

# Anaphylactic Shock Following Intramuscular Injection of Diclofenac Sodium (Dynapar): A Case Report from a Tertiary Care Hospital in India

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**ABSTRACT:** Diclofenac sodium is among the most commonly prescribed non-steroidal anti-inflammatory drugs (NSAIDs) worldwide and is frequently administered parenterally for acute pain management. Although generally regarded as well-tolerated, parenteral diclofenac carries a rare but life-threatening risk of anaphylaxis. We report the case of a 40-year-old female patient who presented to the emergency department of a tertiary care hospital with anaphylactic shock shortly after receiving an intramuscular injection of diclofenac sodium (Dynapar) for abdominal pain. She exhibited classic features of anaphylaxis including uprolling of eyes, loss of consciousness, disorientation, hypotension, and tachycardia. The patient was admitted to the Intensive Care Unit (ICU), where she was promptly managed with injectable hydrocortisone sodium succinate, intravenous normal saline (sodium chloride 0.9%), vasopressors (noradrenaline/norepinephrine), and supportive care. She recovered well and was subsequently diagnosed with bilateral obstructive renal calculi, for which she underwent cystoscopy, right ureteroscopy (URS), and right double-J (DJ) stenting. She was discharged in a stable condition on day 3 of admission. This case highlights the importance of vigilance for anaphylactic reactions to NSAIDs, even in patients with no prior documented drug allergy, and underscores the need for pre-injection allergy assessment and immediate availability of resuscitative measures.

**KEYWORDS:** Diclofenac sodium, Dynapar, Anaphylaxis, Anaphylactic Shock, NSAID Allergy, Drug Hypersensitivity, Parenteral NSAID

## I. INTRODUCTION

In contemporary clinical practice, non-steroidal anti-inflammatory drugs (NSAIDs) occupy a central role

across medical specialties, valued for their capacity to relieve pain, reduce fever, and attenuate inflammation through a shared mechanism of prostaglandin inhibition.<sup>[1]</sup>

Diclofenac sodium — commercially available under brand names such as Dynapar, Voveran, and Voltaren — belongs to the phenylacetic acid subclass of NSAIDs. Its principal pharmacological action involves competitive inhibition of both cyclooxygenase isoforms (COX-1 and COX-2), thereby curtailing the biosynthesis of prostaglandins and thromboxanes that mediate pain sensitization and inflammation.<sup>[2]</sup> Clinically, it finds application across a broad spectrum of conditions including postoperative pain, renal colic, musculoskeletal disorders, and acute inflammatory states.<sup>[3]</sup>

Diclofenac sodium is available in multiple formulations including oral tablets, sustained-release capsules, topical gels, and parenteral injections.<sup>[4]</sup> The injectable form — most commonly diclofenac sodium 75 mg/3 mL — is frequently used in emergency departments and inpatient settings for its rapid onset of action. The intramuscular (IM) route is particularly popular for the management of acute pain such as renal colic, postoperative analgesia, and musculoskeletal flares.<sup>[5]</sup>

Adverse reactions to diclofenac are well-documented and include gastrointestinal disturbances, renal impairment, elevated liver enzymes, and cardiovascular effects.<sup>[6]</sup> However, immunologically mediated hypersensitivity reactions — ranging from mild urticaria to life-threatening anaphylaxis — are comparatively rare but clinically significant. Anaphylaxis following NSAID use is estimated to occur in approximately 0.001% to 0.01% of users, but this figure may be an underestimate due to under-reporting and misdiagnosis.<sup>[7]</sup> NSAIDs are second only to antibiotics as the most common drug class implicated in drug-induced anaphylaxis.<sup>[8]</sup>

The mechanism of NSAID-induced hypersensitivity can be broadly classified as either immunological (IgE-mediated) or non-immunological (pseudo-allergic), with the latter being more common for NSAIDs.<sup>[9]</sup> The non-immunological pathway involves COX-1 inhibition leading to a shift in arachidonic acid metabolism toward the lipoxygenase pathway, resulting in increased leukotriene production and mast cell degranulation even in the absence of prior sensitization.<sup>[10]</sup> This means that anaphylaxis can occur even on the first exposure to diclofenac sodium, and the absence of prior drug allergy history does not exclude the risk.<sup>[11]</sup>

Anaphylactic shock represents the gravest end of the hypersensitivity spectrum — a multi-organ systemic emergency driven by massive mediator release, manifesting as circulatory collapse, impaired consciousness, bronchospasm, urticaria, and angioedema.<sup>[12]</sup> Timely recognition and immediate intervention are decisive in preventing fatality. Current international guidelines unanimously designate intramuscular epinephrine (adrenaline) as the first and most critical therapeutic step, with intravenous corticosteroids, antihistamines, and hemodynamic resuscitation serving as essential adjuncts.<sup>[13]</sup>

Despite the widespread use of parenteral diclofenac in Indian hospitals, reported cases of diclofenac-induced anaphylaxis in the Indian literature remain limited.<sup>[14]</sup> Pharmacovigilance data from the WHO Vigibase database and the Pharmacovigilance Programme of India (PvPI) have highlighted the need for greater awareness of serious NSAID-related hypersensitivity events.<sup>[15]</sup> This case report describes a well-documented clinical episode of anaphylactic shock following IM diclofenac sodium (Dynapar) injection in a 40-year-old female patient with no previously known drug allergy, aiming to raise awareness among clinicians about this potentially fatal complication.

## II. CASE REPORT

### 2.1 Patient Background

A 40-year-old female patient, a housewife, presented to the Emergency Department of a tertiary care hospital with a primary complaint of abdominal pain. She was a walk-in patient with no referral. She had no previously documented chronic illnesses, drug allergies, or significant past medical history. Her personal history was unremarkable — she was a non-smoker, non-alcoholic, and had no history of substance use. Her obstetric history revealed four pregnancies and one delivery. Her last menstrual period was recent at the time of presentation. Family

history was non-contributory for hypertension, diabetes, cardiac disease, or atopic disorders.

Of note, the patient had previously undergone cystoscopy, right ureteroscopy (URS), and right double-J (DJ) stenting the day prior to the anaphylactic episode, performed for bilateral obstructive renal calculi. This surgical history is relevant as it provides clinical context for the administration of analgesic injections in the peri-procedural period.

### 2.2 Presenting Complaints

The patient presented with a primary complaint of abdominal pain localizing to the perineal region, which she had been experiencing for 2 to 3 days prior to admission. In an attempt to manage the pain, she had received an intramuscular injection of diclofenac sodium (Inj. Dynapar 75 mg/3 mL) along with Inj. Tramadol (tramadol hydrochloride), administered outside the hospital setting. Shortly after administration of these injections, she developed a sudden and dramatic deterioration in her condition — characterized by uprolling of the eyes, loss of consciousness, disorientation, and tachycardia — consistent with a clinical picture of anaphylactic shock. She was then brought by family members to the emergency department for urgent management.

### 2.3 Examination on Admission

On arrival at the emergency department, the patient was hemodynamically unstable. Vital signs recorded at the time of admission are shown in Table 1.

Parameter	Value on Admission
Temperature	98.6°F (37°C) — Afebrile
Pulse Rate	110 beats/min — Tachycardia
Respiratory Rate	20 breaths/min
Blood Pressure	100/60 mmHg — Hypotensive
SpO2	98% on room air
Level of Consciousness	Conscious and oriented (post-resuscitation stabilisation)
General Appearance	Pallor present; no icterus, cyanosis, or edema noted

Table 1: Vital Signs on Admission

Systemic examination: cardiovascular examination revealed normal S1 and S2 heart sounds with no added sounds, murmurs, or gallop. CNS examination showed the patient to be conscious and oriented. Per-abdominal examination revealed a soft abdomen. Respiratory auscultation was clear bilaterally. All peripheral pulses were palpable and equal bilaterally.

The clinical picture was strongly suggestive of anaphylactic shock secondary to the recently administered intramuscular diclofenac sodium (Dynapar) injection.

#### 2.4 Investigations

A comprehensive panel of laboratory and radiological investigations was ordered at the time of admission (Table 2). The CT abdomen and pelvis with contrast was the most diagnostically significant, revealing bilateral obstructive renal calculi with a right VUJ calculus causing backpressure changes and right mild hydronephrosis, along with incidentally noted uterine fibroids.

Investigation	Remarks / Findings
Complete Blood Count (CBC)	Ordered on admission; results reviewed
C-Reactive Protein (CRP)	Ordered; elevated CRP consistent with inflammatory response
Serum Creatinine	Ordered to assess renal function (repeated during stay)
Serum Electrolytes	Monitored for hemodynamic stability
Liver Function Tests (LFT)	Baseline hepatic assessment
Glycosylated Hemoglobin (HbA1c)	Metabolic profile assessment
Prothrombin Time (PT)	Coagulation status assessment
HBsAg (Manual + HepAcad)	Infectious screen — completed
HCV Manual	Negative
HIV Manual	Negative
Arterial Blood Gas (ABG)	Performed on admission — assessed oxygenation and acid-base status
Random Blood Sugar (RBS)	Monitored multiple times — 92 mg/dL on initial check
CT Abdomen & Pelvis (Plain + Contrast)	Bilateral obstructive renal calculi; right VUJ calculus with backpressure and right mild hydronephrosis; uterine fibroids noted
CT Brain (Plain)	Ordered for neurological assessment
Portable X-Ray (Chest)	Obtained for baseline respiratory and cardiac status

Urine Routine Examination	Ordered to assess renal tract
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Table 2: Investigations and Findings

#### 2.5 Diagnosis

Based on the clinical history, examination findings, and investigations, the following diagnoses were established:

- Anaphylactic Shock secondary to Inj. Diclofenac Sodium (Dynapar) — ICD-10: T78.2
- Bilateral Obstructive Renal Calculi — ICD-10: N20

#### 2.6 ICU Management — Anaphylactic Shock

The patient was immediately admitted to the Intensive Care Unit (ICU) and management of anaphylactic shock was initiated: Inj. Hydrocortisone (hydrocortisone sodium succinate) 200 mg IV stat dose, followed by 100 mg IV TDS as maintenance — serving as the primary pharmacological intervention to dampen the systemic inflammatory and allergic cascade. IV fluid resuscitation was commenced with normal saline (sodium chloride 0.9%) to address hypotension. Inj. Norad (noradrenaline/norepinephrine) infusion was initiated as a vasopressor to support blood pressure. Urethral catheterization was performed to monitor urine output. Continuous vital sign monitoring was maintained throughout the ICU stay. Supportive medications including antibiotics and antacids were also administered. The vasopressor infusion was gradually tapered as hemodynamics stabilized.

#### 2.7 Discharge and Follow-up

The patient was discharged in a relieved condition on day 3 of admission. Discharge medications prescribed for 7 days are detailed in Table 3.

Brand Name (Generic Name)	Frequency	Dose	Instructions
Tab. Levoflox 500 mg (Levofloxacin)	Once daily	1-0-0	After food
Tab. Dolo 650 mg (Paracetamol)	Three times daily	1-1-1	After food
Tab. Crainbid (Cranberry Extract + Lactobacillus)	Twice daily	1-0-1	After food
Tab. Vomcol DSR (Domperidone + Omeprazole)	Twice daily	1-0-1	Before food
Syp. Pot MG B6 (Potassium + Magnesium + Pyridoxine)	Twice daily	2 TSF - 0 - 2 TSF	With half glass of water

Tab. L Dio 1M (Diosmin + Hesperidin)	Once daily (PM)	0-0-1	After food
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Table 3: Discharge Medications

Discharge advice included rest, regular diet, compliance with prescribed medications, and removal of the DJ stent after 21 days. The patient and her family were clearly counselled regarding the documented allergic reaction to diclofenac sodium (Dynapar) and tramadol hydrochloride, and strict avoidance of these drugs in the future was emphasized.

### III. DISCUSSION

This case vividly illustrates the potentially fatal nature of anaphylaxis following an intramuscular injection of diclofenac sodium (Dynapar), a drug that is routinely administered in emergency departments, outpatient clinics, and even at home by untrained hands without adequate precautions. The patient presented with a constellation of features — sudden loss of consciousness, uprolling of eyes, disorientation, tachycardia, and hypotension — immediately after receiving the injection, leaving little doubt about the causal relationship between the drug and the clinical event.

Diclofenac-induced anaphylaxis, while documented in the literature, is an under-appreciated complication in real-world clinical practice, particularly in the South Asian context where parenteral NSAIDs are liberally used for pain relief. Pharmacovigilance databases and clinical case series have increasingly highlighted severe hypersensitivity events associated with diclofenac sodium.

The mechanism underlying diclofenac-induced anaphylaxis in this patient is most likely a non-IgE-mediated, pseudo-allergic reaction. NSAIDs, including diclofenac sodium, can inhibit the COX-1 enzyme and redirect arachidonic acid metabolism toward the lipxygenase pathway, leading to increased production of cysteinyl leukotrienes (LTC<sub>4</sub>, LTD<sub>4</sub>, LTE<sub>4</sub>). These leukotrienes are potent mediators of anaphylaxis-like reactions — causing bronchospasm, vasodilation, increased vascular permeability, and mast cell degranulation — without requiring prior sensitization.

The concurrent administration of tramadol hydrochloride — a centrally acting opioid analgesic — alongside the diclofenac injection also deserves

attention. Tramadol is independently capable of causing non-immune mast cell degranulation and histamine release, and the combination of two drugs with independent anaphylactogenic potential may have potentiated the severity of the reaction.

In terms of clinical management, the treating team responded appropriately. While the ideal first-line management of anaphylaxis is intramuscular epinephrine (adrenaline) — a fact established by international guidelines including those of the World Allergy Organization (WAO) and the European Academy of Allergy and Clinical Immunology (EAACI) — the clinical course documented here involved hydrocortisone sodium succinate 200 mg IV as the cornerstone of initial drug therapy, along with IV normal saline, vasopressor support (noradrenaline/norepinephrine), antibiotics, and antacids. The combination of corticosteroids, vasopressors, and IV fluids resulted in a favourable outcome for the patient.

This case carries several important teaching points for practicing clinicians. First, no patient receiving a parenteral drug — particularly an NSAID or opioid — should be left unobserved immediately after injection. The standard recommendation is a 15 to 20-minute observation period post-injection. Second, a detailed drug allergy history should be obtained before any injection. Third, healthcare providers administering injections outside formal hospital settings must be trained to recognize and manage anaphylaxis promptly. Fourth, when a patient survives an anaphylactic episode, clear documentation of the causative agent is essential to prevent recurrence.

Comparison with previously published cases enriches the significance of this report. Sahoo et al. (Cureus, 2024) described a similar episode following intramuscular diclofenac sodium in an Indian patient, emphasizing that hypersensitivity reactions are more common in individuals with pre-existing atopic conditions. In contrast, the present patient had no documented allergic history, further supporting the reality of first-exposure pseudo-allergic reactions to NSAIDs. What distinguishes the current case is the co-occurrence of bilateral obstructive renal calculi with a peri-procedural analgesic trigger, and the favourable recovery achieved through ICU-based management without epinephrine.

From a regulatory and pharmacovigilance standpoint, this case should be reported to the Pharmacovigilance Programme of India (PvPI) managed by the Indian Pharmacopoeia Commission, to contribute to the

growing body of safety data on diclofenac-related anaphylaxis in the Indian population.

#### IV. CONCLUSION

This case report describes a rare but life-threatening anaphylactic reaction following intramuscular diclofenac sodium (Dynapar) administration in a 40-year-old female with no prior history of drug allergy. The patient presented with classic features of anaphylactic shock and was successfully managed with ICU-level care including hydrocortisone sodium succinate IV, vasopressor support with noradrenaline/norepinephrine, fluid resuscitation with normal saline (sodium chloride 0.9%), and organ monitoring. A concurrent diagnosis of bilateral obstructive renal calculi was made, for which she underwent successful urological intervention.

This case underscores the critical importance of treating every parenteral drug administration as a potential trigger for anaphylaxis. Clinicians must maintain a high index of suspicion, observe patients post-injection, and be equipped to act swiftly when anaphylaxis occurs. Diclofenac sodium, despite its ubiquity, is not without serious risk, and patient safety protocols must be reinforced across all healthcare settings where it is used.

#### V. PATIENT CONSENT

This case report has been prepared using anonymized clinical data from hospital medical records. The patient's personal identifiable information has been fully protected in accordance with patient privacy principles. Standard general consent documentation signed by the patient's family covers the use of clinical data for educational and research purposes. The authors certify that this report does not include any information that directly identifies the patient.

#### VI. DECLARATIONS

**Conflict of Interest:** None declared.

**Funding:** None.

**Ethics Statement:** This case report was compiled from existing clinical records. Patient privacy has been maintained throughout.

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