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Postpartum nonsteroidal anti-inflammatory drug exposure does not increase risk for flare in patients with inflammatory bowel disease

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Abstract

Background Postpartum pain management is an important part of maternal healthcare. Nonsteroidal anti-inflammatory drugs (NSAIDs) and acetaminophen are typically offered as first-line pharmacologic therapies for postpartum pain. There is a belief that NSAIDs may play a role in exacerbating inflammatory bowel disease (IBD); as a result, some obstetricians avoid NSAIDs for postpartum pain management in patients with IBD. However, data concerning the relationship between short-term NSAID use and IBD flares are inconsistent. The aim of this study was to assess whether hospital postpartum NSAID use is associated with postpartum IBD flare within 9 months from delivery.

Methods This single-center retrospective cohort study included patients with IBD, aged 18 years or older, who had singleton live births between January 1, 2016, and November 30, 2023. Chart review data for each eligible patient were collected for a 9-month postpartum period.

Results Among the 187 patients included in the study, there was no difference between NSAID-exposed and non-exposed patients in postpartum IBD flare: 10/114 (9%) vs. 10/73 (14%), respectively, P=0.335. Based on multivariate regression analysis, NSAID exposure was not associated with postpartum IBD flare, adjusted for active disease at conception and IBD flare during pregnancy: adjusted odds ratio (aOR) 0.6, 95% confidence interval (CI) 0.2-1.7; P=0.327. The same was true for mode of delivery and inpatient opioid exposure: aOR 0.6, 95%CI 0.1-1.5; P=0.291.

Conclusions Postpartum NSAID use for pain control is not associated with IBD flare 9 months after delivery. Large prospective studies are needed to confirm this finding.

Keywords Nonsteroidal anti-inflammatory drugs, pregnancy, inflammatory bowel disease, flare

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Introduction

Inflammatory bowel disease (IBD), encompassing ulcerative colitis (UC) and Crohn's disease (CD), is a chronic, relapsing, and potentially debilitating disorder of the gastrointestinal (GI) tract affecting approximately 2.39 million individuals in the United States [1,2]. The peak incidence of IBD typically occurs in early adulthood and, since approximately 55% of affected individuals are women, a significant proportion of patients require disease management during their reproductive years. Despite this, data on the optimal management of IBD during pregnancy and lactation remain limited [3], though this is changing rapidly.

Effective pain management in the postpartum period is a critical component of maternal care. Given the well-

documented risks associated with opioid use—including dependency, overdose and other adverse outcomes—the use of non-opioid analgesics is increasingly prioritized. According to a 2011 report by the Institute of Medicine, 60% of women report experiencing severe pain during and after their first childbirth, underscoring the need for effective and safe postpartum analgesia [4]. In the context of the ongoing opioid crisis, and the well-studied association between opioid exposure and poor IBD outcomes, optimizing pain control strategies is essential—not only to improve patient outcomes, but also to enhance the overall quality of postpartum healthcare [5,6].

Nonsteroidal anti-inflammatory drugs (NSAIDs) are among the most frequently prescribed analgesics globally, with agents such as ketorolac, ibuprofen and naproxen commonly used for postpartum pain control. However, NSAIDs are known to pose GI risks, and their impact on IBD disease activity remains a subject of ongoing debate [7-11]. Previous data suggested that NSAID exposure may precipitate clinical relapse in up to 20% of patients with quiescent IBD [12,13]. Furthermore, a case–control study by Evans *et al* reported an association between NSAID use and increased risk of emergency hospital admission among IBD patients; however, the study lacked detailed data on the dosage and duration of NSAID exposure [14]. Newer studies have suggested that NSAID use, particularly over a short duration, is not associated with worsened clinical outcomes or risk of flare in patients with IBD [7,15]. These uncertainties highlight the clinical dilemma of selecting appropriate analgesic regimens for patients with IBD during the postpartum period.

The primary aim of this study was to assess whether hospital postpartum NSAID use is associated with postpartum IBD flare within 9 months from delivery. A secondary aim was to investigate the association of NSAID use with postpartum IBD-related hospitalizations, patients needing postpartum GI visits, and the number of such visits made.

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Patients and methods

Study design and population

This was a single-center retrospective cohort study of postpartum patients aged 18 years or older who had singleton live births between January 1, 2016, and November 30, 2023. Patients with IBD were identified using International Classification of Diseases (ICD)-10 codes from the electronic medical record, and the study was further limited to those who were diagnosed with IBD at least 1 year prior to conception. Patients who had spontaneous or elective terminations, multiple gestations, those with missing data (i.e., patients who did not receive IBD care at the primary site), and those without accessible inpatient medication administration records of NSAIDs were excluded. This study was approved by the Institutional Review Board of the Beth Israel Deaconess Medical Center, with a waiver of consent for retrospective analysis of de-identified data (approved on September 25, 2024: IRB#2021P000725).

Study outcomes

The primary aim of the study was the association of NSAID exposure with postpartum IBD flare within 9 months after delivery. Flare was defined as the addition of steroids (oral/rectal); change in medical treatment (including advanced therapies, treatment optimization not based on proactive therapeutic drug monitoring); surgical intervention; or IBD-related hospitalization. Medication use during inpatient course was determined based on inpatient administration records, any history of taking NSAIDs, either selective or non-selective, including but not limited to ibuprofen, ketorolac, and naproxen. Secondary outcomes were postpartum IBD-related hospitalizations, patients needing postpartum GI visits, and the number of such visits made within 9 months after delivery.

Statistical analysis

A per-pregnancy event analysis was performed. Categorical variables were described as frequency with percentage and compared between groups using chi-square or Fisher's exact test, as appropriate. Continuous variables were described as median with interquartile range (IQR), and were compared between groups using the Mann-Whitney *U* test. Multivariable binary regression analyses were performed to determine the independent effect of NSAID use associated with postpartum IBD flare. In view of the small number (n=20) of events (postpartum flares), we restricted the multivariable analyses to a maximum of 2 covariates, as follows: model 1 (IBD medication at conception and IBD flare during pregnancy; model 2 (active disease at conception and corticosteroids at the time of delivery; model 3 (mode of delivery and inpatient opioid exposure); model 4 (active disease at conception and IBD flare during pregnancy). Unadjusted and adjusted odds

ratios (ORs) with 95% confidence intervals (CI) were also provided. All analyses were performed using SPSS version 28.0 (SPSS, Chicago, IL, USA) and GraphPad Prism version 5.03 for Windows (GraphPad Software, San Diego, CA, USA).

Results

Demographic and clinical characteristics

A total of 398 pregnancies in 336 patients were screened. Including only the patients with accurate inpatient medication administration and available GI and obstetric charts, a total of 187 pregnancies were ultimately included in the analysis, with 114 exposed to NSAIDs during the postpartum hospitalization and 73 unexposed (Table 1). The median maternal age at delivery was 32 years (IQR: 35-38), and the median length of hospitalization was 3 days (IQR: 3-5) in both groups, with no significant differences between NSAID users and non-users (P=0.986 and P=0.718, respectively). Regarding IBD subtype, UC, CD and IBD-unclassified (IBD-U) were present in 47%, 48% and 5% of the cohort, respectively. Distribution by IBD type did not significantly differ in relation to NSAID exposure (P=0.151). The median disease duration was 10 years (IQR: 6-16), comparable across groups (P=0.416). Median

Table 1 Demographic and clinical characteristics (per pregnancy analysis)

Characteristics	Overall, n=187	NSAIDs, n=114	No NSAIDs, n=73	P-value
Age at delivery, median, (IQR), years	35 (32-38)	35 (31-38)	35 (32-37)	0.986
Race, (%)				0.116
White	170 (91)	107 (94)	63 (86)	
Other	17 (9)	7 (6)	10 (14)	
Ethnicity, (%)				0.769
Hispanic	174 (93)	105 (92)	69 (95)	
Non-Hispanic	13 (7)	9 (8)	4 (5)	
IBD type, (%)				0.151
UC	88 (47)	60 (53)	28 (38)	
CD	89 (48)	48 (42)	41 (56)	
IBD-U	10 (5)	6 (5)	4 (6)	
Prior IBD-related surgery, (%)	40 (21)	23 (20)	17 (23)	0.715
Prior biologic therapy, (%)	64 (34)	37 (32)	27 (37)	0.532
IBD duration, median, (IQR), years	10 (6-16)	9 (5-15.5)	10 (6-16)	0.416
Preconception BMI, kg/m^2	23.2 (21.5-27.7)	23.3 (21.6-28.3)	23.1 (21-26.6)	0.198
Smoking status, (%)	27 (14)	16 (14)	11 (15)	0.835
Insurance status, (%)				0.424
Medicaid	16 (9)	8 (7)	8 (11)	
Other	171 (91)	106 (93)	65 (89)	
IBD medication at conception, (%)				0.559
None	30/185 (16)	20 (18)	10/71 (14)	
Non-biological therapy	83/185 (45)	41 (36)	30/71 (42)	
Biological therapy	72/185 (39)	53 (46)	31/71 (44)	
Low dose aspirin during pregnancy, (%)	27/153 (18)	20/95 (21)	7/58 (12)	0.193
Hospitalization, median (IQR), days	3 (3-5)	3 (3-5)	3 (3-5)	0.718
Active disease at conception, (%)	24/155 (15)	14/96 (15)	10/59 (17)	0.820
IBD flare during pregnancy, (%)	42 (22)	22 (19)	20 (27)	0.212
Inpatient narcotics, (%)	75 (40)	43 (38)	32 (44)	0.446
Inpatient acetaminophen, (%)	172/184 (93)	107 (94)	65/70 (93)	0.768
Inpatient morphine equivalent dose, median, (IQR), mg	30 (10-76.3)	30 (10-68)	28.1 (15-112.5)	0.511
CS at the time of delivery, (%)	14/180 (8)	8/110 (7)	6/70 (9)	0.780
Mode of delivery, (%)				0.115
Vaginal	122 (65)	69 (61)	53 (73)	
Cesarean section	65 (35)	45 (39)	20 (27)	

NSAIDs, nonsteroidal anti-inflammatory drugs; IQR, interquartile range; UC, ulcerative colitis; CD, Crohn's disease; IBD-U, inflammatory bowel disease unclassified; BMI, body mass index; CS, corticosteroids

preconception body mass index was similar between NSAID and non-NSAID users (21.6 vs. 21.0 kg/m^2, P=0.198).

Fourteen percent (14%) of subjects were current smokers, with no difference between groups (P=0.835). Medicaid insurance coverage was reported in 9% of patients, and insurance status did not differ significantly by NSAID use (P=0.424). At conception, 16% of patients were on no IBD medications, 45% were on non-biologic therapy and 39% on biologics. There was no significant difference between groups in IBD medication use at conception (P=0.559). Use of low-dose aspirin during pregnancy occurred in 18% of participants and did not differ between groups (P=0.193). Active disease at conception was present in 15% of the cohort, and 22% of the cohort flared during pregnancy, with no differences between NSAID-exposed and non-exposed groups (14/96 [15%] vs. 10/59 [17%] P=0.820, and 22/114 [19%] vs. 20/73 [27%] P=0.212, respectively). Inpatient narcotic use during delivery hospitalization occurred in 40% of cases and was similar between NSAID users and non-users (43/114 [38%] vs. 32/73 [44%], P=0.446), as was acetaminophen use (93% overall, P=0.768). No statistically significant difference was observed in morphine equivalent dosage use between NSAID users and non-users (30 mg [10-76.3], P=0.511). Vaginal delivery was more common overall (65% vs. 35%), but the mode of delivery did not differ significantly in relation to NSAID exposure (P=0.115).

NSAIDs and postpartum IBD flare

Among the 187 pregnancies analyzed, postpartum IBD flare occurred in 9% of cases, with no statistically significant difference between NSAID-exposed and unexposed groups (P=0.335) (Table 2). Based on multivariate regression analysis (Table 3), NSAID exposure was not associated with postpartum IBD flare, adjusted for active disease at conception and IBD flare during pregnancy (aOR 0.6, 95%CI 0.2-1.7; P=0.327), as well as mode of delivery and inpatient opioid exposure (aOR 0.6, 95%CI 0.1-1.5; P=0.291).

NSAIDs and other outcomes

Postpartum IBD-related hospitalizations were reported in 13% of cases overall, and rates were identical among NSAID exposed and non-exposed patients (13% in each group, P>0.99 with 0% risk difference between groups). Most patients (89%) had postpartum GI visits, with a median of 2 visits (IQR: 1-3). Visit frequency and proportion of patients seen post-delivery were similar between groups, with a 1% risk difference between exposed and non-exposed patients (P=0.796 and P=0.264, respectively) (Table 2).

Discussion

This study's findings indicate that short-term NSAID use appears to be a safe and effective approach to postpartum pain control for patients with IBD, and is in alignment with current American College of Obstetricians and Gynecologists' (ACOG) first-line recommendations [16,17]. In this study, the use of NSAIDs for postpartum analgesia was not associated with an increased risk of IBD flare in the 9 months following delivery, and NSAID use was not linked to IBD-related hospitalizations in the postpartum period.

ACOG recommends NSAIDs as a first-line therapy for postpartum pain management, with opioids added only if needed, and for the shortest reasonable duration [17]. The recommendation of non-opioid analgesics for the general postpartum patient population is intended to minimize the risk of development of opioid use disorders among opioid-naïve patients, and to reduce the chances of opioid-induced side-effects. Furthermore, although both NSAIDs and opioids are excreted into breastmilk, breastfed infants of individuals prescribed opioids may experience central nervous system depression and neonatal sedation, making non-opioid analgesics a safer option for breastfed infants. Despite this, and perhaps as a result of the widely held belief that NSAIDs increase the likelihood of IBD flares, many patients with IBD and their obstetricians may avoid NSAIDs during postpartum recovery.

The literature linking NSAIDs to IBD flares is inconclusive. It is known that NSAIDs are associated with mucosal injury, and several studies have reported an increased likelihood of IBD flares after NSAID exposure [18-20]. However, a meta-analysis of 18 studies that examined the relationship between NSAIDs and IBD exacerbations found no significant association between NSAID use and the risk of UC or CD exacerbation [15]. Additionally, a cohort study published in 2023, which included 15,705 patients with IBD, with and without NSAID exposure, suggested that the relationship between NSAIDs and IBD exacerbations might be attributable to "protopathic bias", in which NSAIDs might be used to treat

Table 2 Postpartum outcomes by NSAID exposure

Outcomes	Overall, n=187	NSAIDs, n=114	No NSAIDs, n=73	P-value	Absolute risk	Risk difference
Postpartum IBD flare, (%)	20 (11)	10 (9)	10 (14)	0.335	9%	-5%
Postpartum IBD-related hospitalizations, (%)	19/151 (13)	12/95 (13)	7/56 (13)	>0.99	13%	0%
Postpartum GI visits, (%)	135/152 (89)	84/94 (89)	51/58 (88)	0.796	89%	1%
Number of postpartum GI visits, median, (IQR)	2 (1-3)	2 (1-3)	2 (1-5)	0.264	N/A	N/A

NSAIDs, nonsteroidal anti-inflammatory drugs; IBD, inflammatory bowel disease; GI, gastroenterology; IQR, interquartile range; N/A, not applicable

Table 3 Association of NSAID use with postpartum IBD flare

NSAIDs	OR	95%CI	P-value	VIF
	0.6	0.2-1.5	0.291	N/A
	aOR	95%CI	P-value	
Model 1 (IBD medication at conception, IBD flare during pregnancy)	0.7	0.3-1.7	0.407	1.005
Model 2 (active disease at conception, CS at the time of delivery)	0.6	0.2-1.7	0.314	1.001
Model 3 (mode of delivery, inpatient opioid exposure)	0.6	0.2-1.5	0.291	1.177
Model 4 (active disease at conception, IBD flare during pregnancy)	0.6	0.2-1.7	0.327	1.291

NSAIDs, nonsteroidal anti-inflammatory drugs; OR, odds ratio; CI, confidence interval; VIF, variance inflation factor; aOR, adjusted odds ratio; IBD, inflammatory bowel disease; N/A, not applicable; CS, corticosteroids

early signs of an IBD flare (e.g., joint pain) and consequently their association with the flare cannot be considered causal [7].

Existing literature on postpartum pain control for patients with IBD is limited. In a recent retrospective cohort study, Johnson *et al* found that postpartum use of ketorolac—a commonly administered intravenous NSAID—was not associated with increased clinical disease activity up to 6 weeks postpartum [21]. In that study, the use of oral (PO) NSAIDs, and the NSAID dosage and frequency, were not reported. A recent grading recommendation by Mahadevan *et al* emphasized the safety of NSAIDs for postpartum pain management, and recommended them over opiates, considering their safety profile [22]. Our study adds to the existing literature with its inclusion of both IV and PO NSAIDs, and with a longer duration of follow up following delivery to assess for the longitudinal risk of postpartum IBD flare.

This study had some limitations. First, as a retrospective observational analysis, it cannot establish causality between NSAID exposure and postpartum IBD outcomes. Second, because the cohort was derived from a single center, generalizability to other practice settings may be limited. Although NSAID dose, route and duration were abstracted, the sample size limited the statistical power for detailed subgroup comparisons, while direct comparisons across specific NSAIDs are difficult to interpret, given the absence of an accepted standardized potency-equivalence framework. In addition, outpatient NSAID use after discharge was not reliably captured, as many NSAIDs are obtained over the counter and prescription records do not confirm actual use. Consistently with the retrospective design, we did not contact participants to ascertain post-discharge exposure, in order to avoid introducing recall bias that might have led to underestimation of the total postpartum NSAID use. Finally, because we used a more stringent definition of postpartum flare, milder or subclinical changes in disease activity may not have been detected. Importantly, postpartum flare rates were low across the cohort despite these constraints, supporting the conclusion that NSAID use during the delivery hospitalization, as assessed here, was not associated with an increased risk of postpartum flare.

In conclusion, our findings suggest that short-term NSAID use during the postpartum period is not associated with an increased risk of IBD flare within 9 months of delivery. Future research should include larger, prospective, multicenter studies to better characterize how NSAID dosing, duration and formulation may influence disease activity in the postpartum patient population.

Summary Box

What is already known:

- The diagnosis of inflammatory bowel disease (IBD) peaks in the third decade of life and frequently overlaps with pregnancy
- Pain management is essential during the postpartum period
- Nonsteroidal anti-inflammatory drugs (NSAIDs) are known to cause gastrointestinal toxicity and their role in IBD exacerbation remains controversial

What the new finding is:

- Short term NSAID use during postpartum period for pain control was not associated with IBD flare up to 9 months after delivery

References

1. Lewis JD, Parlett LE, Jonsson Funk ML, et al. Incidence, prevalence, and racial and ethnic distribution of inflammatory bowel disease in the United States. *Gastroenterology* 2023;**165**:1197-1205.
2. Wan J, Zhou J, Wang Z, et al. Epidemiology, pathogenesis, diagnosis, and treatment of inflammatory bowel disease: insights from the past two years. *Chin Med J (Engl)* 2025;**138**:763-776.
3. Rabinowitz LG, Rabinowitz DG, Silver EM, Oxentenko AS, Williams KE, Silver JK. Disparities persist in inclusion of female, pregnant, lactating, and older individuals in inflammatory bowel disease clinical trials. *Gastroenterology* 2022;**163**:8-13.
4. Eshkevari L, Trout KK, Damore J. Management of postpartum pain. *J Midwifery Womens Health* 2013;**58**:622-631.
5. Leziak K, Yee LM, Grobman WA, Badreldin N. Patient experience

with postpartum pain management in the face of the opioid crisis. *J Midwifery Womens Health* 2021;**66**:203-210.
6. Rabinowitz LG, Zylberberg HM, Yang J, et al. De novo ostomy placement is associated with increased outpatient opioid use in patients with inflammatory bowel disease. *Dig Dis Sci* 2022;**67**:4033-4042.
7. Cohen-Mekelburg S, Van T, Wallace B, et al. The association between nonsteroidal anti-inflammatory drug use and inflammatory bowel disease exacerbations: a true association or residual bias? *Am J Gastroenterol* 2022;**117**:1851-1857.
8. Klein A, Eliakim R. Non steroidal anti-inflammatory drugs and inflammatory bowel disease. *Pharmaceuticals* 2010;**3**:1084-1092.
9. Meyer AM, Ramzan NN, Heigh RI, Leighton JA. Relapse of inflammatory bowel disease associated with use of nonsteroidal anti-inflammatory drugs. *Dig Dis Sci* 2006;**51**:168-172.
10. Hijos-Mallada G, Sostres C, Gomollón F. NSAIDs, gastrointestinal toxicity and inflammatory bowel disease. *Gastroenterol Hepatol* 2022;**45**:215-222.
11. Kefalakes H, Stylianides TJ, Amanakis G, Kolios G. Exacerbation of inflammatory bowel diseases associated with the use of nonsteroidal anti-inflammatory drugs: myth or reality? *Eur J Clin Pharmacol* 2009;**65**:963-970.
12. Kvasnovsky CL, Aujla U, Bjarnason I. Nonsteroidal anti-inflammatory drugs and exacerbations of inflammatory bowel disease. *Scand J Gastroenterol* 2015;**50**:255-263.
13. Felder JB, Korelitz BI, Rajapakse R, Schwarz S, Horatagis AP, Gleim G. Effects of nonsteroidal antiinflammatory drugs on inflammatory bowel disease: a case-control study. *Am J Gastroenterol* 2000;**95**:1949-1954.
14. Evans JM, McMahon AD, Murray FE, McDevitt DG, MacDonald TM. Non-steroidal anti-inflammatory drugs are associated with emergency admission to hospital for colitis due to inflammatory bowel disease. *Gut* 1997;**40**:619-622.
15. Moninuola OO, Milligan W, Lochhead P, Khalili H. Systematic review with meta-analysis: association between acetaminophen and nonsteroidal anti-inflammatory drugs (NSAIDs) and risk of Crohn's disease and ulcerative colitis exacerbation. *Aliment Pharmacol Ther* 2018;**47**:1428-1439.
16. Telfer L, Dalessio S, Tinsley A, Coates M. Short-term and long-term opioid use is associated with poor outcomes in outpatients with inflammatory bowel disease. *Gastro Hep Adv* 2023;**2**:1022-1024.
17. Pharmacologic stepwise multimodal approach for postpartum pain management: ACOG Clinical Consensus No. 1. *Obstet Gynecol* 2021;**138**:507-517.
18. Long MD, Kappelman MD, Martin CF, Chen W, Anton K, Sandler RS. Role of nonsteroidal anti-inflammatory drugs in exacerbations of inflammatory bowel disease. *J Clin Gastroenterol* 2016;**50**:152-156.
19. Takeuchi K, Smale S, Premchand P, et al. Prevalence and mechanism of nonsteroidal anti-inflammatory drug-induced clinical relapse in patients with inflammatory bowel disease. *Clin Gastroenterol Hepatol* 2006;**4**:196-202.
20. Forrest K, Symmons D, Foster P. Systematic review: is ingestion of paracetamol or non-steroidal anti-inflammatory drugs associated with exacerbations of inflammatory bowel disease? *Aliment Pharmacol Ther* 2004;**20**:1035-1043.
21. Johnson S, DeBolt CA, Rao MG, et al. Ketorolac for postpartum pain management in patients with inflammatory bowel disease. *Am J Obstet Gynecol MFM* 2024;**6**:101238.
22. Mahadevan U, Seow CH, Barnes EL, et al; Global Consensus Group for Pregnancy and IBD. Global consensus statement on the management of pregnancy in inflammatory bowel disease. *J Crohns Colitis* 2025;**19**:jjaf129.