International Journal of Basic & Clinical Pharmacology
Ashwini J et al. Int J Basic Clin Pharmacol. 2025 Sep;14(5):791-796
http://www.ijbcp.com
pISSN 2319-2003 | eISSN 2279-0780

DOI: https://dx.doi.org/10.18203/2319-2003.ijbcp20252578

Original Research Article

A study on the prevalence of risk factors associated with non-steroidal anti-inflammatory drugs-induced adverse effects among inpatients at a tertiary care hospital

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Received: 14 June 2025
Revised: 14 July 2025
Accepted: 15 July 2025

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ABSTRACT

Background: Non-steroidal anti-inflammatory drugs (NSAIDs) are widely used for pain relief and as antipyretics in day-to-day life. However, their adverse effects can be intensified in the presence of certain risk factors. This study aims to identify the risk factors associated with NSAID-induced adverse effects. It also serves to support clinical pharmacists in minimizing these risks and enhancing patient safety.
Methods: Data from a total of 175 inpatients were collected prospectively using a structured data collection form based on defined inclusion and exclusion criteria. The study was conducted over a period of six months. The WHO-UMC causality assessment scale was used to evaluate adverse drug reactions (ADRs), and Hartwig's severity assessment scale was used to determine the severity. SPSS software was employed for analysing the categorized data.
Results: Among 175 patients receiving NSAID therapy, the most prevalent risk factor identified was polypharmacy (64%), followed by older age (38%). Preventive co-therapies, such as proton pump inhibitors (PPIs), were administered in 81.1% of cases. The study also reported a specific ADR generalized pruritus induced by diclofenac, which was assessed as probable on the WHO-UMC causality scale and mild (Level 2) on Hartwig's severity scale.
Conclusions: The study concludes that all NSAID prescriptions were rational and appropriate. Identifying risk factors and prescribing preventive co-therapies during NSAID treatment contributes to improved patient care and therapeutic outcomes.

Keywords: NSAIDs, Risk factors, Drug-drug interactions, Prevalence

INTRODUCTION

Non-steroidal anti-inflammatory drugs (NSAIDs) are one of the most commonly prescribed classes of drugs, having anti-inflammatory, antipyretic and analgesic properties. Because of their significant efficacy, low abuse potential and long-term clinical use to relieve various types of pain, they are one of the most widely used drugs in primary healthcare.[1] NSAIDs are very effective in treating pain due to inflammation and, unlike morphine, they do not depress the CNS, do not cause physical dependence, have no addiction potential or any other negative effects. NSAIDS are also known as non-narcotic, non-opioid and aspirin-like analgesics. Based on their chemical structure, NSAIDs can be classified into four classes they are non-selective cox inhibitors (Traditional NSAIDs), preferential cox-2 inhibitors, selective cox-inhibitors and analgesic anti-pyrectics with poor anti-inflammatory Action.[2] NSAIDs block prostaglandin synthesis through the inhibition of cyclooxygenase enzymes (COX-1 and COX-2). COX-1 produces prostaglandins and thromboxane A2 (TXA2), which control the mucosal barrier in the gastrointestinal tract, renal homeostasis and platelet

aggregation. COX-2 produces prostaglandins, which are related to inflammation, pain, and fever.[1,3] Side effects have a greater influence in terms of morbidity, mortality, quality of life and also increase healthcare resource utilization, such as diagnostic testing, doctor visits, hospitalizations and medications. Identification of specific risk factors and precise determination of NSAID-induced adverse effects will help us to develop appropriate management strategies for patients taking NSAID therapy, leading to maximum potential benefits and minimum adverse events.[4] The aim of the study is to identify and assess ADRs in both inpatients and outpatients by monitoring new or worsening symptoms, utilizing laboratory investigations, and applying standardized tools such as the WHO causality assessment scale and Hartwig severity scale, with the objective of improving early detection, appropriate therapy adjustments, and overall patient safety.

Adverse effects and risk factors of NSAIDS

Although NSAIDs are commonly prescribed, their clinical utility is limited by adverse reactions. NSAIDs mainly affect the gastrointestinal, renal and cardiovascular systems.[5] The inhibition of COX-1 plays a major role in gastrointestinal side effects and renal toxicities, and the inhibition of COX-2 has the risk of cardiovascular events.[1,6] However, the majority of the patients taking therapeutic doses of these NSAIDs for a shorter duration of treatment usually tolerate them well. However, with longer duration of treatment and in the presence of comorbidities, higher risk may emerge. Overall, NSAID therapy is challenging, since decisions must be made based on the patient's present condition and the risk: benefit ratio.[7] Gastrointestinal side effects include unfavourable signs like dyspepsia or vomiting, to more serious problems including gastroduodenal ulcers and bleeding.[6] Cardiovascular side effects such as thrombotic events (myocardial infarction, thrombosis and stroke) and blood pressure, especially when taken in high doses over a prolonged period of time.[3] NSAID use can cause reversible impairment of glomerular filtration, acute or chronic renal failure, interstitial nephritis, papillary necrosis, salt and water retention, edema, or hyperkalemia. Long-term use of NSAIDs (usually high doses) has been associated with analgesic nephropathy. The common CNS side effects include headache, vertigo, dizziness, nervousness, tinnitus, depression, confusion, drowsiness, insomnia, and visual disturbances.[6] Headaches are a common side effect of NSAIDs therapy, even though they are not clinically relevant. NSAID use may increase the risk of excessive bleeding or post-operative blood loss due to a more pronounced effect on platelets, thrombocytopenia, hemolytic anaemia, and agranulocytosis. Patients using NSAIDs may experience minor elevations of one or more liver tests, especially ALT or AST levels.[6,7] Based on the clinical parameters of each patient, the prevalence of GI problems can vary significantly. Therefore, it is essential to identify the NSAID-related GI risk factors in order to choose the best line of treatment for each patient. Numerous studies have already revealed that a number of factors, such as increasing age, concomitant use of systemic steroids or anticoagulants, history of a GI bleed, certain patterns of prior NSAID use, history of cardiovascular disease, smoking and alcohol status, and NSAID-related GI symptoms.[8] The risk factors include Older age, peptic ulcer, higher NSAIDs dose, alcohol, *Helicobacter pylori* infection and comorbidities.[9,10] The elderly population is at risk for drug interactions due to their multiple medical problems that require drug therapy. Elderly patients may also have less capability for drug metabolism and excretion.[11]

WHO defines ADR as "any response to a drug which is noxious and unintended, and which occurs at doses normally used in man for prophylaxis, diagnosis or therapy of disease or for the modification of physiologic functions."[12] According to Rawlins-Thompson classification, ADR are broadly classified as type A, type B, type C, type D, type E, type F, type G, type H and type U.[13]

Type A: Type A adverse reaction is dose dependent, predictable and reversible on reducing the dose or withdrawing the drug. It is also known as an augmented reaction. Predisposing factors of type A reactions are dose, age, pharmaceutical variation in drug formulation, pharmacokinetic or pharmacodynamic abnormalities, and drug-drug interactions.

Type B: Type B reactions are serious, non-dose related and less common than type A reactions. It is also known as a bizarre reaction.

Type C: Type C reactions are chronic reactions, dose-related and time-related. It is uncommon. It can be managed by reducing the dose or withholding the drug.

Type D: It is known as delayed reactions. This type of reaction is uncommon, dose-related and occurs or becomes apparent sometime after use of the drug.

Type E: Type E reactions are known as end-of-use, uncommon and occur soon after the withdrawal of the drug. It can be managed by reintroducing the drug or by slow withdrawal of the drug.

Type F: It is also known as the unexpected failure of therapy. This type of reaction is common, dose-related and often caused by drug interactions. It can be managed by increasing the dosage or considering the effects of concomitant therapy.

METHODS

This was a prospective observational study conducted over a period of 6 months (April to September 2022) at the Vijaya group of hospitals, a unit of Vijaya medical and educational trust, Vadapalani. All in-patients of either gender who were prescribed NSAIDs, aged above 18

years, receiving either monotherapy or combination therapy with NSAIDs, and all available dosage forms were included in the study. Pediatric patients, cases of intentional overdose, pregnant and lactating women, outpatients, casualty cases, HIV patients, and patients with communicable diseases were excluded.

Data were collected from case sheets using a specifically designed data collection form, which included patient demographics (age, sex, weight), co-morbidities, past and present medication history, social history, medical history, newly diagnosed diseases, drug treatment regimen, laboratory parameters, and other investigations (such as USG scans).

Following approval from the institutional ethics committee, eligible patients were selected based on the inclusion and exclusion criteria. Data were then recorded using the patient data collection sheet. Risk factors for NSAID-induced complications were identified by analyzing the collected data and categorizing them based on factors such as organ damage, age, polypharmacy, and co-morbidities. NSAID-induced ADRs were assessed using the WHO-UMC causality scale and Hartwig's severity assessment scale.

Patients were followed up daily until discharge. Any new complaints, relevant laboratory parameters (hematology, RFT, LFT, INR, PT, BT, APTT, cardiac markers), newly prescribed medications, progress notes, and adverse events, if any, were documented daily. Data analysis was carried out using IBM statistical package for the social sciences (SPSS), version 21.0. The study was approved by the institutional ethics committee of Vijaya group of hospitals (Approval No. IECVBMHR/LTR/2022/030).

RESULTS

Among 175 patients, most belonged to the age group of >60 years (44.6%), followed by 50-59 (20.6%), and the lowest population was between the age group 19-29 (9.1%). In our study, most of the patients were male, 104 (59.4%), and females were 71 (40.6%). The 77.1% patients were prescribed NSAIDs for pain, 15.4% patients were prescribed for inflammation, and about 7.4% patients were prescribed for fever during our study, as shown in Figure 1. Out of 175 patients prescribed NSAIDs during hospitalization, paracetamol 159 (90.85%) and diclofenac 27 (15.42%) were the most commonly prescribed NSAIDs, and the least prescribed NSAID was mefenamic acid 1 (0.57%). The selective COX 2 inhibitor was not prescribed as shown in Table 1. The 115 (65.7%) patients were prescribed 1 NSAID, 50 (28.6%) were prescribed 2 NSAIDs, and 10 (5.7%) were prescribed 3 NSAIDs. Within our study population, most of the patients were prescribed monotherapy (68.6%), although some patients were prescribed combination therapy (31.4%), as shown in Figure 2. Most of the NSAIDs were prescribed via oral route (78.28%), followed by parenteral route (38.8%), followed by topical (5.71%), and combination of parenteral and oral route (18.28%). However, the rectal (0.57%) and topical (5.71%) NSAIDs were the least prescribed. Patients were also prescribed other drugs for their co-morbidities. From the selected population, 36.57% patients were prescribed 10-15 drugs, and 4.57% patients were prescribed <5 drugs, found to be the least prescribed, as shown in Table 2. Based on the risk factors, polypharmacy (64.57%) was found to be a major risk factor for NSAIDs-induced adverse effects and renal disorder (3.42%) was the least risk factor, as shown in Table 3. The number of risk factors also contributes to inducing ADRs. In this study, 93 patients had between 3 and 5 risk factors, and 23 patients had more than>5 risk factors, as shown in Table 4. In our study, 142 patients were prescribed PPIs, 6 patients were prescribed H2 receptor antagonists, and 2 patients were prescribed antacids. Among these 25 patients, none were co-prescribed with PPI, H2 antagonist or antacids, as shown in Figure 3. Moderate and minor drug interactions were found during our study period and monitored closely, as shown in Table 5. Most of the drug interaction was found between acetaminophen (n=6), followed by ketorolac (n=3) and aceclofenac (n=3). During our study period, general pruritus induced by diclofenac was identified. It was monitored and managed with tramadol. The risk factor identified in particular patients was older age and polypharmacy. The suspected adverse drug reaction was reported and documented in the suspected adverse drug reaction form. According to the WHO causality scale, it was classified as a probable adverse drug reaction, and severity was classified as level 2 under the mild category according to Hartwig's severity scale.

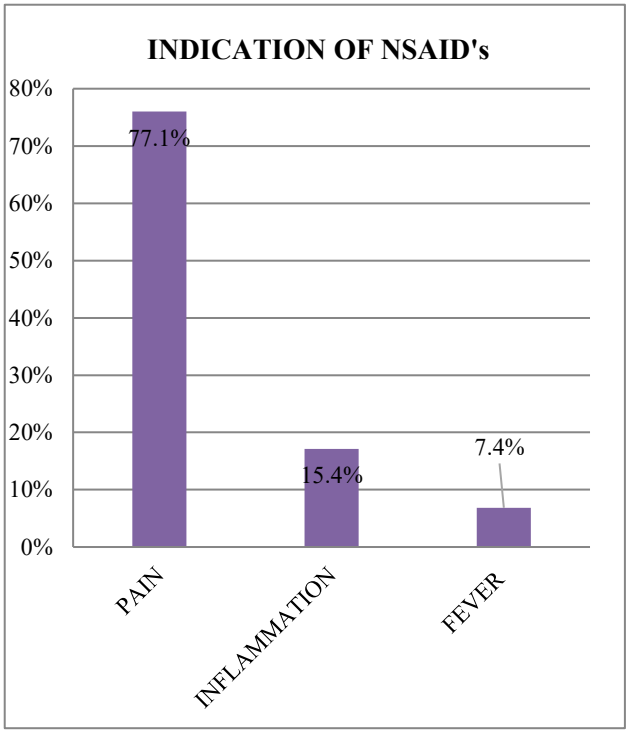

Figure 1: Indication of NSAIDs.

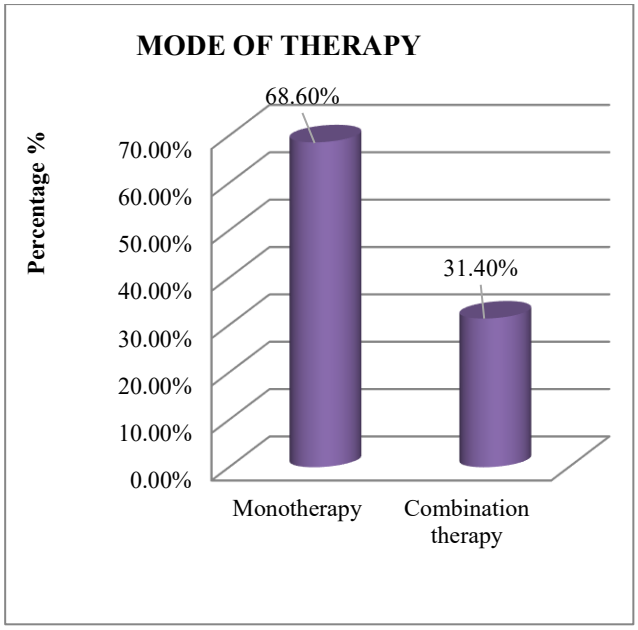

Figure 2: Mode of therapy of NSAIDs.

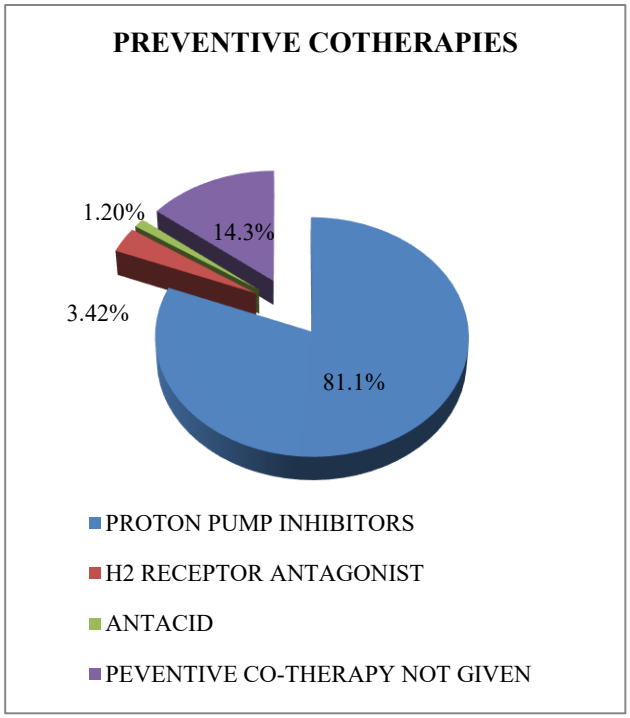

Figure 3: Gastro-protective agents.

Table 1: Distribution of NSAIDS prescribed.

NSAIDs prescribed	N	Percentage (%)
Paracetamol	159	90.85
Diclofenac	27	15.42
Ketoralac	13	7.42
Aceclofenac	9	5.14
Ibuprofen	4	2.28
Naproxen	4	2.28
Piroxicam	4	2.28
Mefenamic acid	1	0.57

Table 2: Number of drugs prescribed, (n=175).

Parameters	N	Percentage (%)
<5 drugs	8	4.57
5-10 drugs	54	30.85
10-15 drugs	64	36.57
15-20 drugs	36	20.57
20-25 drugs	13	7.42

Table 3: Risk factors.

Parameters	N	Percentage (%)
Older age	68	38.84
Polypharmacy	113	64.57
Preventive co-therapies not given	25	14.28
Previous gastro intestinal problem	9	5.14
Alcohol	11	6.28
Anti-coagulants	37	21.14
Corticosteroid	35	20.0
Drug-drug interaction	16	9.14
Diabetes mellitus	68	38.85
Hypertension	81	46.28
Liver disorder	12	6.85
Renal disorder	6	3.42
Heart disorder	59	33.79
Bleeding disorder	20	11.42

Table 4: Number of risk factors, (n=175).

Risk factors	N	Percentage (%)
0-2	59	33.71
3-5	93	53.15
>5	23	13.14

Table 5: Drug-drug interactions.

NSAIDs	Other drugs	Effect of mechanism
Acetaminophen	Metronidazole	Metronidazole increases effect of acetaminophen by CYP2E1 metabolism
	Enoxaparin	Acetaminophen increase effect of enoxaparin by unknown mechanism.
	Heparin	Acetaminophen increase effect of heparin
	Phenytoin	Phenytoin decreases level of acetaminophen by increasing metabolism.
Diclofenac	Metoprolol	Both increase serum potassium
Ketorolac	Gentamycin	Ketorolac increases the level of gentamycin by decreasing renal clearance
	Dexamethazone	Risk of gastrointestinal ulceration
	Metoprolol	Ketorolac decreases prostaglandin synthesis, both increase serum potassium

Continued.

NSAIDs	Other drugs	Effect of mechanism
Aceclofenac	Dalteparin	Aceclofenac and dalteparin both increase anti coagulation
	Prednisolone	Risk of gastrointestinal ulceration
	Propranolol	Aceclofenac decrease prostaglandin synthesis, both increase serum potassium
Piroxicam	Ciprofloxacin	Increased risk of CNS stimulation and seizures
	Propranolol	Piroxicam decrease prostaglandin synthesis, both increase serum potassium

DISCUSSION

Identification of specific risk factors and precise determination of NSAID-induced adverse effects will help us to develop appropriate management strategies for patients taking NSAID therapy, leading to maximum potential benefits and minimum adverse events. A total of 175 subjects were enrolled during the 6-month study period. The study subjects were enrolled based on the designed inclusion and exclusion criteria. Within the study population, most of the patients prescribed NSAIDS belonged to the age category of >60 years (44.6%), followed by the age category of 50-59 (20.6%). This study is inconsistent with another study done by Inamdar et al in which NSAIDS were most commonly prescribed for the below 60-year age group (47%), followed by the 61-70-year age group (21%).[14] Among the study population, male patients (59.4%) were more predominant than female patients (40.6%). This is inconsistent with the study conducted by Shidhani et al in which most of the patients were females (54%).[1] Within our study population, NSAIDs were predominantly prescribed for pain (n=135), followed by inflammation (n=27) and fever (n=13). This is consistent with the study conducted by Kumar et al where NSAIDs are mostly prescribed for pain, followed by inflammation.[15] Our study reveals that, during the therapy, 65.7% of patients were prescribed 1-NSAID, followed by 28.6% on 2-NSAIDs and 5.7% patients were given 3-NSAIDs. This is inconsistent with the study conducted by Gul et al which concluded that 2-NSAIDs (69%) were mostly prescribed.[16] Among our study population, paracetamol (90.85%) was the most prescribed NSAID, followed by diclofenac (15.42%). This is consistent with the study conducted by Vaishnavi et al in which the study concluded paracetamol (27.03%) was prescribed more, followed by diclofenac.[17] Among the study population, NSAIDs were mostly administered via the oral route (78.28%) and followed by the parenteral route (38.8%). This is similar to the findings reported in a study conducted by Bahreini et al where oral therapy was found to be the most commonly used route of administration, with a frequency of 47.34% followed by parenteral therapy with 31.02%.[18] Within our 175 subjects, most of the patients were under monotherapy (68.6%), although some patients were prescribed combination therapy (31.4%). Contrast findings were found in a study conducted by Rafaniello et al where combination therapy (34.1%) was more prescribed than monotherapy (27%).[19] NSAIDs are co-prescribed with PPI, H2 receptor antagonists or antacids to prevent NSAID-induced GI adverse effects. During the study period, pantoprazole (81.1%) was given more commonly as a gastroprotective agent than ranitidine (3.42%) and sucralfate (1.2%), which is similar to a study conducted by Ravi Teja et al in which pantoprazole was mostly prescribed as a gastroprotective agent (75.83%).[20] Polypharmacy is defined as the concurrent use of several drugs. An increase in life expectancy has brought about an increase in the number of certain chronic illnesses, which involve hospital admissions, multiple medications and their associated ADR's. Achieving an ideal balance between risks and benefits becomes more challenging and thus increases the risk of ADRs. During our study period, ADR was found to be caused by diclofenac. The risk factor identified in particular patients was older age and polypharmacy. In our study, 36.57% patients were prescribed 10-15 drugs, followed by 30.85% were prescribed 5-10 drugs. Similar findings were reported in study conducted by Lipworth et al which concluded that more than 7 drugs were prescribed in 153 patients.[21] Older age, polypharmacy, anticoagulant use, corticosteroid use, alcohol use, previous gastrointestinal problem, bleeding disorder and no co-administration of gastric protective agent, comorbid disease are risk factors contributing to adverse reaction. Our findings revealed that polypharmacy (64.57%) and older age (38.84%) were the most prevalent risk factors. This is consistent with the study conducted by Lee et al which showed that the most prevalent risk factor was older age and polypharmacy.[22]

This study has some limitations. The short duration of six months limited the detection of long-term or delayed NSAID-induced adverse effects, such as renal or gastrointestinal complications. The sample size of 175, while statistically justified, may not be large enough to detect rare or severe ADRs. Post-discharge follow-up was not conducted, potentially excluding late-onset adverse events. The exclusion of pediatric patients, pregnant/lactating women, and patients with HIV or communicable diseases limits the applicability of findings to these groups. Finally, the study focused only on inpatients, leaving out outpatient NSAID use, which is often unsupervised and more prone to irrational use. Broader, multi-centred studies are needed for more comprehensive risk assessment.

CONCLUSION

This prospective study assessed the prevalence of risk factors associated with NSAID-induced adverse effects among 175 inpatients at a tertiary care hospital. The most commonly prescribed NSAID was paracetamol, and oral administration was predominant. Key risk factors identified were polypharmacy (64.57%) and older age (44.6%). Most patients (86%) received gastroprotective

agents, indicating preventive efforts in prescribing practices. Only one ADR, generalised pruritus due to diclofenac, was reported, assessed as "probable" (WHO-UMC) and "mild" (Hartwig scale). Although ADR occurrence was low, the findings highlight the importance of continuous monitoring and individualized risk assessment during NSAID therapy. The study emphasizes the role of clinical pharmacists in identifying high-risk patients, ensuring rational NSAID use, and improving therapeutic outcomes. Early identification of risk factors can minimize adverse events and enhance patient safety in hospital settings.

ACKNOWLEDGEMENTS

Authors would like to thanks to chairman, Prof. K. R. Arumugam, M. Pharm, for providing the necessary facilities and his unwavering support for education.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee Vijaya group of hospital-IECVBMHR/LTR/2022/030.

REFERENCES

1. Shidhani A, Rawahi N, Rawahi A. Non-steroidal anti-inflammatory drugs (NSAIDs) use in primary health care centers: A Clinical Audit. Oman Med J. 2015;30(5):366-74.
2. Tripathi KD. Essentials of medical pharmacology 9th ed. New Delhi: Jaypee Brothers medical publishers (p) Ltd. 2019;209-26.
3. Tomic M, Micov A, Pecikoza U, Stepanovic-Petrovic R. Clinical uses of nonsteroidal anti-inflammatory drugs (NSAIDs) and potential benefits of NSAIDs modified-release preparations. In Microsized and nanosized carriers for nonsteroidal anti-inflammatory drugs. 2017;1(1):1-29.
4. Laine L, Curtis SP, Cryer B, Kaur A, Cannon CP. Risk factors for NSAID associated upper GI clinical events in a long-term prospective study of 34,701 arthritis patients. Aliment Pharmacol Therapeut. 2010;32(10):1240-8.
5. Peck TE, Hill SA. Pharmacology for anaesthesia and intensive care, 4th edition. Cambridge University Press. 2014;1-355.
6. Jame DS. The multisystem adverse effects of NSAID therapy. J Osteopath Med. 1999;99(11):1-7.
7. Harirforoosh S, Asghar W, Jamali F. Adverse effects of Non-steroidal anti-inflammatory drugs: an update of gastrointestinal, cardiovascular and renal complications. J Pharmacy Pharmaceut Sci. 2013;16(5):821-47.
8. Lee SH, Han CD, Yang IH, Ha CW. Prescription pattern of NSAIDs and the prevalence of NSAID-induced gastrointestinal risk factors of orthopaedic patients in clinical practice in Korea. J Korean Med Sci. 2011;26(4):561-78.
9. Bardou M, Barkun AN. Preventing the gastrointestinal adverse effects of nonsteroidal anti-inflammatory drugs: from risk factor identification to risk factor intervention. Joint Bone Spine. 2010;77(1):6-12.
10. Bjorkman DJ. Current status of Non-steroidal anti-inflammatory drug (NSAID) use in the United States: risk factors and frequency of complications. Am J Med. 1999;107(6):3-8.
11. Weinblatt ME. Drug liberations With Non-Steroidal Anti-Inflammatory Drugs (NSAIDs). Scandinavian J Rheumatol. 1989;18(1):7-10.
12. Parthasarathi G. A textbook of clinical pharmacy practice 2nd ed. University Press (India) private limited. 2020;104-22.
13. Pirmohamed M, Breckenridge AM, Kitteringham NR, Park BK. Adverse drug reactions. BMJ. 1998;316(7140):1295-8.
14. Inamdar Z, Sarwamangala SN, Padmavati V, Dalal VI, Bali PN. Prescription Pattern Analysis of Non-Steroidal Anti-Inflammatory Drugs In a tertiary care hospital. Asian J Pharmaceut Sci Clin Res. 2022;15(7):177-80.
15. Kumar S, Thakur PK, Sowmya K, Priyanka S. Evaluation of prescribing pattern of NSAIDs in south Indian teaching hospital. J Chitwan Med College. 2016;6(4):54-8.
16. Gul S, Ayub M. Prevalence of prescribing pattern of more than one NSAID in Pakistan. J SciInnov Res. 2014;3(2):148-54.
17. Vaishnavi PR, Gaikwad N, Dhaneria SP. Assessment of nonsteroidal anti-inflammatory drug use pattern using world health organization indicators: A cross-sectional study in a tertiary care teaching hospital of Chhattisgarh. Indian J Pharmacol. 2017;49(6):445-53.
18. Bahreini A, Koneri R. Prescription Pattern Analysis of Nonsteroidal Anti- Inflammatory Drugs in the Southeastern Karnataka Population, India. Arch Pharma Pract. 2020;11(S1):116-9.
19. Rafaniello C, Ferrajolo C, Sullo MG, Sessa M, Sportiello L. Risk of gastrointestinal complications associated to NSAIDs, low-dose aspirin and their combinations: Results of a pharmacovigilance reporting system. Pharmacolog Res. 2016;104(1):108-14.
20. Ravi Teja P, Sandari B, Priya L, Kishore R. A Clinical Study on prescribing pattern of NSAIDs and Assessment of drug interactions. ACTA Scientific Pharmaceut Sci. 2020;4(5):84-7.
21. Lipworth L, Abdel-Kader K, Morse J, Stewart TG, Kabagambe EK, Parr SK, et al. High prevalence of Non-steroidal anti-inflammatory drug use among acute kidney injury survivors in the southern community cohort study. BMC Nephrol. 2016;17(1):1-9.
22. Lee SH, Han CD, Yang IH, Ha C. Prescription pattern of NSAID-induced gastrointestinal risk factors of orthopaedic patients in clinical practice in Korea. J Kor Med Sci. 2011;26(4):561-7.

Cite this article as: Ashwini J, Benisha XC, Hemalatha K, Sangeetha B. A study on the prevalence of risk factors associated with non-steroidal anti-inflammatory drugs-induced adverse effects among inpatients at a tertiary care hospital. Int J Basic Clin Pharmacol 2025;14:791-6.