laparoscopic-endoscopic Totally Extraperitoneal (TEP) repair has shown lower c-reactive protein (CRP) levels and analgesic needs than open repairs, suggesting that less invasive methods reduce the inflammatory response [15].

The relationship between surgery, inflammation, and recovery is biologically complex rather than mechanical. The ideal repair balances mechanical stability with the host response, preventing excessive inflammation, nociception, edema, and long-term pain syndromes from dysregulated immune signaling [16,17].

Recent international HerniaSurge guidelines have identified tension-free mesh repair as the preferred treatment for symptomatic inguinal hernia, due to reduced recurrence rates and chronic postoperative inguinal pain (CPIP) [18]. However, the biological mechanisms underlying these benefits remain unclear, particularly in settings where tissue-based repair is still used [19]. Postoperative recovery depends on both mechanical support and the host's inflammatory response to surgical trauma and prosthetics [20].

The advantage of tension-free repair extends beyond removing suture tension to creating a favorable mechanobiological environment. Tissue-based repair can increase local ischemia and nerve irritation, whereas tension-free methods minimize tissue damage and reduce analgesic needs. The foreign-body response varies with mesh type, fixation, and surgical approach [21,22].

Enhanced outcomes resulting from mesh repair are due to both mechanical and immunological factors: reduced tissue strain, nerve compression, and regulated inflammation. When dysregulated, these processes can lead to edema, persistent pain, and chronic complications [23].

Recent findings suggest that pain intensity after surgery, complication rates, and recovery patterns indicate the body's inflammatory response. Acute postoperative pain varies, and severe pain is associated with chronic postsurgical pain and worse outcomes [24, 25]. Studies have shown that pain patterns correlate with recovery progress in the first month after surgery, suggesting that early symptoms reflect broader recovery processes [26].

Clinically, procedures that minimize tissue damage and ischemia reduce the inflammatory burden. Laparoscopic techniques result in less pain, fewer paresthetic symptoms, and better outcomes compared to open repair for inguinal hernia repair, according to randomized evidence [27]. In older patients, laparoscopic repair shows fewer complications, less need for pain medication, and faster recovery than open surgery [28]. This supports that reducing mechanical stress and tissue damage decreases inflammatory signaling and improves recovery.

When inflammation is excessive, the biological response that aids tissue repair may cause swelling, delayed healing, seroma, hematoma, infection, and ongoing nociceptive signaling. This makes postoperative pain and complications valuable indicators of the balance between adaptive repair and maladaptive inflammation [24,26].

The interplay between surgical trauma and systemic immune activation influences endothelial function, oxidative stress pathways, and tissue remodeling. Surgery triggers innate immune and acute-phase pathways, with postoperative increases in interleukin-6 (IL-6), CRP, and other mediators indicating the body's response to tissue damage [29]. At the vascular level, inflammatory activation is associated with endothelial dysfunction and oxidative stress, disrupting microcirculatory homeostasis [30,31]. Monocyte–endothelial interactions regulate inflammatory recruitment and repair; however, ongoing activation can lead to vascular leakage and maladaptive remodeling [32].

The reparative phase is directed by immune-cell programs that determine healing resolution or fibrosis. Macrophages manage debris clearance, cytokine signaling, angiogenesis, and matrix remodeling, whereas improper polarization can lead to chronic inflammation and scarring [33–35]. Oxidative stress intersects these processes, as macrophage-derived reactive oxygen species affect fibrosis and tissue regeneration [33,36].

Yu et al. reported that cellular mechanical changes affect inflammation, tissue remodeling, and stress responses. Mechanical stimuli, such as fluid flow, microgravity, and strain, alter mechanosensitivity and calcium signaling, influencing inflammatory and reparative responses. Excessive tissue tension during hernia repair may enhance inflammation through mechanotransduction. The biological response to repair relies on anatomical reconstruction, tissue integrity, and stress response regulation [37].

These findings support including immunological endpoints in surgical outcome assessments, extending evaluation beyond anatomical success to assess inflammatory balance, endothelial integrity, oxidative burden, tissue remodeling, and risk of complications [31,34].

Comparative clinical research is needed to evaluate both traditional surgical outcomes and their immuno-inflammatory counterparts. This could clarify how tissue damage, prosthetic placement, and inflammation affect pain, complications, and recovery after inguinal hernia repair, beyond anatomical correction. Comparative studies have shown that repair techniques impact postoperative pain and inflammation: randomized trials have revealed lower CRP levels and less pain relief needed after laparoscopic TEP repair versus Bassini, Nyhus, and Lichtenstein, suggesting that less invasive methods reduce inflammation. Mesh-based repairs show better recurrence rates than non-mesh repairs, while postoperative pain and quality of life are often similar, highlighting the need to study biological mechanisms alongside clinical outcomes.

This study aims to compare autoplasty and tension-free alloplasty for primary inguinal hernias, focusing on clinical outcomes and immuno-inflammatory responses. This aligns with findings on mesh characteristics and fixation methods influencing host response, with lighter meshes linked to less inflammation and connective tissue, and suture fixation affecting pain. It also addresses the global need for cost-effective, biologically informative repair strategies in various settings, including resource-limited areas.

2. Methods

This single-center, prospective comparative observational study was conducted at the National Surgical Center in Bishkek, Kyrgyz Republic (2020–2025). The study aimed to compare the clinical outcomes of autoplasty versus tension-free alloplasty in patients with primary inguinal herniae and analyze the results from an immuno-inflammatory perspective. The study followed the Declaration of Helsinki principles (2013) with the local ethics committee of the National Surgical Center approval (Protocol No. 8, dated June 5, 2025). All participants provided written informed consent.

A total of 118 individuals diagnosed with primary inguinal hernias were included in this study. The selection criteria for participants were as follows: Inclusion criteria: individuals aged ≥ 18 years, those with a clinically confirmed primary inguinal hernia, candidates deemed suitable for elective surgery, and those who consented to participate. Exclusion criteria: patients with strangulated or acutely complicated inguinal hernias requiring emergency surgery, recurrent hernias, decompensated cardiovascular, respiratory, or endocrine disorders, active infection at the surgical site, clinically significant bowel ischemia, perforation, widespread peritonitis, severe systemic inflammatory conditions, and those who declined participation. The study population was predominantly male, which aligns with the epidemiological trends of inguinal hernias.

Participants were divided based on the surgical method: Group I (autoplasty, n = 50) underwent tissue-based repairs using Shouldice, Spasokukotsky, or variations, reconstructing the posterior wall of the inguinal canal with native tissues under tension. Group II (alloplasty, n = 68) underwent tension-free mesh repairs with a polypropylene prosthesis following the Lichtenstein technique. All surgeries were performed by skilled surgeons under stan-

dardized protocols using local infiltration or spinal anesthesia based on patient needs.

All patients received uniform perioperative care, including preventive antibiotics, pain management per guidelines, and early movement as suitable. Postoperative evaluations occurred on days 1, 2, and 5, reviewing clinical parameters and complications.

Primary clinical outcomes assessed were surgery duration (minutes) from the initial incision to the final stitch, postoperative pain using the verbal descriptor scale (VDS) on days 1, 2, and 5, complications per Clavien-Dindo classification (2004), and functional recovery indicators: mobilization time, analgesic duration, and hospital stay length.

Systemic biomarkers such as CRP, neutrophil-to-lymphocyte ratio (NLR), IL-6, tumor necrosis factor-alpha (TNF-α), and lymphocyte count were assessed to evaluate immune-inflammatory responses. Blood samples were collected from the peripheral veins before surgery and on the first and third days post-surgery using aseptic techniques. Each participant provided approximately 5 mL of blood using sterile vacutainer systems. An automated hematology analyzer was used to determine the complete blood count, including the NLR. Serum CRP levels were measured using immunoturbidimetric assays, and IL-6 and TNF-α concentrations were assessed using ELISA kits according to the manufacturer's instructions. All tests adhered to the standardized quality control procedures of the institutional central diagnostic laboratory. The biomarkers were selected based on their roles in the acute-phase inflammatory response, surgical stress physiology, immune activation, and postoperative recovery. Elevated CRP, IL-6, TNF-α, and NLR levels indicate increased systemic inflammatory activation and tissue damage after surgery.

Data analysis was performed using IBM SPSS Statistics for Windows, Version 23.0, and Microsoft Excel 2019. Continuous variables are shown as mean ± standard deviation, while categorical variables are shown as frequencies and percentages. The Student's *t*-test was used for normally distributed data, the Mann–Whitney U test for non-parametric data, and Pearson's χ^2 test for categorical variables. Pearson 's correlation coefficients (r) were evaluated to determine the relationships between operative parameters, clinical outcomes, and immuno-inflammatory indicators. A p-value < 0.05 was deemed significant.

All procedures followed ethical guidelines for research involving humans. Patient confidentiality was maintained through data anonymization prior to analysis.

3. Results

All 118 patients underwent surgery without any significant complications during the procedure. The mean age was 46 ± 12.1 years, with men comprising a substantial majority (93%) (**Table 1**).

Table 1. Baseline characteristics of the study population.

	Autoplasty (n = 50)	Alloplasty (n = 68)	*p*-Value
Age (years, mean ± SD)	46.8 ± 12.5	45.3 ± 11.9	>0.05
Male, n (%)	46 (92.0%)	64 (94.1%)	>0.05
Female, n (%)	4 (8.0%)	4 (5.9%)	>0.05
Right-sided hernia, n (%)	36 (72.0%)	49 (72.1%)	>0.05
Left-sided hernia, n (%)	14 (28.0%)	19 (27.9%)	>0.05
Duration of hernia (months)	14.2 ± 5.6	13.8 ± 6.1	>0.05

Note: Data presented as n (%), n = No. of patients, % = Percentage of patients, Mean ± Standard Deviation, p-value < 0.05.

During the analysis, a notable difference in procedure duration was identified: autografting took 59.6 ± 2.6 min, whereas alloplasty required 48.4 ± 1.5 min (p < 0.05) (**Table 2**).

Table 2. Operative and early postoperative clinical outcomes.

	Autoplasty (n = 50)	Alloplasty (n = 68)	*p*-Value
Operative time (min)	59.6 ± 2.6	48.4 ± 1.5	<0.05
Pain (Day 1, VDS)	5.7 ± 0.6	4.5 ± 0.5	<0.05
Pain (Day 2, VDS)	2.4 ± 0.3	1.1 ± 0.2	<0.05
Pain (Day 5, VDS)	1.2 ± 0.2	0.5 ± 0.1	<0.05
Time to mobilization (hours)	21.4 ± 3.6	7.2 ± 2.1	<0.01
Duration of analgesic use (days)	2.7 ± 0.6	1.2 ± 0.4	<0.05
Hospital stay (days)	5.1 ± 1.2	3.6 ± 0.9	<0.05

Note: Data presented as Mean ± Standard Deviation, p-value < 0.05.

From an immuno-inflammatory perspective, shorter surgery time indicates less surgical trauma and reduced activation of inflammatory pathways, including lower release of damage-associated molecular patterns and neutrophil activation. Conversely, extended tissue handling in autoplasty can lead to prolonged inflammatory signaling and stress responses from ischemia.

Pain was the primary differentiator between the groups. On the first day after surgery, the pain levels were 5.7 ± 0.6 points for autografting and 4.5 ± 0.5 for alloplasty ($p < 0.05$). By the second day, these values dropped to 2.4 ± 0.3 and 1.1 ± 0.2, respectively. By the fifth day, patients still experienced discomfort following autografting (1.2 ± 0.2), whereas pain had nearly vanished after alloplasty (0.5 ± 0.1) (**Table 2**).

This trend is not accidental. In autografting, tissue tension, nerve branch compression, and aponeurotic ischemia contribute to the nociceptive aspect of pain. Alloplasty eliminates tension: the polypropylene mesh covers the defect without altering anatomy, preserving nerve function and blood circulation. This pain reduction is supported by studies by Aiolfi et al. [38] and Chu et al. [39], who showed that tension-free techniques can lower the risk of chronic postoperative pain by 35–40%.

The physiological advantages of the method were clearly demonstrated by objective data: with autografting, the average duration of analgesic use was 2.7 ± 0.6 days, compared to 1.2 ± 0.4 days with alloplasty ($p < 0.05$) (**Table 3**). Additionally, 82% of patients did not require narcotic analgesics following alloplasty. This indicates a significant difference not only in pain threshold but also in the extent of injury: tension on the aponeurosis during autografting causes continuous ischemic stimulation of pain receptors, necessitating pharmacological management.

Table 3. Postoperative complications (Clavien-Dindo classification).

	Autoplasty (n = 50)	Alloplasty (n = 68)	*p*-Value
Any complication (%)	28.8%	8.4%	<0.05
Seroma	8 (16.0%)	3 (4.4%)	<0.05
Infiltrate	5 (10.0%)	4 (5.9%)	>0.05
Hematoma	3 (6.0%)	3 (4.4%)	>0.05
Wound supparation	4 (8.0%)	-	<0.05
Persistent pain	2.7 ± 0.6	1.2 ± 0.4	<0.05
Clavien-Dindo Grade I–II	Majority	Majority	-
Clavien-Dindo Grade III	Present	None	-

Note: Data presented as n (%), n = No. of patients, % = Percentage of patients, Mean ± Standard Deviation, *p*-value < 0.05.

Complications occurred in 28.8% of patients undergoing autografting, compared to 8.4% with allografting. According to the Clavien-Dindo classification, most complications were categorized as grade I (hematoma, seroma), with fewer cases classified as grade II (infiltrate, need for antibiotics). Grade III cases (wound suppuration) were only observed with autografting.

These complications may indicate differences in the strength of the local inflammatory response and tissue repair processes. Seromas and infiltrates indicate heightened vascular permeability and inflammatory fluid leakage, whereas wound suppuration points to compromised immune control and secondary infection risk. The lower complication rates in the alloplasty group suggest a more regulated inflammatory response.

These distinctions underscore the impact of tissue tension as a factor in secondary ischemia and exudation. In the alloplasty group, seromas and hematomas were minimal and did not require surgical intervention. Thus, the 3.4-fold decrease in complications with alloplasty is not coincidental but a result of its biomechanical benefits.

In cases of autoplasty, the occurrence rates were as follows: seroma, 16.0%; infiltrate, 10.0%; suppuration, 8.0%; hematoma, 6.0%; and pain, 22.0%. For alloplasty, the rates were 4.4% for seroma, 5.9% for infiltrate, and 4.4% for hematoma (**Table 3**).

The integrated clinical and immune-inflammatory findings indicate possible biological differences between autoplasty and alloplasty (**Table 4**). Mechanobiology and surgical stress suggest that longer surgery times and increased tissue manipulation during autoplasty may lead to heightened inflammatory responses and postoperative immune activation. Tissue tension is crucial; autoplasty causes mechanical stress, ischemia, and inflammation, whereas tension-free alloplasty reduces these. Higher seroma formation in autoplasty indicates increased vascular permeability. Wound infections in autoplasty suggest impaired immune regulation. Increased analgesic needs reflect ongoing inflammation, whereas decreased requirements in alloplasty suggest effective suppression. Delayed mobilization in autoplasty aligns with greater systemic inflammation, and early mobilization in alloplasty indicates

quicker resolution. Extended hospital stays in autoplasty result from prolonged inflammation, whereas shorter stays in alloplasty show reduced activation. These findings demonstrate the clinical superiority of alloplasty in reducing surgical trauma-related inflammation.

Table 4. Immuno-inflammatory interpretation of clinical outcomes.

	Autoplasty	Alloplasty	Immuno-Inflammatory Interpretation
Operative time	Longer	Shorter	Higher DAMP release and immune activation (Increased duration)
Pain intensity	Higher	Lower	Elevated cytokine-mediated nociception (IL-6, TNF-α)
Tissue tension	High	Minimal	Tension → ischemia → amplified inflammatory cascade
Seroma formation	Higher	Lower	Increased vascular permeability and exudation
Wound infection	Present	Absent	Local immune dysregulation and impaired healing
Analgesic requirement	Higher	Lower	Reflects sustained inflammatory signaling
Mobilization	Delayed	Early	Reduced systemic inflammation enables faster recovery
Hospital stay	Longer	Shorter	Prolonged inflammatory phase delays discharge

Note: DAMPs = Damage-Associated Molecular Patterns, IL-6 = Interleukin-6, TNF-α = Tumor necrosis factor-alpha.

Patients who had alloplasty were active within 4 to 6 h post-surgery, while autografting patients began standing on the second day. After mesh implantation, patients required minimal pain relief, with only one initial dose of a narcotic. Patients walked independently after 7.2 ± 2.1 h, vs. 21.4 ± 3.6 h for autografting ($p < 0.01$) (**Table 2**). These findings show that tension-free allografting enables natural progression, reduces pain and wound complications, and promotes early functional recovery, which is particularly important for working-age patients.

Early movement and reduced pain relief needs indicate lower systemic inflammation and pain sensitization from cytokines. This suggests that tension-free repair enables a better immune response and quicker recovery.

The variation in recovery times is reflected in hospital stays: 5.1 ± 1.2 days after autografting compared to 3.6 ± 0.9 days after alloplasty ($p < 0.05$) (**Table 2**). Faster recovery and reduced pain enabled discharge 1.5 days earlier. This represents both reduced hospital days and a measure of intervention effectiveness. Shorter hospital stays indicate faster recovery and fewer postoperative complications. From a systems perspective, this suggests more efficient care and lower resource use.

Correlation analysis indicated a direct link between the length of surgery and the intensity of pain on the first day ($r = 0.64$; $p < 0.01$), as well as a moderate association between surgical duration and the rate of complications ($r = 0.48$; $p < 0.05$) (**Table 5**). An inverse relationship was observed between the type of surgery (autograft versus alloplasty) and the length of hospital stay ($r = -0.57$; $p < 0.01$). These findings suggest that less surgical trauma and shorter anesthesia times lead to quicker pain relief and a reduced risk of complications.

Table 5. Correlation analysis of clinical and immune-inflammatory parameters.

Variables	Correlation Coefficient (r)	*p*-Value	Interpretation
Operative time vs. Pain (Day 1)	0.64	<0.01	Strong positive correlation (higher trauma → increased inflammation)
Operative time vs. Complications	0.48	<0.05	Moderate correlation (surgical stress → immune dysregulation)
Alloplasty vs. Hospital stay	−0.57	<0.01	Negative correlation (reduced inflammation → faster recovery)

Note: *p*-value < 0.05.

The intensity of post-surgery pain may partly indicate inflammatory mediator activity and tissue stress responses associated with cytokine signaling. Greater pain in the autoplasty group suggests increased inflammation due to tissue tension and reduced blood flow.

Therefore, postsurgical pain serves not only as a measure of subjective experience but also as a direct indicator of the operation's mechanics whether stressful or physiological.

Laboratory assessments of biomarkers showed that the autoplasty group had a higher inflammatory response than the alloplasty group (**Table 6**). Patients who underwent autoplasty showed higher CRP levels (32.5 ± 6.8 mg/L) than alloplasty patients (21.3 ± 5.4 mg/L; $p < 0.05$) on day 1, indicating increased tissue damage. This difference persisted on day 3, suggesting slower inflammation resolution. The NLR was higher in the autoplasty group on day 1 (5.8 ± 1.2 vs. 3.9 ± 0.9; $p < 0.05$) and day 3 (3.6 ± 0.8 vs. 2.4 ± 0.6; $p < 0.05$), indicating heightened immune response. IL-6 levels were higher in patients who underwent autoplasty (48.7 ± 10.2 pg/mL vs. 29.4 ± 8.6 pg/mL; $p < 0.01$), reflecting increased surgical stress. Elevated levels continued on day 3. TNF-α levels were higher in the

autoplasty group (26.5 ± 7.3 pg/mL vs. 17.2 ± 5.8 pg/mL; $p < 0.05$), supporting an increased pro-inflammatory response. Alloplasty patients maintained stable lymphocyte counts (1.6 ± 0.4 × 10^9/L vs. 1.2 ± 0.3 × 10^9/L; $p < 0.05$), whereas autoplasty patients showed relative lymphopenia. The CRP/NLR ratio was higher in autoplasty (5.6 ± 1.4 vs. 3.2 ± 1.1; $p < 0.05$), indicating a greater inflammatory burden. These results demonstrate that autoplasty causes more intense inflammation, whereas alloplasty leads to reduced inflammation and faster resolution.

Table 6. Immuno-inflammatory marker profile in Autoplasty vs. Alloplasty.

	Time Point	Autoplasty (n = 50)	Alloplasty (n = 68)	*p*-Value	Immunological Interpretation
CRP (mg/L)	Day 1	32.5 ± 6.8	21.3 ± 5.4	<0.05	Higher acute-phase response due to tissue injury and ischemia
	Day 3	18.2 ± 4.5	10.6 ± 3.2	<0.05	Sustained inflammation in autoplasty
NLR	Day 1	5.8 ± 1.2	3.9 ± 0.9	<0.05	Increased neutrophil-driven inflammation in autoplasty
	Day 3	3.6 ± 0.8	2.4 ± 0.6	<0.05	Slower resolution of systemic inflammation
IL-6 (pg/Ml)	Day 1	48.7 ± 10.2	29.4 ± 8.6	<0.01	Elevated cytokine surge reflecting surgical stress
	Day 3	22.1 ± 6.4	12.8 ± 4.1	<0.01	Persistent inflammatory signalling in autoplasty
TNF-α	Day 1	26.5 ± 7.3	17.2 ± 5.8	<0.05	Amplified pro-inflammatory cytokine response
Lymphocyte count (×10^9/L)	Day 1	1.2 ± 0.3	1.6 ± 0.4	<0.05	Relative lymphopenia due to stress-induced immune suppression
CRP/NLR ratio	Day 1	5.6 ± 1.4	3.2 ± 1.1	<0.05	Integrated marker of systemic inflammation severity

Note: Data presented as Mean ± Standard Deviation, *p*-value < 0.05. CRP = C-reactive protein, NLR = Neutrophil-to-lymphocyte ratio, IL-6 = Interleukin-6, TNF-α = Tumor necrosis factor-alpha.

4. Discussion

This study focused on clinical outcomes and systemic inflammatory biomarkers, rather than mechanistic endpoints. Thus, interpretations of macrophage activation, endothelial dysfunction, oxidative stress, cytokine-driven nociception, and Damage-Associated Molecular Pattern signaling are plausible hypotheses based on the existing literature, not the mechanisms shown in this study.

The most prevalent hernia observed was an oblique right-sided inguinal hernia, which is in accordance with the epidemiology of inguinal hernias and the predominance of right-sided cases in clinical studies [40]. The patient groups, divided by surgery type, were comparable in terms of age, sex, body mass index, American Society of Anesthesiologists score, and initial clinical characteristics, with no significant differences between the groups ($p > 0.05$). This similarity reduces selection bias and enhances the internal validity of the comparison [41]. This baseline equivalence indicates that variations in postoperative pain, complications, recurrence, return to activity, and recovery outcomes are more likely due to the repair technique rather than preoperative patient characteristics [41,42]. Thus, the study design enables a more reliable evaluation of the effectiveness and safety of surgical methods.

Hernia incarceration affects postoperative inflammatory responses, regardless of the type of surgery. Incarcerated hernias increase tissue pressure, venous congestion, ischemia, disrupted microcirculation, and potential bowel issues, thereby heightening systemic inflammation and cytokine release pre-surgery. Consequently, inflammatory biomarkers and recovery patterns in incarcerated or strangulated hernias differ from those in straightforward elective primary inguinal hernia repairs. This study focused on elective primary hernia cases without severe acute ischemic complications; therefore, the inflammatory profiles should be understood in this context.

A 11-min reduction is clinically significant because it can lower anesthesia exposure, reduce tissue handling time, and enhance operating room efficiency. In inguinal hernia repair, quicker and standardized procedures have been linked to better perioperative outcomes, including decreased surgical time and comparable postoperative results. Mesh-based methods, such as the Lichtenstein repair, are quicker than tissue-based repairs while maintaining safety and recurrence rates [43]. Self-gripping or simplified mesh techniques can reduce surgical time without increasing complications [41,44]. Clear anatomical landmarks make anterior inguinal hernia repair more straightforward and reduce surgical time while maintaining outcomes [45]. Gutlic et al. observed that mesh hernioplasty enhanced procedural consistency and reduced surgical time, even for less experienced surgeons, highlighting the importance of standardizing techniques [46]. Alloplasty is technically simpler and more reproducible because it

maintains anatomical orientation, uses identifiable landmarks, and reduces technical errors, leading to a more predictable repair [45,47].

The use of polypropylene endoprostheses for tension-free hernioplasty offers benefits over traditional tissue-based methods for reconstructing the inguinal canal. The reduced operative time of the procedure demonstrates its technical ease, involving less tissue dissection and eliminating aponeurotic tension and extensive suturing, contributing to standardization in surgical practice [48,49].

The physiological basis for decreased postoperative pain and complications is clear. Unlike autoplastic repair, tension-free mesh techniques reduce tissue traction and nerve irritation, maintain microcirculation, and lessen ischemic stress in the surgical area [50, 51]. These factors explain why patients report less discomfort, quicker mobilization, and faster recovery.

Our results align with studies showing that mesh-based methods, such as Lichtenstein and laparoscopic TAPP/TEP repairs, deliver reliable outcomes with reduced pain and quicker rehabilitation compared to traditional tissue repair [48, 52]. Current HerniaSurge guidelines endorse mesh repair as the standard for primary inguinal hernia in adults due to its low recurrence rate, favorable pain profile, and reproducibility in surgical practice [53].

The threefold reduction in complications in our study indicates the effectiveness of alloplasty in a multidisciplinary hospital environment. Shorter hospital stays and quicker returns to activities reflect surgical efficiency and a superior physiological profile. The data support that minimal tension results in less trauma, improved healing, and faster rehabilitation, with corresponding socioeconomic benefits.

The distinction between the methods was statistically significant and clinically meaningful. For surgeons, this indicates reliable results; for patients, it means reduced pain and quicker discharge; and for the healthcare system, it suggests decreased costs and improved resource use. This interpretation is supported by evidence showing that Lichtenstein repair is linked to shorter surgery duration, less need for pain relief, quicker return to work, and lower recurrence rates compared to Shouldice repair for primary unilateral inguinal hernia [43].

Nevertheless, the non-randomized design might have led to selection bias, and the lack of long-term follow-up limits the evaluation of recurrence and CPIP. This limitation is crucial because long-term outcomes are not captured by early postoperative measures, and studies have shown that CPIP and recurrence require extended follow-up for accurate assessment [54]. Variations in surgical technique may also have influenced surgery duration and dissection extent, aligning with evidence that surgeon and patient characteristics can significantly impact Lichtenstein outcomes [55].

Despite similar baseline characteristics between the groups, unmeasured confounding factors could impact outcomes. The single-center study might limit the applicability of the results to institutions with different demographics and protocols. Although the sample size detected significant primary outcome differences, it remains small for subgroup analyses. Patients with compromised immune systems may have altered inflammatory and healing responses after hernia repair. Factors such as diabetes, corticosteroid use, cancer, immunosuppressive treatments, poor nutrition, systemic illnesses, and chronic inflammatory conditions can affect cytokine regulation, wound healing, infection risk, and recovery. Although individuals with severe systemic diseases were excluded, baseline variations in immune status may have still impacted inflammatory biomarkers and outcomes. Variations in surgical techniques and surgeon experience were not fully standardized, which could influence outcomes. The study did not assess patient-reported quality-of-life measures beyond early postoperative parameters. This study did not assess mechanistic endpoints, such as macrophage polarization, oxidative stress markers, endothelial dysfunction, reactive oxygen species production, or damage-associated molecular pattern signaling. Therefore, mechanistic interpretations should be exploratory and hypothesis-generating. Despite these limitations, this study provides valuable insights into the clinical and immune-inflammatory differences between autoplasty and alloplasty, forming a basis for future randomized studies.

The data endorse alloplasty as the preferred approach for primary inguinal hernia repair and provide a foundation for future prospective studies on long-term outcomes and quality of life.

Results support that inguinal hernia repair effectiveness depends on anatomical correction and interplay between surgical mechanics and host immune response. Differences between autoplasty and alloplasty highlight tissue tension, ischemia, and surgical trauma as key factors influencing postoperative inflammation and outcomes. The study found a strong correlation between surgical duration and early postoperative pain ($r = 0.64$), demonstrat-

ing how surgical trauma triggers an inflammatory cascade. Longer surgical procedures may increase tissue strain and trigger inflammatory responses associated with heightened cytokine-driven pain signaling and postoperative discomfort. Higher CRP, NLR, IL-6, and TNF-α levels in the autoplasty group indicate heightened systemic inflammatory response. These biomarkers show immune activation, and their persistence suggests delayed inflammation resolution, accounting for higher complications and extended recovery. Tissue tension determines inflammatory burden. In autoplasty, tissue tension can cause localized ischemic stress, changes in microcirculatory dynamics, and heightened nociceptive stimulation, possibly intensifying postoperative inflammatory reactions. Tension-free alloplasty reduces strain and maintains tissue perfusion, fostering controlled healing. While mesh implantation induces controlled inflammatory reaction for integration, this response appears more regulated than inflammation from excessive tissue tension. This highlights mesh characteristics' importance in influencing host response and outcomes.

Hagan et al. demonstrated that compromised cellular integrity reduces mechanotransduction and increases cellular vulnerability to stress. This is relevant to hernia repair, where tissue approximation and strain can cause tissue damage, disrupt signaling, and cause inflammation. Tension-free methods may better maintain stability and reduce cellular stress [56].

The findings of this study significantly impact surgical choices for inguinal hernia repair. Tension-free alloplasty reduces surgery time, postoperative discomfort, complications, and hospital stays, supporting its standard use for primary inguinal hernia repair. It decreases surgical stress and immuno-inflammatory activation, which is crucial for recovery, making it preferable for high-risk patients. Lower pain scores in the alloplasty group suggest better cytokine-mediated nociception control, highlighting tissue tension minimization. The use of markers, such as CRP, NLR, IL-6, and TNF-α, in assessments helps evaluate outcomes. Higher marker levels in autoplasty patients indicate more inflammation. Lower complication rates in alloplasty stress the need for a controlled inflammatory response. Early mobilization and shorter hospital stays favor minimally invasive methods. In resource-limited settings where autoplasty is chosen for cost, reducing tissue tension is essential. These findings advocate for broader access to mesh technology. In conclusion, inguinal hernia repair should be viewed as a mechanobiological process, in which reducing immune activation ensures optimal outcomes.

Tuladhar et al.'s finding highlights membrane repair's role in preserving cellular integrity under mechanical stress. Disrupted repair is correlated with increased damage, inflammation, and delayed recovery. These findings explain how surgical tension and tissue manipulation during autoplasty can increase inflammation and delay the recovery process. Conversely, tension-free alloplasty may reduce cellular disruption and promote tissue healing [57].

Interpreting systemic inflammatory biomarkers such as CRP, NLR, IL-6, and TNF-α requires a comprehensive framework, including structural, functional, and clinical recovery outcomes. Recent research has shown that isolated biomarker evaluation may not reflect biological responses unless analyzed with functional and structural tissue changes. Multimodal strategies combining circulating markers with imaging, histology, and functional outcomes enhance the understanding of tissue injury and recovery under inflammation and stress. This study integrated factors such as postoperative pain, mobilization, complications, and hospital stay with systemic biomarkers to better understand recovery and inflammation after hernia repair [58,59].

Drawing from clinical and immuno-inflammatory insights, several recommendations can enhance surgical outcomes. Tension-free mesh repair is recommended for adult primary inguinal hernias, offering a shorter operative time, less pain, fewer complications, and quicker recovery. The reduction in inflammatory markers confirms its benefits. Clinicians should monitor biomarkers, such as CRP, NLR, IL-6, and TNF-α, in high-risk patients as indicators of the inflammatory response. Surgeons should minimize tissue handling, ischemia, and nerve compression. When autoplasty is necessary, careful technique should limit inflammation. Mesh selection and fixation should minimize foreign-body reactions and chronic inflammation. Enhanced recovery after surgery protocols are advised, including early mobilization and optimized pain management. Training programs should emphasize standardized mesh-based techniques to reduce complications. Healthcare systems in low-resource settings should prioritize access to affordable mesh materials to enable the adoption of favorable techniques. Future research should focus on large-scale trials with long-term follow-up, including immuno-inflammatory markers. Validated tools for pain, quality of life, and functional recovery should complement clinical endpoints. Hernia repair should be viewed as a mechanobiological intervention, balancing structural repair with the immune response. These recommendations support precision surgery, integrating technical excellence with biological insight to enhance outcomes.

In addition to reducing inflammation, postoperative recovery depends on tissue repair, neuromuscular health, and systemic adaptation. Studies have shown that adjusting inflammatory and cellular pathways helps preserve tissue and muscle function by maintaining neuromuscular junctions and minimizing tissue dysfunction. These findings suggest that hernia repair recovery is influenced by local inflammation and systemic responses affecting tissue remodeling and mobility restoration. The earlier mobilization and reduced pain relief requirement after alloplasty may indicate beneficial interactions between surgical techniques, inflammation control, and recovery processes [37].

5. Conclusions

This study shows tension-free alloplasty yields better clinical results than tissue-based autoplasty for primary inguinal hernia. Alloplasty is associated with shorter surgery times, less postoperative discomfort, fewer complications, quicker mobilization, and shorter hospital stays. These benefits are associated with a favorable immunoinflammatory profile, marked by reduced CRP, NLR, IL-6, and TNF-α levels, and quicker resolution of systemic inflammation. Autoplasty, due to tissue tension and ischemia, is associated with heightened inflammation, increased nociceptive signaling, and slower recovery. The relationships between surgery duration, pain, and complications emphasize the impact of surgical stress and immune dysregulation on outcomes. These findings support that inguinal hernia repair is a mechanobiological process, in which optimal results rely on minimizing tissue damage while encouraging healing. Tension-free mesh repair achieves this by reducing strain and modulating the inflammatory response. Overall, alloplasty should be the preferred method for primary inguinal hernia repair; future studies should focus on long-term outcomes, biomarker-driven stratification, and technique refinement to enhance recovery.

Author Contributions

Conceptualization, K.M.; methodology, Z.A.; software, T.T.; validation, U.A.; formal analysis, T.T.; investigation, K.M., Z.A., U.A., B.N., O.D., and S.K.; data curation, S.K.; writing—original draft preparation, B.N., O.D., and T.T.; writing—review and editing, T.T. All authors have read and agreed to the published version of the manuscript.

Funding

This work received no external funding.

Institutional Review Board Statement

The study followed the Declaration of Helsinki principles (2013) with the local ethics committee of the National Surgical Center approved this study (Protocol No. 8, dated June 5, 2025).

Informed Consent Statement

Informed consent was obtained from all subjects involved in the study.

Data Availability Statement

Data are available from the corresponding author upon reasonable request.

Conflicts of Interest

The authors declare no conflict of interest.

AI Use Statement

While drafting this manuscript, the authors used Paperpal solely to refine the language, grammar, spelling, and scientific readability. No AI tools were used for data generation, statistical analyses, result interpretation, content fabrication, or conclusion formulation. All scientific interpretations, clinical analyses, and final revisions were independently reviewed and approved by the authors, who assumed full responsibility for the manuscript's integrity, originality, and accuracy.

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