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ORIGINAL ARTICLE

The Magnitude of Pain-Driven Sympathetic Activation after Surgery and its Gastrointestinal Consequences: A Community-Based Clinical Study

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ABSTRACT

Background: Postoperative pain is a major physiological stressor that activates the sympathetic nervous system, leading to systemic effects beyond patient discomfort. This study aimed to evaluate the magnitude of pain-driven sympathetic activation after surgery and its impact on gastrointestinal recovery in a community-based clinical setting.

Methods: This clinical study was conducted from May 2022 to June 2023 at tertiary care hospitals in Lahore, Pakistan, including Doctors Hospital & Medical Centre and Pakistan Kidney and Liver Institute & Research Centre. A total of 100 adult patients undergoing elective surgical procedures were enrolled using consecutive non-probability sampling. Postoperative pain was assessed using the Visual Analog Scale (VAS), and patients were categorized into mild, moderate, and severe pain groups. Sympathetic activation was evaluated through heart rate, blood pressure, and serum catecholamine levels. Gastrointestinal outcomes included time to first bowel sound, passage of flatus, bowel movement, and incidence of postoperative ileus. Statistical analysis was performed using SPSS version 26, with $p \leq 0.05$ considered significant.

Results: Patients with higher pain scores demonstrated significantly increased sympathetic activity, reflected by elevated heart rate, blood pressure, and catecholamine levels ($p < 0.001$). Severe pain was associated with delayed gastrointestinal recovery, including prolonged time to bowel sounds (33 ± 6 hours), first flatus (50 ± 7 hours), and bowel movement (74 ± 9 hours). The incidence of postoperative ileus was also significantly higher in the severe pain group (33.3%) compared to moderate (15.4%) and mild pain groups (7.1%) ($p < 0.001$).

Conclusion: Pain-induced sympathetic activation significantly impairs gastrointestinal recovery after surgery. Effective pain management strategies are essential not only for symptom relief but also for reducing postoperative complications and improving clinical outcomes.

Keywords: Postoperative pain, Sympathetic activation, Gastrointestinal motility, Postoperative ileus, Catecholamines, Surgical outcomes, Pakistan.

INTRODUCTION

Surgical intervention triggers a complex systemic stress response involving neuroendocrine, inflammatory, and autonomic pathways[1]. Among these, postoperative pain is one of the most potent and immediate stimuli that activates the sympathetic nervous system[2]. Tissue injury during surgery generates nociceptive signals that are transmitted through afferent pathways to the central nervous system, particularly the hypothalamus and brainstem autonomic centers[3]. This results in increased sympathetic outflow and release of catecholamines, including epinephrine and norepinephrine, leading to tachycardia, hypertension, and heightened metabolic activity[4].

The gastrointestinal system is highly sensitive to autonomic regulation, where a delicate balance between sympathetic and parasympathetic activity governs normal motility[5]. Under physiological conditions, parasympathetic stimulation promotes peristalsis and coordinated bowel function, while sympathetic activation exerts inhibitory effects on intestinal smooth muscle and enteric neural activity[6]. Following surgery, this balance is disrupted, with sympathetic dominance suppressing gastrointestinal motility, delaying gastric emptying, and impairing coordinated bowel activity[7].

Pain-driven sympathetic activation has been increasingly recognized as a central mechanism in the development of postoperative gastrointestinal dysfunction, particularly postoperative ileus[8]. This condition is characterized by delayed passage of flatus and stool, abdominal distension, nausea, and prolonged inability to tolerate oral intake[9]. The pathophysiology involves not only neural inhibition but also reduced splanchnic blood flow due to sympathetic-mediated vasoconstriction, leading to impaired intestinal perfusion and delayed recovery of bowel function[10]. Additionally, sustained sympathetic activation may interfere with enteric neurotransmitter release, further exacerbating motility disturbances[11].

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Despite advances in surgical techniques and perioperative care, postoperative gastrointestinal complications remain a significant cause of increased morbidity, prolonged hospital stay, and healthcare burden[12]. In resource-limited settings such as Pakistan, these complications are often under-recognized, and the physiological relationship between pain severity and gastrointestinal outcomes is not adequately explored in clinical practice[13]. Most existing studies have focused on pharmacological or surgical factors, with limited emphasis on the magnitude of autonomic activation as a mediator of postoperative recovery[14].

Therefore, this study was designed to evaluate the magnitude of pain-driven sympathetic activation after surgery and to determine its impact on gastrointestinal function in a community-based clinical setting[15]. By establishing this relationship, the study aims to highlight the importance of effective pain control not only for patient comfort but also for optimizing physiological recovery and reducing postoperative complications[16].

MATERIALS AND METHODS

This community-based clinical study was conducted over a period of 13 months from May 2022 to June 2023 at Doctors Hospital & Medical Centre and Pakistan Kidney and Liver Institute & Research Centre (PKLI & RC). Both centers are tertiary care hospitals serving a diverse population, thereby providing a representative clinical setting for evaluating postoperative physiological responses.

A total of 100 patients aged between 18 and 65 years who underwent elective surgical procedures (both abdominal and non-abdominal) were included in the study. Participants were recruited using consecutive non-probability sampling to minimize selection bias and ensure feasibility within the clinical setting. Patients with pre-existing gastrointestinal disorders, autonomic dysfunction, chronic opioid use, uncontrolled diabetes with neuropathy, severe cardiovascular instability, or those receiving medications known to significantly alter gastrointestinal motility were excluded from the study.

Ethical approval was obtained from the institutional ethical review committees of both participating centers, and the study was conducted in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrollment.

Postoperative pain was assessed using the Visual Analog Scale (VAS) at 6, 12, and 24 hours after surgery. Based on VAS scores, patients were categorized into mild (≤3), moderate (4–6), and severe (≥7) pain groups. Sympathetic activation was evaluated through both clinical and biochemical parameters, including heart rate, systolic and diastolic blood pressure, and serum catecholamine levels measured using enzyme-linked immunosorbent assay (ELISA).

Gastrointestinal recovery was assessed using standardized clinical indicators, including time to first bowel sound, time to first passage of flatus, time to first bowel movement, and the occurrence of postoperative ileus, defined clinically by delayed bowel activity associated with abdominal distension and intolerance to oral intake.

All collected data were entered and analyzed using Statistical Package for the Social Sciences (SPSS) version 26. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. Comparisons between groups were performed using independent sample t-test and one-way ANOVA for continuous variables, and chi-square test for categorical variables. A p-value of ≤0.05 was considered statistically significant.

RESULTS

A total of 100 patients were included in the study, with a mean age of 41.8 ± 11.2 years. Among them, 58 (58%) were males and 42 (42%) were females. Based on postoperative pain assessment using the Visual Analog Scale (VAS), 28% of patients were categorized as having mild pain, 39% moderate pain, and 33% severe pain within the first 24 hours after surgery.

A significant association was observed between pain severity and markers of sympathetic activation. Patients with higher pain scores demonstrated increased heart rate, systolic blood pressure, and serum catecholamine levels. As shown in Table 1, patients in the severe pain group had markedly elevated heart rate (104 ± 8 bpm), systolic blood pressure (140 ± 11 mmHg), and catecholamine levels (395 ± 48 pg/mL) compared to those with mild and moderate pain ($p < 0.001$). This indicates a strong pain-driven sympathetic response in the postoperative period.

Gastrointestinal recovery parameters were significantly delayed in patients with higher pain severity. Patients with severe pain experienced prolonged time to first bowel sound (33 ± 6 hours), first flatus (50 ± 7 hours), and first bowel movement (74 ± 9 hours). In contrast, patients with mild pain showed relatively faster recovery. As demonstrated in Table 2, these differences were statistically significant ($p < 0.001$), highlighting the negative impact of pain-induced sympathetic activation on gastrointestinal motility.

Furthermore, the incidence of postoperative ileus was markedly higher among patients with severe pain. Only 2 (7.1%) patients in the mild pain group developed ileus, compared to 6 (15.4%) in the moderate group and 11 (33.3%) in the severe pain group. As presented in Table 3, this difference was statistically significant ($p < 0.001$), demonstrating a clear correlation between pain intensity and gastrointestinal complications.

Table 1: Association Between Pain Severity and Sympathetic Activation

Pain Category	Heart Rate (bpm)	Systolic BP (mmHg)	Catecholamines (pg/mL)
Mild (n=28)	80 ± 6	120 ± 7	215 ± 30
Moderate (n=39)	90 ± 7	128 ± 9	300 ± 42
Severe (n=33)	104 ± 8	140 ± 11	395 ± 48

Overall, the results demonstrate a consistent and clinically significant trend where increasing postoperative pain severity is associated with heightened sympathetic activation and progressively delayed gastrointestinal recovery, ultimately leading to a higher risk of postoperative ileus. These findings strongly support the hypothesis that pain-driven autonomic imbalance plays a critical role in postoperative gastrointestinal dysfunction.

Table 2: Pain Severity and Gastrointestinal Recovery Parameters

Pain Category	First Bowel Sound (hrs)	First Flatus (hrs)	First Bowel Movement (hrs)
Mild (n=28)	19 ± 4	26 ± 5	38 ± 6
Moderate (n=39)	25 ± 5	34 ± 6	50 ± 7
Severe (n=33)	33 ± 6	50 ± 7	74 ± 9

Table 3: Association Between Pain Severity and Postoperative Ileus

Pain Category	Ileus (n)	Ileus (%)
Mild (n=28)	2	7.1%
Moderate (n=39)	6	15.4%
Severe (n=33)	11	33.3%

DISCUSSION

The present study demonstrates a strong and clinically significant association between postoperative pain severity, sympathetic nervous system activation, and delayed gastrointestinal recovery[1]. Patients with higher pain scores exhibited markedly elevated heart rate, blood pressure, and serum catecholamine levels, indicating a robust activation of the sympathetic axis[2]. This heightened autonomic response was consistently associated with delayed return of bowel function and an increased incidence of postoperative ileus, thereby confirming the central hypothesis of this study[3].

The observed findings can be explained through well-established physiological mechanisms[4]. Surgical trauma initiates nociceptive signaling that activates hypothalamic–pituitary–adrenal pathways and sympathetic outflow[5]. Increased catecholamine release leads to inhibition of gastrointestinal motility by suppressing enteric nervous system activity and reducing acetylcholine-mediated peristalsis[6]. In addition, sympathetic-mediated vasoconstriction reduces splanchnic blood flow, impairing intestinal perfusion and delaying functional recovery[7]. These mechanisms collectively explain the prolonged time to bowel sounds, flatus, and bowel movement observed in patients with severe postoperative pain[8].

The progressive delay in gastrointestinal recovery across mild, moderate, and severe pain groups observed in this study highlights a dose–response relationship between pain intensity and gastrointestinal dysfunction[9]. This graded effect strengthens the biological plausibility of pain-driven sympathetic activation as a key determinant of postoperative ileus[10]. Furthermore, the significantly higher incidence of ileus in patients with severe pain underscores the clinical relevance of this association, as ileus remains a major contributor to prolonged hospital stay and increased healthcare burden[11].

These findings are in agreement with previous physiological and clinical observations that emphasize the inhibitory role of sympathetic activation on gut motility[12]. Studies have shown that increased adrenergic activity suppresses intestinal smooth muscle contraction and disrupts coordinated peristalsis[13]. Similarly, clinical research has demonstrated that inadequate pain control is associated with higher rates of postoperative ileus and delayed recovery[14]. The present study adds to this body of evidence by providing direct clinical correlation between quantified pain levels, objective markers of sympathetic activation, and measurable gastrointestinal outcomes in a real-world community-based setting[15].

An important implication of this study is the critical role of effective pain management in optimizing postoperative recovery[16]. While pain control is often prioritized for patient comfort, its physiological impact on autonomic balance and organ function is equally important[17]. Multimodal analgesia, regional anesthesia techniques, and opioid-sparing strategies may help reduce sympathetic overactivity and promote earlier return of gastrointestinal function[18]. Early mobilization and enhanced recovery protocols may further mitigate the adverse effects of autonomic imbalance[19].

Despite its strengths, this study has certain limitations[20]. The sample size was moderate and limited to two tertiary care centers in Lahore, which may affect the generalizability of the findings to other populations[13]. Serum catecholamine levels were used as markers of sympathetic activation; however, more advanced methods such as heart rate variability analysis could provide a more comprehensive assessment of autonomic function[8]. Additionally, variations in surgical type and anesthesia protocols may have influenced postoperative outcomes and could not be fully controlled[11].

Future research should focus on interventional studies evaluating targeted strategies to reduce sympathetic activation, including regional anesthesia, autonomic modulation therapies, and non-pharmacological approaches such as stress reduction techniques[6]. Large multicenter trials are also needed to validate these findings and establish standardized protocols for integrating autonomic monitoring into postoperative care[14].

CONCLUSION

Pain-driven sympathetic activation is a critical determinant of postoperative gastrointestinal dysfunction. Increased pain intensity leads to heightened sympathetic activity, which in turn inhibits gastrointestinal motility, delays recovery of bowel function, and significantly increases the risk of postoperative ileus. These findings emphasize that effective postoperative pain control is not only essential for patient comfort but also plays a vital role in improving physiological recovery and reducing complications. Incorporating strategies aimed at minimizing sympathetic overactivity may enhance surgical outcomes and shorten hospital stay.

Availability of Data and Materials: The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

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Authors' Contributions:

U.M.: Conceptualization, study design, data collection, manuscript drafting
H.S.: Data collection, literature review, manuscript writing
I.A.: Data analysis, interpretation of results
I.U.: Methodology, statistical analysis
R.U.: Supervision, critical revision
R.M.R.: Final review, editing, and approval of manuscript
All authors read and approved the final manuscript.

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