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Effects of Intraperitoneal Administration of Flunixin Meglumine, Metamizole Sodium, Diclofenac Sodium, and Carprofen on Experimentally Induced Intra-Abdominal Adhesion Formation in Rats

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Abstract

Postoperative intra-abdominal adhesions remain a major complication following abdominal surgery and may lead to intestinal obstruction, infertility, chronic abdominal pain, and difficulties during repeat surgical procedures. This experimental study investigated the effects of intraperitoneally administered flunixin meglumine, metamizole sodium, diclofenac sodium, and carprofen on postoperative adhesion formation in a rat cecal abrasion model. Sixty female Sprague–Dawley rats were randomly allocated into six groups including sham, control, flunixin meglumine, metamizole sodium, diclofenac sodium, and carprofen groups (n=10 each). Following standardized cecal abrasion, the treatment agents were administered intraperitoneally immediately after induction of peritoneal injury. Adhesion formation was evaluated macroscopically on postoperative day 10 using the Nair adhesion scoring system. Hematological and biochemical parameters together with tissue hydroxyproline concentrations were also analyzed. All treatment groups demonstrated significantly lower adhesion scores compared with the control group. Metamizole sodium and carprofen produced the most pronounced reduction in adhesion severity. Hydroxyproline concentrations were significantly lower in the carprofen group compared with the flunixin meglumine, metamizole sodium, and diclofenac sodium groups. The findings suggest that intraperitoneal administration of selected NSAIDs may reduce postoperative intra-abdominal adhesion formation and may represent a supportive pharmacological strategy for adhesion prevention.

Key words: Intra-abdominal adhesion, NSAID, Hydroxyproline, Rat, Peritoneal adhesion

Introduction

Postoperative intra-abdominal adhesions are among the most common complications following abdominal surgery and represent a major clinical problem in both human and veterinary medicine. Adhesion formation has been associated with serious consequences such as intestinal obstruction, chronic abdominal pain, infertility and increased difficulty during subsequent surgical interventions (Ellis, 1971; Menzies and Ellis, 1990). Despite improvements in surgical techniques and perioperative management, adhesion development remains a frequent postoperative outcome.

Adhesions are formed as a result of peritoneal injury that initiates a complex inflammatory cascade involving fibrin deposition, leukocyte migration and

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fibroblast activation. Under normal physiological conditions, fibrinolytic activity removes temporary fibrin bridges formed between injured peritoneal surfaces; however, impairment of this mechanism leads to persistent fibrotic connections and permanent adhesion formation (Hellebrekers et al., 2000). Several cytokines, including interleukin-1, interleukin-6 and tumor necrosis factor-alpha, have been shown to play important regulatory roles during adhesion development following peritoneal trauma (Hershlag et al., 1991; Saba et al., 1996).

Various strategies have been investigated to prevent postoperative adhesion formation, including mechanical barrier materials, fibrinolytic agents and pharmacological approaches targeting inflammatory pathways (Treutner and Schumpelick, 2000). Recent advances in adhesion prevention research have highlighted the importance of biomaterial-based barriers and targeted pharmacological delivery systems designed to modulate inflammatory signaling at the site of injury (Rodgers et al., 1998). In addition, several standardized experimental animal models have been developed to evaluate the mechanisms of adhesion formation and to test preventive strategies under controlled conditions (Cofer et al., 1994).

Among pharmacological agents, non-steroidal anti-inflammatory drugs (NSAIDs) have received considerable attention because of their inhibitory effects on cyclooxygenase activity and prostaglandin synthesis during the early stages of adhesion formation (Siegler et al., 1980). Experimental studies have demonstrated that several NSAIDs, including ibuprofen and meclofenamate, reduce adhesion severity by modulating inflammatory mediator release and fibroblast proliferation during peritoneal healing (Muscara et al., 2000).

In veterinary medicine, commonly used NSAIDs such as flunixin meglumine, diclofenac sodium, metamizole sodium and carprofen are widely administered for their analgesic and anti-inflammatory effects. However, comparative evaluation of their potential roles in preventing postoperative intra-abdominal adhesion formation following local intraperitoneal administration remains limited. Since these agents differ in their pharmacological properties, anti-inflammatory potency and cytokine-modulating effects, evaluation of their relative influence on adhesion severity may contribute to identifying more effective pharmacological strategies for postoperative adhesion prevention.

Although several experimental studies have evaluated the effects of individual NSAIDs on adhesion formation, direct comparative investigations assessing the relative effectiveness of these commonly used veterinary agents within the same experimental adhesion model remain scarce. Therefore, the aim of the present study was to investigate the effects of intraperitoneal administration of flunixin meglumine, metamizole sodium, diclofenac sodium and carprofen on postoperative adhesion formation using a standardized rat cecal abrasion model.

Materials and methods

Ethics Statement

This study was approved by the Institutional Animal Experimentation Ethics Committee. All the experimental procedures were conducted in accordance with national and institutional guidelines for the care and use of laboratory animals.

Animals and experimental design

The study was conducted at the Experimental Animal Research Laboratory of the Faculty of Veterinary Medicine, Adnan Menderes University. A total of sixty healthy adult female Sprague-Dawley rats weighing approximately 236±17 g were used. The animals were housed in standard laboratory cages under controlled environmental conditions and had free access to commercial pellet feed and

drinking water throughout the experimental period.

Following overnight fasting, the animals were randomly allocated into six equal groups (n=10 per group): sham group, control group, flunixin meglumine group, metamizole sodium group, diclofenac sodium group and carprofen group.

Surgical Procedure and Drug Administration

General anesthesia was induced by intraperitoneal administration of ketamine hydrochloride (75 mg/kg) and xylazine hydrochloride (10 mg/kg). Following shaving and aseptic preparation of the abdominal region, a 3 cm ventral midline laparotomy was performed.

The cecum was exteriorized gently, and a standardized abrasion model was created by applying repeated strokes to the anterior cecal serosa using a sterile toothbrush until petechial hemorrhage became visible.

Immediately after induction of the adhesion model, treatment solutions were administered intraperitoneally in equal final volumes of 2 mL/kg in all experimental groups in order to standardize intraperitoneal exposure.

The treatment groups received:

- Flunixin meglumine: 2.5 mg/kg
- Metamizole sodium: 50 mg/kg
- Diclofenac sodium: 2.5 mg/kg
- Carprofen: 2.5 mg/kg

The control group received sterile 0.9% NaCl solution in the same final intraperitoneal volume. The abdominal wall was closed routinely in two layers using absorbable suture material for the muscle layer and non-absorbable suture material for the skin.

Postoperative monitoring and sample collection

Following recovery from anesthesia, animals were returned to their cages and maintained under standard laboratory conditions. Ten days after surgery, all animals were euthanized with an overdose of ketamine anesthesia.

The abdominal cavity was reopened through a reverse U-shaped incision to allow complete visualization of intra-abdominal organs. Adhesion formation between the cecum and surrounding tissues was evaluated macroscopically.

Blood samples were collected by cardiac puncture for hematological and biochemical analyses. Hemoglobin concentration and leukocyte counts were determined using an automated blood analyzer. Serum albumin levels were measured using standardized laboratory procedures.

Evaluation of adhesion formation

Adhesion severity was evaluated macroscopically on postoperative day 10 according to the Nair (1974) adhesion scoring system:

Grade	Description
0	No adhesions
1	Single thin adhesion
2	Two thin adhesions
3	More than two adhesions or localized dense adhesions
4	Generalized dense adhesions involving viscera and abdominal wall

All evaluations were performed independently by two blinded observers.

Determination of hydroxyproline levels

Adhesion tissue samples were collected on postoperative day 10 and stored at −85 °C until analysis. Hydroxyproline concentrations were determined spectrophotometrically according to the modified method previously described by Muscara et al. (2000). Results were expressed as µg/g tissue.

Statistical analysis

Adhesion scores were analyzed using the Kruskal–Wallis test. Hematological and biochemical parameters were evaluated by one-way analysis of variance (ANOVA), followed by Duncan's multiple comparison test when appropriate. Statistical analyses were performed using SPSS software, and

differences were considered statistically significant at $P<0.05$.

Results

Macroscopic evaluation of adhesion formation

Macroscopic examination performed on postoperative day 10 demonstrated that intra-abdominal adhesions were mainly located between the cecum and adjacent abdominal wall structures in animals subjected to the abrasion procedure. Adhesion severity differed among the experimental groups (Figure 1).

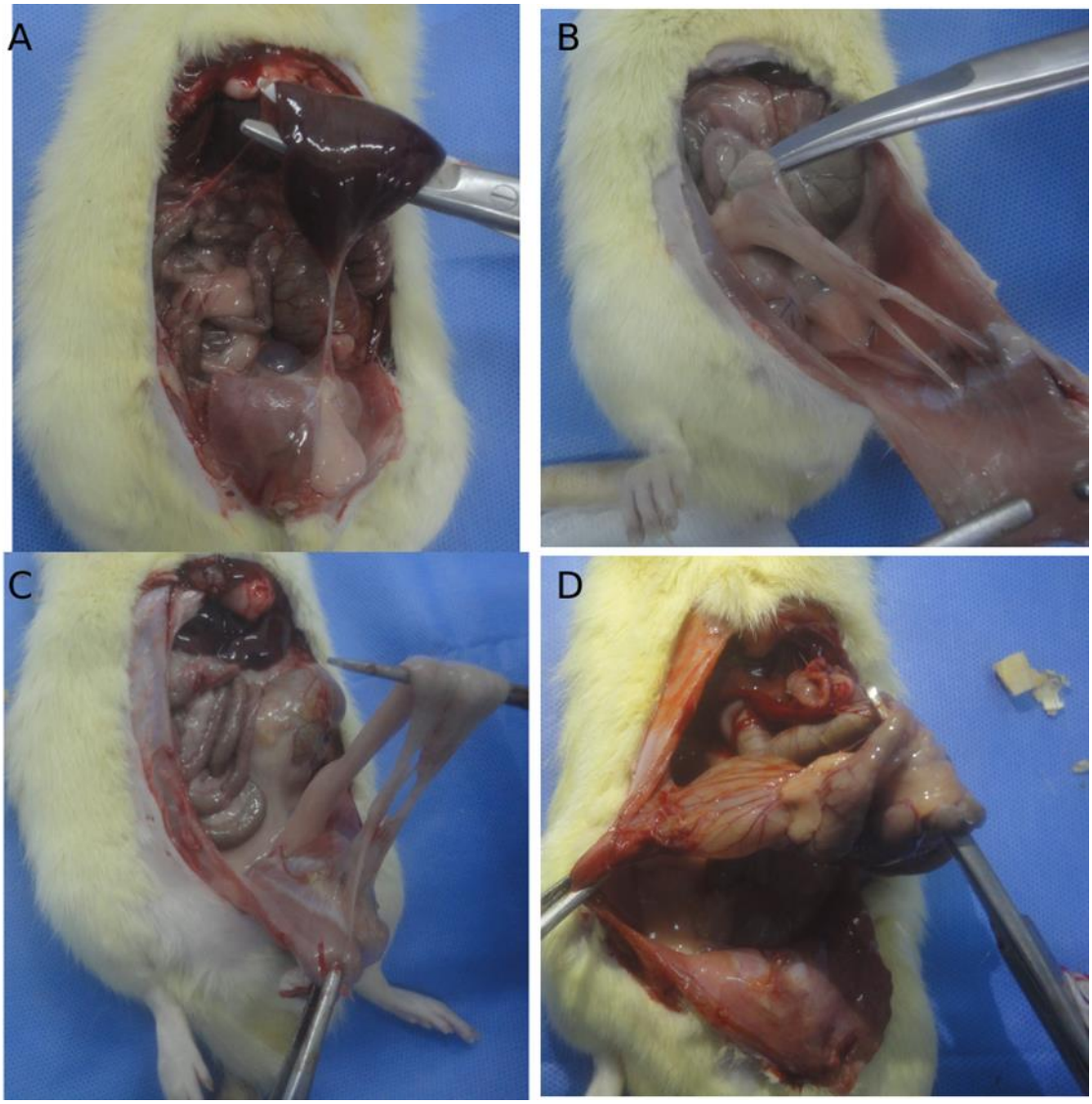

Figure 1: Representative intra-abdominal adhesion formations observed in rats (A) Grade 1 mild adhesion formation from control group. (B) Grade 2 moderate adhesion formation from flunixine group (C). Grade 3 marked adhesion formation from control group and (D) Grade 4 severe adhesion formation from control group.

The highest adhesion scores were observed in the control group, whereas all treatment groups showed reduced adhesion formation compared with controls. Among the evaluated agents, metamizole sodium and carprofen produced the most pronounced reduction in adhesion severity. The distribution of individual adhesion grades and mean adhesion scores for each group are presented in Table 1.

Statistical analysis revealed that adhesion scores in the flunixin meglumine, metamizole sodium, diclofenac sodium and carprofen groups were significantly lower

than those of the control group (P<0.05) (Table 1).

Hematological and biochemical findings

Mean erythrocyte counts, leukocyte counts, packed cell volume values and serum albumin concentrations were similar among experimental groups and did not show statistically significant differences. Detailed hematological and biochemical findings are summarized in Table 2.

However, eosinophil counts were significantly lower in the metamizole sodium group compared with the flunixin meglumine group (P<0.05). In addition, eosinophil values in the carprofen group were significantly reduced compared with the control group (P<0.05) (Table 2). No statistically significant differences were detected in other differential leukocyte parameters.

Table 1: Individual and mean (±SEM) adhesion scores of the rats from the sham, control and treatment groups

Groups	n	Grade					Average [a]
		0	1	2	3	4	
Sham	10	0	2	2	1	3	2.10+0.37
Control	10	0	1	2	4	1	2.20+0.42
Flunixin Meglumine	10**	4	3	1	0	1	0.90+0.18
Metamizol	10*	8	2	0	0	0	0.20+0.05
Diclofenac	10**	5	4	1	0	0	0.60+0.24
Carprofen	10*	7	2	1	0	0	0.30+0.11

* Statistically different from control (P<0.005).
** Statistically different from control (P<0.01).
[a] The data were expressed as means ± standard errors (SEM).

Table 2: Albumin, erythrocyte and total and differential leucocyte counts in the all groups (mean ± SE)

Parameters	Sham (*n* = 10)	Control (*n* = 10)	Flunixin Meglumine (*n* = 10)	Metamizol (*n* = 10)	Diclofenac (*n* = 10)	Carprofen (*n* = 10)
RBC (10^6/µl)	7.90 ± 0.73	7.53 ± 0.21	7.42 ± 0.60	7.81 ± 0.86	7.92 ± 0.60	7.85 ± 0.66
WBC (10^3/µl)	7.70 ± 1.02	7.44 ± 0.42	8.08 ± 0.97	7.83 ± 0.19	7.91 ± 1.93	7.47 ± 0.52
PCV(%)	40.80 ± 1.07	39.70 ± 0.84	39.00 ± 1.46	39.80 ± 0.94	40.77 ± 0.46	39.95 ± 0.74
Neu (%)	39.50 ± 4.52	36.20 ± 4.95	37.87 ± 3.40	40.80 ± 2.30	36.47 ± 0.40	41.00 ± 4.30
Eos (%)	3.16 ± 0.56	3.60 ± 0.72	3.88 ± 0.60	2.71 ± 0.88*	3.08 ± 0.20	3.71 ± 0.18
Baso (%)	0.56 ± 0.23	0.50 ± 0.26	0.87 ± 0.32	0.57 ± 0.20	0.77 ± 0.22	0.47 ± 0.20**
Lym (%)	55.20 ± 1.78	59.60 ± 1.96	54.45 ± 0.67	60.28 ± 0.92	61.85 ± 0.76	58.28 ± 0.52
Monocyte(%)	2.60 ± 0.10	2.78 ± 0.41	3.00 ± 1.43	2.82 ± 0.20	2.00 ± 0.23	2.52 ± 0.40
Albumin	3.76 ± 0.5	3.70 ± 0.2	3.88 ± 0.60	3.61 ± 0.58	3.98 ± 0.30	3.71 ± 0.18

* Statistically different from control groups (P<0.05).
** Statistically different from flunixin group (P<0.05).

Hydroxyproline analysis

Hydroxyproline concentrations measured in adhesion tissue samples varied between groups and reflected differences in collagen deposition within adhesion areas.

The lowest hydroxyproline levels were detected in the carprofen-treated group. This reduction was statistically significant compared with the flunixin meglumine, metamizole sodium and diclofenac sodium groups ($P<0.05$). Quantitative hydroxyproline values obtained from each experimental group are presented in Table 3.

Table 3: Effect of flunixin meglumine, metamizole, diclofenac and carprofen on hydroxyproline content (pg/mg protein) of adhesion tissue (Values are mean±SE).

Parameter	Control	Sham	F.meglumine	Metamizol	Diclofenac	Carprofen
Hidroxyproline (µg/g tissue)	13.1±3.6	15.2±3.3	29.9±6.4	22.1±7.0	21.6±5.1	6.1±0.7*

*Statistically different from flunixin, metamizol and diclofenac group, P<0,05; ± SE (Standart error).

Discussion

Postoperative intra-abdominal adhesions remain one of the most important complications following abdominal surgery and continue to represent a significant clinical challenge in both human and veterinary practice. Adhesion formation results from a cascade of inflammatory responses triggered by peritoneal injury, leading to fibrin deposition, fibroblast proliferation and extracellular matrix remodeling (Hellebrekers et al., 2000). In the present study, intraperitoneal administration of flunixin meglumine, metamizole sodium, diclofenac sodium and carprofen significantly reduced adhesion severity compared with the control group (Table 1). These findings are consistent the with previous experimental studies demonstrating that modulation of early inflammatory mediators plays a critical role in limiting postoperative adhesion formation (Siegler et al., 1980; Baykal et al., 1997).

Peritoneal healing depends on the balance between fibrin deposition and fibrinolytic activity following tissue injury. Disruption of this balance results in persistent fibrotic adhesions between adjacent tissues (Ellis, 1971). Cyclooxygenase-mediated prostaglandin synthesis contributes to inflammatory cell recruitment and fibroblast activation during early wound healing. Therefore, inhibition of cyclooxygenase activity by NSAIDs may reduce adhesion formation through suppression of inflammatory signaling pathways (Muscara et al., 2000). The significantly lower adhesion scores observed in treatment groups compared with controls (Table 1), support this proposed mechanism.

Among the evaluated agents, metamizole sodium and carprofen demonstrated particularly pronounced reductions in adhesion severity. Similar reductions in adhesion formation have been reported in recent experimental studies evaluating pharmacological agents targeting inflammatory and fibrotic pathways in rat adhesion models (Kakanezhadi et al., 2022; Bayhan et al., 2016). These effects may be associated with inhibition of cytokines such as interleukin-6 and tumor necrosis factor-alpha, which are known to play important roles during adhesion development (Saba et al., 1996; Hershlag et al., 1991).

Evaluation of hematological parameters revealed no marked systemic alterations among experimental groups (Table 2), suggesting that intraperitoneal administration of the investigated NSAIDs did not produce significant systemic adverse effects. Similar safety profiles have been reported in the previous experimental adhesion prevention studies evaluating locally administered anti-inflammatory agents (Siegler et al., 1980).

Hydroxyproline concentration is widely accepted as a biochemical indicator of collagen accumulation and fibrosis in healing tissues (Muscara et al., 2000). Reduced hydroxyproline levels observed particularly in the carprofen-treated group (Table 3) suggest decreased collagen deposition within adhesion tissues. Similar findings have been reported in experimental adhesion prevention studies evaluating antifibrotic strategies targeting extracellular matrix remodeling (Baykal et al., 1997). The lower hydroxyproline concentrations observed particularly in the carprofen-treated group may reflect reduced collagen deposition and attenuation of excessive fibrotic tissue remodeling during the healing process. Hydroxyproline is widely accepted as an indirect biochemical marker of collagen synthesis and extracellular matrix accumulation within adhesion tissues. Therefore, reduced hydroxyproline levels may indicate suppression of fibroblast activity and limitation of pathological fibrosis rather than impaired tissue healing. Similar reductions in hydroxyproline concentration have been associated with decreased adhesion severity in the previous experimental anti-adhesion studies.

In addition, the observed differences among sham, control, and treatment groups may be explained by variations in the degree of peritoneal injury and inflammatory response. The sham group underwent laparotomy without severe peritoneal abrasion and therefore exhibited minimal inflammatory stimulation and limited adhesion formation. In contrast, the control group developed pronounced inflammatory reactions and extensive fibrin deposition following standardized cecal abrasion, resulting in higher adhesion scores. Treatment groups demonstrated reduced adhesion severity, likely due to the anti-inflammatory and antifibrotic effects of the administered NSAIDs, which may have modulated cytokine release, fibroblast proliferation, and extracellular matrix remodeling during peritoneal healing.

Recent systematic reviews have emphasized that although several pharmacological and biomaterial-based strategies show promising results in experimental adhesion prevention models, no single intervention has completely eliminated adhesion formation in clinical practice (Fatehi Hassanabad et al., 2021; Schaefer et al., 2024). Therefore, identification of safe and accessible pharmacological alternatives remains an important research objective. The results obtained in the present study suggest that locally administered NSAIDs may represent a useful supportive strategy for reducing postoperative adhesion severity.

Another important aspect of the present study is the use of intraperitoneal administration immediately after induction of peritoneal injury. Local drug delivery may allow higher concentrations at the site of injury while minimizing systemic exposure, which may enhance anti-adhesive efficacy. Similar advantages of localized pharmacological strategies have been highlighted in recent adhesion prevention research (Cofer et al., 1994, Pu et al.,).

Despite these promising findings, further experimental studies including longer observation periods, histopathological evaluation and cytokine profiling are required to better clarify the molecular mechanisms underlying the observed anti-adhesive effects. In addition, controlled clinical investigations will be necessary to determine whether similar outcomes can be achieved under clinical surgical conditions.

In conclusion, intraperitoneal administration of flunixin meglumine, metamizole sodium, diclofenac sodium, and carprofen reduced postoperative intra-abdominal adhesion formation in the experimental rat model.

Among the evaluated agents, metamizole sodium and carprofen demonstrated the most pronounced anti-adhesive effects. Reduced hydroxyproline concentrations in

the carprofen group further supported decreased collagen deposition and fibrosis within adhesion tissues.

The findings suggest that local intraperitoneal administration of selected NSAIDs may represent a useful supportive pharmacological strategy for reducing postoperative adhesion formation.

Further studies involving histopathological evaluation, cytokine analysis, and longer observation periods are warranted to better clarify the underlying molecular mechanisms and potential clinical applicability of these findings.

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Conflict of interest

The authors declare that they have no financial or non-financial conflict of interest regarding authorship and publication of this article

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