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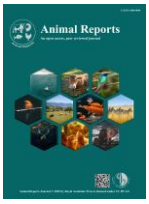

Type: Review

Ecotoxicological effects of emerging pollutants (nanomaterials and microplastics) on fish biology

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Abstract: Emerging contaminants, including heavy metals, nanomaterials, microplastics, and industrial chemicals pose significant threats to aquatic ecosystems and fish health. The emerging pollutants are majorly produced during industrial processes, urbanization, and mining. This review study compiled current knowledge on the ecotoxicological effects of these pollutants on fish biology, encompassing physiological, behavioral, and molecular responses. Innovative methodologies have been used to comprehensively assess toxicological responses, such as high-throughput omics, including transcriptomics, proteomics, and metabolomics can elucidate molecular-level disruptions. In addition, advanced bioimaging techniques e.g., micro-CT scanning can assess internal tissue damage caused by pollutants non-invasively. Furthermore, eco-epidemiological models integrating field and laboratory data will enhance predictive risk assessments. Regulatory frameworks must evolve to incorporate sublethal and chronic toxicity endpoints, ensuring more comprehensive environmental protection strategies. Collaborative efforts among researchers, policymakers, and industries are essential to mitigate contamination sources and develop sustainable remediation approaches for aquatic ecosystems.
Keywords: Emerging Pollutants, Nanotechnology, Microplastics, Fish biology

1 Introduction

The rapid industrialization and urbanization during recent decades have led to the widespread release of emerging contaminants into aquatic ecosystems, posing significant threats to aquatic organisms (Bashir et al., 2020). Among these pollutants, heavy metals (e.g., mercury, lead, cadmium) (Narwal and Kakakhel, 2025; Singh et al., 2023), nanoparticles (e.g., TiO_2, Ag)

(Kakakhel et al., 2023b), organic contaminants (e.g., pesticides, pharmaceuticals) (Gwenzi and Chaukura, 2018), and MPs have become pervasive in freshwater and marine environments (Narwal et al., 2024; Narwal and Katyal, 2024). These contaminants enter water bodies through industrial effluents, agricultural runoff, and improper waste disposal (Singh et al., 2022), where they accumulate and exert toxic effects on aquatic life. Fish, as key bioindicators of aquatic ecosystem health, are particularly vulnerable due to their direct exposure to contaminated water and sediment (Okwuosa et al., 2019). The bioaccumulation and biomagnification of these pollutants through the food web further exacerbate their ecological impacts, making it imperative to understand their effects on fish physiology, behavior, and survival (Oros, 2025).

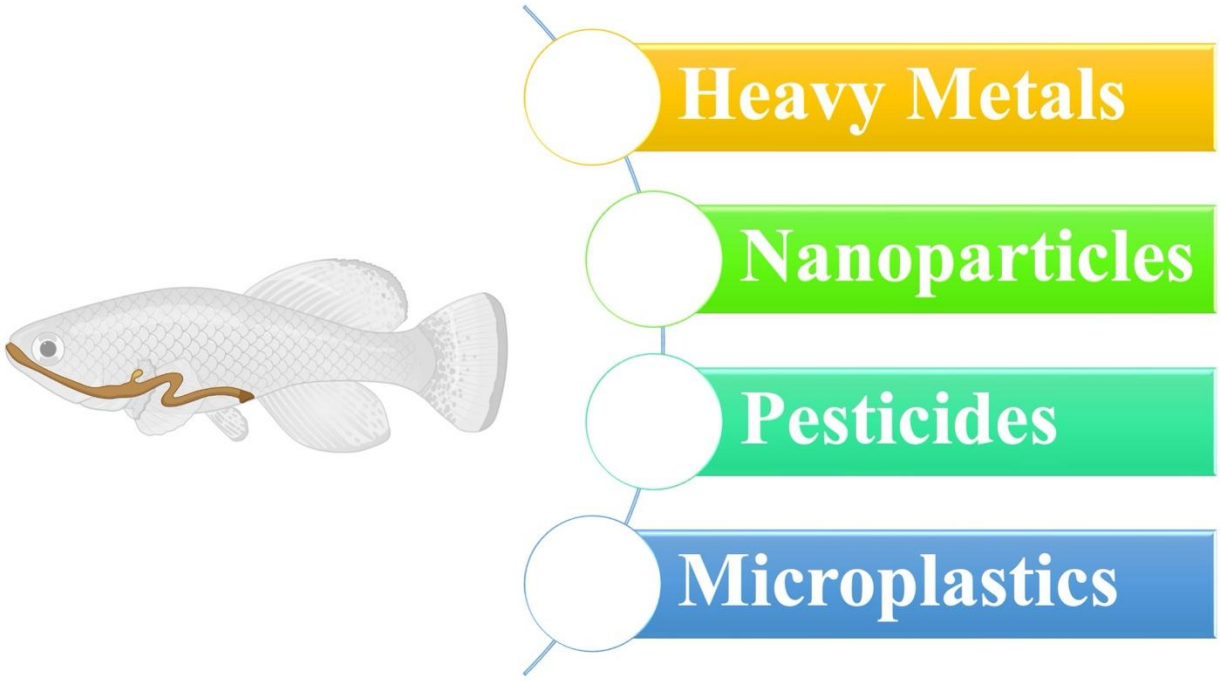

Fig. 1. The list and existence of emerging pollutants, which particularly expose the fish in aquatic ecosystems.

The toxicological effects of emerging contaminants on fish are multifaceted, encompassing biochemical, physiological, and behavioral disruptions (Scott and Sloman, 2004). Heavy metals, for instance, interfere with enzymatic activity, impair osmoregulation, and cause oxidative stress, leading to cellular damage and compromised immune function (Notariale et al., 2021). Nanoparticles, due to their small size and high reactivity (Kakakhel et al., 2021), can penetrate biological membranes, disrupt cellular processes, and induce inflammatory responses (Roy et al., 2014). Organic contaminants, such as endocrine-disrupting chemicals, alter hormone signaling and reproductive success (Amir et al., 2021), while MPs cause physical blockages, leach toxic additives, and serve as vectors for other pollutants (Tumwesigye et al., 2023). These contaminants often co-occur in aquatic environments, leading to complex interactions that may amplify their toxicities (Ding et al., 2022). Despite growing awareness of their hazards, the combined effects of these pollutants on fish remain poorly understood, highlighting the need for comprehensive reviews to synthesize existing knowledge and identify research gaps.

This review aims to consolidate current research on the effects of heavy metals, nanoparticles, organic contaminants, and MPs on fish, with a focus on their mechanisms of toxicity, ecological consequences, and potential mitigation strategies. By examining the latest findings, we seek to elucidate the pathways through which these contaminants affect fish health, from molecular and cellular levels to population and community dynamics. Furthermore, we discuss the implications of these effects for aquatic ecosystem stability and human health, particularly in the context of fisheries and aquaculture. Ultimately, this review underscores the urgency of adopting integrated approaches to monitor, regulate, and remediate emerging contaminants, ensuring the sustainability of aquatic ecosystems and the resources they provide.

2 General toxicity mechanisms

Pollutants exert toxicity in fish through multiple biochemical and physiological pathways, often disrupting cellular integrity and metabolic homeostasis (Benedetti et al., 2015). Heavy metals such as mercury, lead, and cadmium interfere with essential enzymatic functions by binding to sulfhydryl groups in proteins, impairing processes like oxidative phosphorylation and DNA repair (Parida and Patel, 2023). These metals also generate reactive oxygen species (ROS), leading to oxidative stress that damages lipids, proteins, and nucleic acids (Ozougwu, 2016). Nanoparticles, due to their high surface-area-to-volume ratio, penetrate cell membranes and organelles, causing physical damage and inflammation (Abbasi et al., 2023). Additionally, their catalytic properties can exacerbate ROS production, further compromising cellular defenses (Abdal Dayem et al., 2017). Organic contaminants, including pesticides and industrial chemicals, often act as endocrine disruptors by mimicking or blocking hormonal signaling, leading to reproductive dysfunction and developmental abnormalities (Amir et al., 2021).

The gastrointestinal tract and gills serve as primary entry points for pollutants, where they induce localized and systemic effects (Curcio et al., 2022). MPs, for instance, cause physical abrasion in the gut, impair nutrient absorption, and leach plasticizers like phthalates, which disrupt endocrine function (Godswill and Godspel, 2019). Upon entering systemic circulation, pollutants target vital organs such as the liver, kidneys, and brain (Kodavanti et al., 2023). The liver, being the primary detoxification organ, becomes overwhelmed by chronic exposure, leading to lipid peroxidation, necrosis, and compromised detoxification capacity (Apte and Krishnamurthy, 2010). Similarly, nephrotoxic agents like cadmium accumulate in renal tissues, disrupting ion-regulation and excretory functions (Saket, 2022). Neurotoxic pollutants, including certain pesticides and methylmercury, cross the blood-brain barrier, interfering with neurotransmitter synthesis and synaptic transmission (Wang et al., 2023), which manifests as altered behavior and reduced survival fitness.

At the molecular level, pollutants often dysregulate gene expression and immune responses, exacerbating their toxic effects in fish (Das et al., 2025). For example, exposure to polycyclic aromatic hydrocarbons (PAHs) upregulates cytochrome P450 enzymes in fish (Bramatti et al., 2023), which metabolize these compounds into more reactive intermediates that form DNA adducts, increasing mutation rates (Vondráček and Machala, 2021). Concurrently, immunosuppressive pollutants reduce lymphocyte proliferation and antibody production, leaving fish vulnerable to infections (Poulsen and Escher, 2012). Chronic stress from pollutant exposure also elevates cortisol levels, which suppresses growth and reproduction by diverting energy toward stress responses (Angelier, 2022). These multifaceted mechanisms highlight the complex interplay

between pollutant chemistry, exposure duration, and fish physiology, underscoring the need for targeted strategies to mitigate their ecological and economic impacts on aquatic ecosystems.

3 Effects of emerging pollutants on fish

Emerging pollutants, including nanomaterials, microplastics, and industrial chemicals, pose significant threats to fish health (Subaramaniyam et al., 2023) by disrupting endocrine function, impairing reproduction, and causing oxidative stress (Sarasamma et al., 2020). These contaminants accumulate in aquatic ecosystems, leading to bioaccumulation in fish tissues and potential biomagnification through food chains (Ali and Khan, 2019; Zaheer Ud Din et al., 2023).

3.1 Effects of heavy metals on fish

Heavy metals exert toxic effects on fish through multiple physiological and biochemical pathways, disrupting normal metabolic functions and compromising overall health (Shahjahan et al., 2022). Metals such as mercury (Hg), lead (Pb), cadmium (Cd), and arsenic (As) accumulate in tissues, particularly the liver, kidneys, and gills (Garai et al., 2021), where they interfere with enzyme activity and cellular homeostasis. For instance, Hg binds to sulfhydryl groups in proteins, inhibiting critical enzymes involved in energy metabolism and oxidative stress defense (Kumar et al., 2025). For instance, in our previous study, *Labeo rohita* and *Tor putitora* were collected from Indus River Pakistan for the assessment of heavy metals, which showed drastic results i.e., in the liver, highest dry wt trace elements (μg/g) such as Cr (4.32), Pb (7.07), Zn (58.26), Cu (8.38), Mn (50.27), and Fe (83.9) for the *Labeo rohita*; and *Tor Putitora* has significantly greater accumulated concentration (Cr, Pb, Zn, Cu, Mn, Fe) in muscle and liver than did *Labeo rohita* species (Boota et al., 2024). Cd displaces essential ions like calcium and zinc, impairing osmoregulation and skeletal integrity (Jomova et al., 2022), which could impair the swimming performance of fish. Chronic exposure to these metals induces oxidative stress by generating reactive oxygen species (ROS), which damage lipids, proteins, and DNA, leading to cellular dysfunction, tissue necrosis, and increased susceptibility to disease (Jomova et al., 2023). Additionally, heavy metals disrupt endocrine function by mimicking or blocking hormonal signaling, resulting in reproductive impairments, and developmental abnormalities (Liu et al., 2023).

The ecological consequences of heavy metal toxicity extend beyond individual fish to entire aquatic ecosystems (Sharma et al., 2024). Bioaccumulation and biomagnification of metals through the food web pose risks to higher trophic levels, including piscivorous birds and mammals, including humans (Córdoba-Tovar et al., 2022). Behavioral alterations, such as reduced predator avoidance and foraging efficiency, further diminish survival and reproductive success, threatening biodiversity (Rohwäder and Jeltsch, 2022). Sublethal effects, including impaired growth, immunosuppression, and histopathological damage, often precede population declines, making heavy metals a persistent threat to aquatic life (Shaalan, 2024). Mitigation strategies, such as phytoremediation and water quality monitoring, are essential to reduce metal loads and safeguard fish health, particularly in industrially impacted water bodies (Emenike et al., 2022). Understanding these effects is critical for developing effective conservation and pollution control measures to protect aquatic ecosystems.

3.2 Effects of nanoparticles on fish

Metal-based nanoparticles, such as silver (AgNPs), titanium dioxide (TiO_2NPs), and zinc oxide (ZnONPs), exert diverse toxic effects on fish depending on their composition, size, and

exposure duration (Wang et al., 2024). AgNPs are particularly harmful due to their release of Ag^{+} ions, which disrupt ion-regulation in gills, impairing osmoregulation and oxygen uptake (Pereira et al., 2023). These nanoparticles also accumulate in the liver, inducing oxidative stress through the generation of reactive oxygen species (ROS) that damage cellular membranes and DNA (Padmanaban et al., 2023). TiO_2NPs, commonly found in sunscreens and coatings, adsorb onto gill surfaces, causing inflammation and reducing respiratory efficiency (Bevacqua et al., 2023). Additionally, their photocatalytic properties can amplify ROS production under UV light, exacerbating cellular damage (Yong et al., 2023). ZnONPs dissolve into Zn^{2+} ions in aquatic environments, leading to acute toxicity in fish by disrupting zinc-dependent metabolic pathways and causing histopathological changes in the kidneys and intestines (Mahjoubian et al., 2023). Moreover, the silver nanoparticles (AgNPs) have been reported to cause dysbiosis in fish (Chen et al., 2021), for instance, our previous study revealed the effects of blood-mediated AgNPs on *Cyprinus carpio* gut microbiome (Kakakhel et al., 2023a) by elevating phyla Tenericutes, Bacteroidetes, and Planctomycetes.

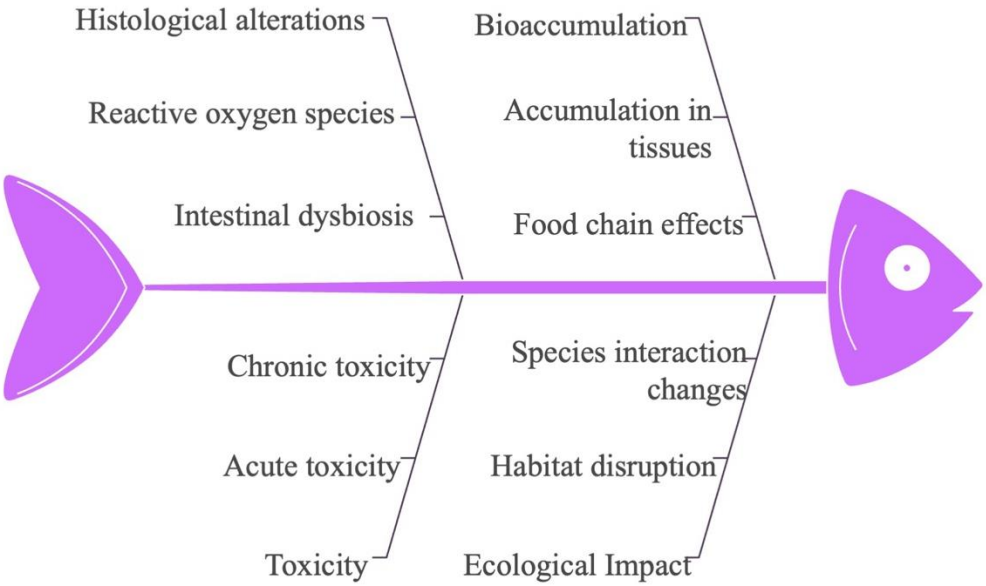

Figure 2. The possible effects of nanotechnology/nanoparticles on aquatic animals.

Carbon-based nanoparticles, including fullerenes (C60) and multi-walled carbon nanotubes (MWCNTs), exhibit unique toxicity profiles due to their structural properties (Ayanda et al., 2024). Fullerenes can penetrate cell membranes, inducing lipid peroxidation and mitochondrial dysfunction, while MWCNTs cause physical damage to gill epithelia and intestinal villi, impairing nutrient absorption (Shah et al., 2021). Polymeric nanoparticles, such as polystyrene nano-plastics, adsorb onto mucosal surfaces, altering gut microbiota composition and promoting dysbiosis (Meng et al., 2025). These particles also act as carriers for co-contaminants like heavy metals and organic pollutants, enhancing their bioavailability and toxicity (Deng et al., 2017). The size-dependent effects of nanoparticles further complicate their ecological risk assessment, as smaller particles (<50 nm) are more readily internalized, leading to greater cellular disruption (Zhang et al., 2018).

Collectively, these findings highlight the urgent need for stricter regulations on nanoparticle use and disposal to mitigate their adverse effects on aquatic ecosystems.

3.3 Effects of pesticides on fish

Pesticides exert a wide range of detrimental effects on fish, spanning from acute toxicity to chronic physiological disruptions (Stanley et al., 2016). Organophosphates and carbamates, commonly used insecticides, inhibit acetylcholinesterase activity in the nervous system, leading to impaired neurotransmission, convulsions, and mortality (Gupta et al., 2011). Pyrethroids, despite their low toxicity to mammals, are highly toxic to fish, causing hyperactivity, loss of equilibrium, and respiratory distress due to their action on sodium channels in nerve cells (Farag et al., 2021). Chronic exposure to sublethal concentrations of pesticides can result in suppressed immune function, reduced reproductive success, and altered behavior, ultimately diminishing population viability (Chmiel et al., 2020). Herbicides like glyphosate disrupt aquatic food webs by eliminating primary producers, indirectly affecting fish through habitat degradation and reduced food availability (Macneale et al., 2010). Furthermore, many pesticides bioaccumulate in fish tissues, posing risks to higher trophic levels, including humans who consume contaminated fish (Ray and Shaju, 2023).

The toxic mechanisms of pesticides in fish involve multiple biochemical pathways and physiological systems (Sabra and Mehana, 2015). Many pesticides induce oxidative stress by generating reactive oxygen species (ROS), which overwhelm antioxidant defenses and cause lipid peroxidation, protein denaturation, and DNA damage (Sule et al., 2022). Endocrine-disrupting pesticides, such as atrazine and DDT, interfere with hormone synthesis, receptor binding, and metabolism, resulting in reproductive abnormalities, skewed sex ratios, and developmental impairments (Zhao et al., 2024). Additionally, pesticides can damage gill epithelia, impairing osmoregulation and respiratory efficiency (Singh, 2014), while others target the gut microbiome, causing dysbiosis and compromising nutrient absorption and immune function (Lima et al., 2022). These multifaceted mechanisms highlight the complex and often synergistic ways in which pesticides impact fish health, highlighting the need for comprehensive risk assessments and the development of safer alternatives.

3.4 Effects of microplastic (MPs) toxicity on Fish

Microplastics exert multifaceted harmful effects on fish, spanning physical, biochemical, and ecological levels of organisms (Nguyen et al., 2023). Upon ingestion, MPs cause intestinal blockage and epithelial damage, impairing nutrient absorption and digestive efficiency (Ding et al., 2023). Smaller particles (<100 μm) translocate across the gut barrier, entering systemic circulation and accumulating in vital organs like the liver and kidneys (Schwarzfischer and Rogler, 2022). Chemical additives leached from MPs, including phthalates and bisphenol A, disrupt endocrine function by interfering with hormone synthesis and receptor binding, leading to reproductive abnormalities and developmental defects (Godswill and Godspel, 2019). Furthermore, MPs act as vectors for adsorbed pollutants such as heavy metals and persistent organic compounds (Narwal et al., 2024), enhancing their bioavailability and synergistic toxicity. Ecological consequences manifest through trophic transfer, as MPs bioaccumulate in food webs, potentially impacting predator species and ecosystem stability (Saikumar et al., 2024). The microplastic possible toxicity mechanisms on fish (Table 1).

Table 1. The possible toxicity mechanisms caused by different types of microplastic on fish.

Microplastic Type	Size Range	Primary Effects on Fish	Mechanisms of Toxicity	Key References
Primary MPs (Virgin pellets)	1–5 mm	- Intestinal blockage - Reduced feeding efficiency	Physical obstruction, false satiety	(Cole et al., 2013)
Secondary MPs (Fragments; phthalates)	0.1–1 mm	- Gut epithelial damage - Inflammation	Mechanical abrasion, leached additives	(Jabeen et al., 2017)
Fibers (Textile-derived)	10 μm–1 mm	- Gill filament entanglement - Reduced O_2 uptake	Physical clogging, mucus disruption	(Silva-Cavalcanti et al., 2017)
Microbeads (Cosmetic MPs)	0.1–1 mm	- Liver stress - Lipid metabolism disruption	Bioaccumulation, oxidative stress (ROS)	(Rochman et al., 2015)
Nanoplastics	<1 μm	- Cellular internalization - Genotoxicity	Mitochondrial dysfunction, DNA damage	(Pitt et al., 2018)
Weathered MPs (Aged)	Variable	- Higher pollutant adsorption - Endocrine disruption	Enhanced chemical leaching (PCBs, PAHs)	(Bhagat et al., 2021)

The toxicity mechanisms of MPs involve both direct physical damage and indirect oxidative stress pathways (Ding et al., 2023). Sharp-edged particles induce mucosal abrasions in the gastrointestinal tract, provoking inflammatory responses and increasing susceptibility to pathogens (Sutkar et al., 2025). At the cellular level, MPs generate reactive oxygen species (ROS) that overwhelm antioxidant defenses, resulting in lipid peroxidation, protein denaturation, and DNA damage (Das, 2023). Chronic exposure alters gut microbiota composition, reducing beneficial symbionts (e.g., *Lactobacillus*) while enriching opportunistic pathogens (e.g., *Aeromonas*), compromising immune function (Gauthier et al., 2019). Neurotoxic effects emerge through acetylcholinesterase inhibition, disrupting neurotransmitter balance and impairing predator avoidance behaviors (Audira et al., 2020). These interconnected mechanisms highlight the complex, multilevel impacts of MPs on fish health, underscoring the need for comprehensive risk assessments and mitigation strategies in aquatic environments.

4 Recommendations and Future Prospects

To advance understanding of ecotoxicological effects from emerging pollutants (nano-materials and microplastics) on fish biology, future research should prioritize long-term, multigenerational exposure studies to assess cumulative impacts on physiological, behavioral, and reproductive endpoints. Advanced omics approaches (transcriptomics, metabolomics, and metagenomics) should be integrated with traditional ecotoxicology to elucidate molecular mechanisms, including epigenetic modifications and microbiome dysbiosis. Standardized testing protocols are needed to evaluate complex pollutant mixtures, reflecting real-world exposure scenarios. Additionally, green nanotechnology and biodegradable alternatives must be developed to mitigate environmental release. Regulatory frameworks should incorporate sublethal and

chronic toxicity data to establish safer thresholds for aquatic ecosystems. Collaborative efforts among researchers, policymakers, and industries are essential to implement sustainable mitigation strategies**,** ensuring the conservation of fish biodiversity and aquaculture productivity in contaminated environments.

5 Conclusion

The ecotoxicological effects of emerging contaminants on fish biology highlight a growing threat to aquatic ecosystems and global biodiversity. Heavy metals, nanoparticles, organic pollutants, and MPs disrupt critical physiological processes in fish, including metabolism, reproduction, immune function, and neurological health. These contaminants induce oxidative stress, endocrine disruption, and cellular damage through diverse mechanisms, often compounded by bioaccumulation and biomagnification in aquatic food webs. MPs, for instance, not only cause physical harm but also act as vectors for adsorbed toxins, while nanoparticles penetrate tissues and trigger inflammatory responses. Similarly, persistent organic pollutants interfere with hormone signaling, and heavy metals impair enzymatic and osmoregulatory functions.

Mitigating these impacts requires integrated strategies, including stringent regulatory policies, advanced wastewater treatment technologies, and sustainable alternatives to high-risk contaminants. Future research should prioritize long-term exposure studies, multi-stressor interactions, and species-specific vulnerability assessments to refine ecological risk frameworks. By addressing these challenges, scientists, policymakers, and industries can collaborate to safeguard aquatic health and ensure the sustainability of fisheries and aquaculture in the face of escalating contaminant pressures.

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Authors Contribution:

Nishita Narwal wrote the initial draft; Zahid Anwar critically revised the manuscript; Mian Adnan Kakakhel illustration and supervision.

Informed consent:

Not applicable.

Conflict of interest statement

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this research article.

Data availability statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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