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Hematological alterations induced by chronic exposure to manganese chloride in wistar rats: a nanotechnology-based perspective on nanoscale hematotoxicity

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Manganese (Mn) is an essential trace element required for several physiological processes; however, chronic exposure can lead to systemic toxicity. The present study investigates the hematological alterations induced by prolonged oral exposure to manganese chloride ($MnCl_2$) in adult male Wistar rats. Twenty-four rats were divided into two groups: a control group receiving saline and an exposed group treated orally with $MnCl_2$ (4.79 mg $Mn \cdot L^{-1}$) for twelve weeks. Hematological parameters including red blood cells (RBCs), white blood cells (WBCs), thrombocytes, and mean corpuscular volume (MCV) were analyzed using an automated hematology analyzer. Blood smears were examined for morphological changes. Chronic Mn exposure significantly ($p < 0.05$) decreased RBC, WBC, and platelet counts, along with MCV values, indicating the development of microcytic anemia and thrombocytopenia. Blood smears revealed poikilocytosis, microcytosis, erythrocyte rouleaux formation, and acanthocytosis. Leukocyte abnormalities such as naked lymphocytes and the presence of blast-like cells were also observed. These findings demonstrate that chronic manganese exposure induces hematological disorders, possibly through iron antagonism and bone marrow suppression, emphasizing the need for stricter control of manganese exposure in occupational and environmental settings. In addition, nanotechnology-based approaches offer deeper insight into manganese-induced hematotoxicity by enabling nanoscale evaluation of oxidative stress, cellular damage, and inflammatory responses, which may support the development of advanced diagnostic and protective strategies.

Keywords: Manganese chloride; Hematology; Wistar rats; Anemia; Leukopenia.

1. INTRODUCTION

Manganese (Mn) is a trace element vital for normal mammalian physiology, acting as a cofactor for numerous enzymes including arginase, glutamine synthetase (GS), pyruvate carboxylase, and manganese superoxide dismutase (Mn-SOD) [1,2]. It is also essential for arginase and agmatinase, enzymes that play a role in the urea cycle within the brain [3,4]. Manganese deficiency can impair bone formation and reduce bone mineral density, whereas adequate supplementation supports skeletal development [5, 6]. As a transition metal, Mn is ubiquitous in the environment, and human exposure occurs through air, water, and dietary sources [7,8]. Industrial applications, such as the production of paints, dry cell batteries, glass, ceramics, and welding, represent major exposure routes for workers [9,10]. Chronic manganese exposure has been associated with neurotoxicity, hepatotoxicity, and hematological disturbances [11,12]. Given these concerns, experimental animal models, particularly Wistar rats, are widely employed due to their physiological resemblance to humans [13,14]. The present study aims to evaluate the hematological alterations induced by chronic oral exposure to manganese chloride in Wistar rats [15]. This work contributes to understanding the systemic effects of Mn toxicity and provides insight into potential mechanisms underlying Mn-induced hematological abnormalities [16,17]. Manganese homeostasis is tightly regulated under physiological conditions, as both deficiency and excess can compromise biological functions [18-20]. While moderate exposure is harmless, chronic or occupational exposure to elevated Mn levels has emerged as a significant public health concern [21,22]. Reports indicate rising Mn concentrations in groundwater, industrial effluents, and urban environments, making long-term exposure increasingly common [23,24]. Despite regulatory efforts, Mn remains one of the most frequently encountered heavy metals in occupational settings, emphasizing the importance of understanding its systemic toxicity [25,26].

Mn plays a central role in redox reactions, antioxidant defense, amino acid metabolism, and mitochondrial function [27,28]. It is required for the optimal performance of Mn-SOD, a vital enzyme that protects tissues from oxidative stress [29,30]. Mn also influences hematopoiesis indirectly through its involvement in enzymatic pathways necessary for cellular growth and metabolism [31,32]. However, excessive Mn disrupts metal homeostasis, particularly iron metabolism, which may impair erythropoiesis and immune function [33,34].

Although several studies have investigated Mn neurotoxicity, fewer have explored its impact on hematological parameters in a controlled experimental setting [35, 36]. Existing literature often focuses on acute exposure, leaving uncertainties regarding the cumulative effects of chronic, low-dose ingestion [37,38]. Moreover, many studies lack detailed morphological assessments of blood cells, limiting understanding of the mechanistic pathways underlying Mn-induced hematological dysfunction [39,40]. Important gaps persist regarding how chronic oral exposure to Mn affects red and white blood cells, platelet dynamics, and specific morphological alterations [41,42]. Clarifying these effects is essential, as hematological parameters serve as early biomarkers of systemic toxicity [43]. The need for experimental evidence is particularly urgent given the increasing environmental presence of Mn and its potential interaction with iron metabolism [44,45].

The present work provides new insights by combining quantitative hematological analysis with qualitative blood smear examinations to identify morphological abnormalities. Unlike many previous studies that focus primarily on neurobehavioral effects, this research emphasizes hematological alterations and provides evidence of microcytic anemia, leukocyte abnormalities, and platelet changes induced by chronic Mn exposure. The primary objective of this study is to investigate the hematological consequences of chronic oral exposure to manganese chloride in adult male Wistar rats. Specifically, the research aims to determine how prolonged Mn intake affects key hematological parameters, including red blood cell, white blood cell, and platelet counts, as well as mean corpuscular volume. Additionally, the study seeks to identify and characterize morphological alterations in blood cells through microscopic evaluation of stained blood smears. By examining both quantitative and qualitative hematological changes, this work aims to clarify potential mechanisms underlying Mn-induced hematotoxicity, particularly those related to iron antagonism and bone marrow suppression. The study intends to generate comprehensive data that contribute to understanding the systemic impact of chronic manganese exposure.

This study utilized twenty-four adult male Wistar rats divided into control and Mn-exposed groups. Animals received oral $MnCl_2$ (4.79 mg.Mn·L^{-1}) for twelve weeks to simulate chronic environmental exposure. Hematological indices—including RBCs, WBCs, platelets, and MCV—were measured using an automated analyzer. Blood smears were microscopically examined to detect morphological abnormalities. The scope is restricted to hematological effects and does not include biochemical or histopathological evaluation of other organs. This investigation enhances current understanding of Mn toxicity by documenting hematological impairments resulting from prolonged oral exposure. The findings may help establish biomarkers for Mn exposure, inform occupational health guidelines, and contribute to risk assessment models for environmental contamination [46-50]. Additionally, the study supports the hypothesis that Mn disrupts iron metabolism, offering a mechanistic basis for observed hematological changes. Recent advances in nanotechnology have improved the understanding of heavy-metal toxicity at the nanoscale level. Nanotoxicology focuses on how metal ions and nano-sized particles interact with blood cells, membranes, and hematopoietic tissues, leading to oxidative stress, inflammation, and structural damage. Applying nanotechnology concepts to manganese toxicity may

provide more precise interpretation of hematological alterations and early biomarkers of systemic exposure [51-55]. In this paper, we present experimental results demonstrating how chronic $MnCl_2$ exposure alters hematological parameters and blood cell morphology in Wistar rats. We discuss the implications of these findings in the context of Mn toxicity and highlight potential mechanisms through which Mn may induce anemia, leukopenia, and thrombocytopenia.

2. MATERIALS AND METHODS

2.1. Experimental animals

Adult male Wistar rats (280 ± 10 g) were used. Animals were housed in polypropylene cages under controlled environmental conditions (22 ± 1°C; 12 h light/dark cycle) with free access to standard rat chow (Aashirwad Food Industries, Chandigarh) and tap water. All animal procedures were conducted in accordance with Algerian legislation and the guidelines of the Algerian Association of Experimental Animal Sciences (AASEA), as described by [8].

2.2. Chemicals and reagents

Manganese chloride tetrahydrate ($MnCl_2 \cdot 4H_2O$) and May-Grünwald Giemsa stain were purchased from Sigma-Aldrich (Germany). All other reagents were of analytical grade. $MnCl_2$ solution was freshly prepared in sterile 0.9% saline [9].

2.3. Experimental design

Twenty-four rats were randomly assigned to two groups (n = 12 per group):
- Control group: Received sterile 0.9% saline (vehicle).
-Mn-exposed group: Received $MnCl_2$ orally at 4.79 mg.Mn·L^{-1} [10] for twelve weeks.
At the end of the exposure period, rats were fasted overnight and anesthetized using 10% chloral hydrate before sacrifice.

2.4. Blood collection and hematological analysis

Blood samples were collected from the inferior vena cava into EDTA-coated tubes. RBC, WBC, platelet counts, and MCV were determined using an automated hematology analyzer (Mindray BC-30s). Blood smears were prepared on glass slides, fixed in 70% ethanol, and stained using the May-Grünwald–Giemsa method [11]. Microscopic observations were made using a light microscope, and images were captured using a Samsung ST93 camera.

2.5. Statistical analysis

Data were expressed as mean ± standard error (SEM). Statistical analysis was performed using one-way ANOVA (SigmaPlot for Windows, version 11.0), followed by Tukey's multiple comparison test. Differences were considered statistically significant at $p < 0.05$.

2.6. Nanotechnology-based toxicological evaluation

Nanotechnology-based analytical concepts can be applied to enhance the assessment of manganese-induced hematotoxicity through nanoscale detection of oxidative injury, membrane alterations, and inflammatory responses. Advanced tools such as high-resolution microscopy, nano-biomarker

analysis, and nanoscale imaging may provide deeper characterization of blood cell damage and improve toxicological interpretation.

3. RESULTS AND DISCUSSION

Chronic Mn exposure produced significant hematological alterations. RBC counts (Figure 1A) significantly decreased ($p < 0.05$) in Mn-exposed rats compared to controls, suggesting anemia potentially resulting from erythrocyte destruction or bone marrow impairment [12, 56]. Similarly, MCV values (Figure 1B) were markedly reduced ($p < 0.05$), indicating microcytosis (RBC < 60 fL) [13, 47]. Platelet counts (Figure 1C) were also significantly lower, reflecting thrombocytopenia, which may arise from impaired thrombopoiesis or enhanced platelet aggregation [14, 57]. A pronounced leukopenia (Figure 1D) was observed ($p < 0.01$), indicating possible bone marrow suppression or immune dysregulation caused by Mn toxicity [15, 9,58].

Microscopic examination (Figure 2) revealed several erythrocyte abnormalities in Mn-exposed rats, including poikilocytosis, microcytosis, erythrocyte rouleaux formation, and acanthocytosis. Leukocyte alterations (Figure 3) included the presence of naked lymphocytes, Gumprecht's shadows, blast-like cells, and bilobed lymphocytes, suggesting cytotoxic or genotoxic impacts of Mn exposure on hematopoietic cells [59-63].

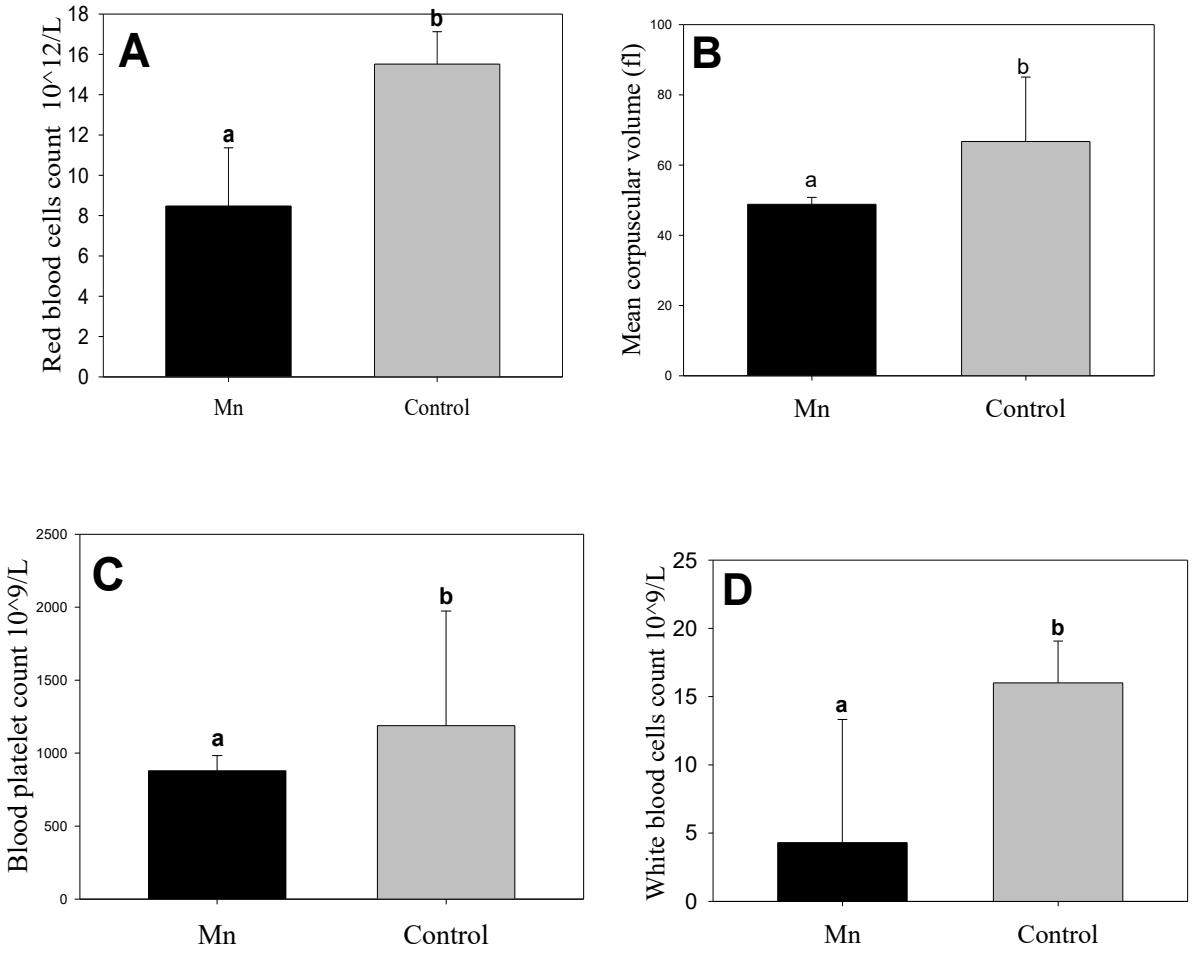

Figure 1 Blood counts in different subgroups of rats. A: Red blood cell count, B: 12.2. mean corpuscular volume (MGV) measurement, C: thrombocyte count, D: Leukocyte count. Data are expressed as the mean ± standard error, and data not sharing a common letter (a–c) differ significantly at $p < 0.05$ (Tuckey's t-test).

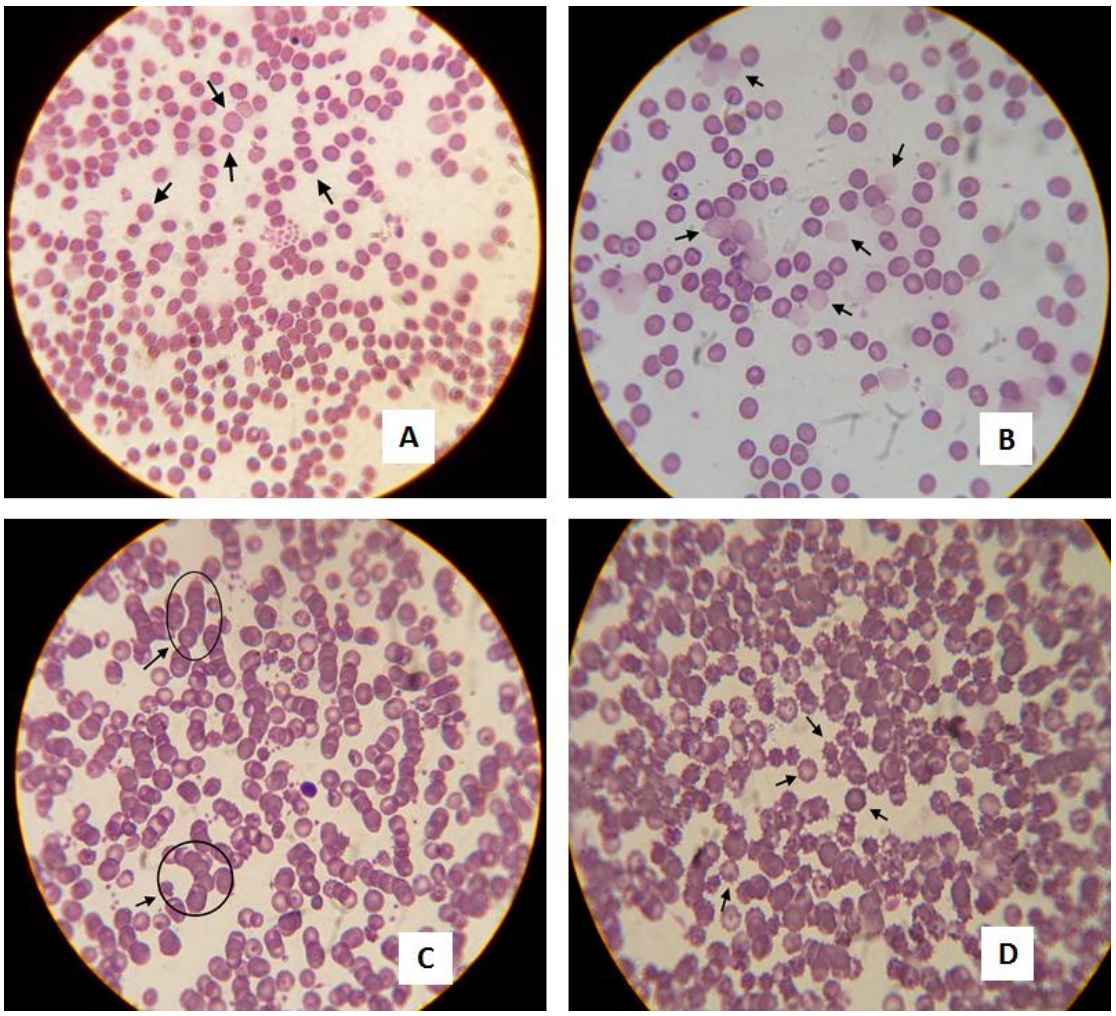

Figure 2 A: Anomalies in shape and size of red blood cells (Poikilocytosis or microcytosis); B: Pale red blood cells; C: Formation of erythrocyte rouleaux (hyper-aggregation); D: Red blood cell with numerous spicules and membrane surface projections (Acanthocytosis). Observations at magnifications (×40), images captured with a SAMSUNG ST93 camera.

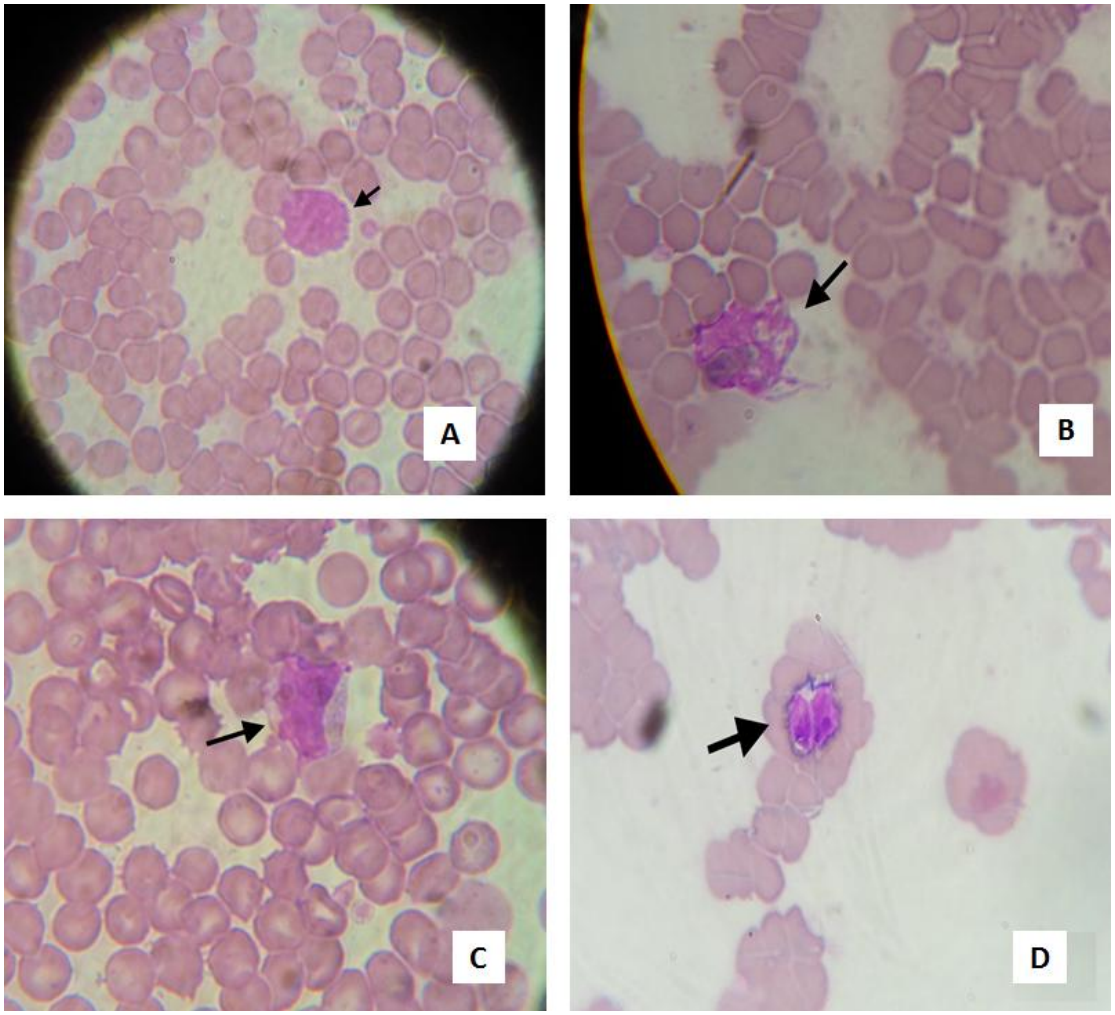

Figure 3 A: Naked lymphocyte; B: Guprecht's shadow; C: Bone marrow stem cell (Blast); D: Bilobed nucleus lymphocytes. Observations at magnifications (×100), images captured with a SAMSUNG ST93 camera.

This study demonstrates that chronic oral exposure to manganese chloride ($MnCl_2$) induces significant hematological alterations in Wistar rats, including microcytic anemia, thrombocytopenia, and leukopenia. These findings align with previous reports describing manganese (Mn) as a potent hematotoxic metal capable of disrupting iron metabolism, impairing hematopoiesis, and inducing oxidative stress [2, 16, 64]. The microcytic anemia observed in Mn-treated rats, characterized by decreased mean corpuscular volume (MCV), is indicative of impaired hemoglobin synthesis and iron deficiency–like effects. Mechanistically, manganese competes with iron (Fe^{2+}) for absorption via the divalent metal transporter-1 (DMT1) in enterocytes, leading to reduced intestinal Fe uptake and systemic iron depletion [17, 18, 65]. Additionally, Mn interferes with transferrin-mediated iron transport and storage by altering ferritin expression and destabilizing Fe homeostasis in erythroid precursors [19, 66]. This antagonism results in decreased hemoglobin content and the production of smaller erythrocytes, consistent with microcytosis [20, 67]. The erythrocyte morphological abnormalities such as poikilocytosis, acanthocytosis, and rouleaux formation further suggest oxidative

damage to the red blood cell membrane [21, 56]. Mn is known to catalyze the formation of reactive oxygen species (ROS) through Fenton-like reactions, leading to lipid peroxidation and membrane rigidity [22, 68]. Elevated ROS levels can oxidize membrane phospholipids and spectrin proteins, resulting in decreased deformability and premature erythrocyte destruction [23, 69]. Consequently, Mn-induced oxidative stress contributes not only to anemia but also to structural abnormalities in circulating erythrocytes [24, 70]. The observed thrombocytopenia and leukopenia indicate that Mn exposure impairs bone marrow hematopoietic activity. Chronic Mn accumulation in bone and bone marrow has been reported to cause mitochondrial dysfunction in hematopoietic stem cells, inhibiting differentiation and proliferation of myeloid and megakaryocytic lineages [25, 71]. Mn can also trigger apoptosis of bone marrow progenitor cells via activation of caspase-3 and disruption of the mitochondrial membrane potential, leading to decreased platelet and white blood cell counts [26]. These effects collectively suggest that bone marrow suppression is a critical mechanism of Mn hematotoxicity. Furthermore, Mn-induced oxidative stress may affect hematological parameters indirectly by altering erythropoietin (EPO) signaling [27, 72]. Excess Mn can impair kidney function and reduce EPO synthesis, thereby diminishing erythropoiesis [28, 73]. At the same time, systemic inflammation induced by Mn exposure could enhance hepcidin expression, further suppressing iron mobilization and exacerbating anemia of inflammation [29, 74]. Collectively, the hematological and morphological alterations observed in this study underscore the multifactorial toxicity of Mn involving oxidative stress, iron antagonism, mitochondrial dysfunction, and bone marrow suppression [75, 76] These pathophysiological effects highlight the vulnerability of the hematopoietic system to chronic Mn exposure and emphasize the need for regular biomonitoring of Mn-exposed individuals, particularly in occupational settings such as mining and welding [77, 79].

The application of nanotechnology concepts provides a more detailed understanding of manganese-induced hematological damage. At the nanoscale level, manganese can alter membrane integrity, promote oxidative stress, and disrupt cellular homeostasis in erythrocytes, leukocytes, and platelets. These processes contribute to anemia, leukopenia, thrombocytopenia, and abnormal blood cell morphology, highlighting the importance of nanotoxicological approaches in hematological research. Table 1 presents a comparison between conventional toxicological methods and nanotechnology-based approaches, demonstrating improved sensitivity and accuracy in detecting manganese-induced hematological damage.

Table 1 Comparison between conventional and nanotechnology-enhanced approaches in evaluating manganese-induced hematotoxicity.

Parameter	Conventional Toxicological Analysis	Nano-Enhanced Analysis
Detection Sensitivity	Moderate	High
Oxidative Stress Analysis	General	Detailed nanoscale
Cellular Damage Detection	Limited	Advanced
Inflammation Assessment	Standard	Nano-biomarker based
Diagnostic Accuracy	Good	Improved

Figure 4 presents the nanoscale mechanisms of manganese toxicity, highlighting oxidative stress pathways, cellular damage, and inflammatory responses contributing to hematological disorders.

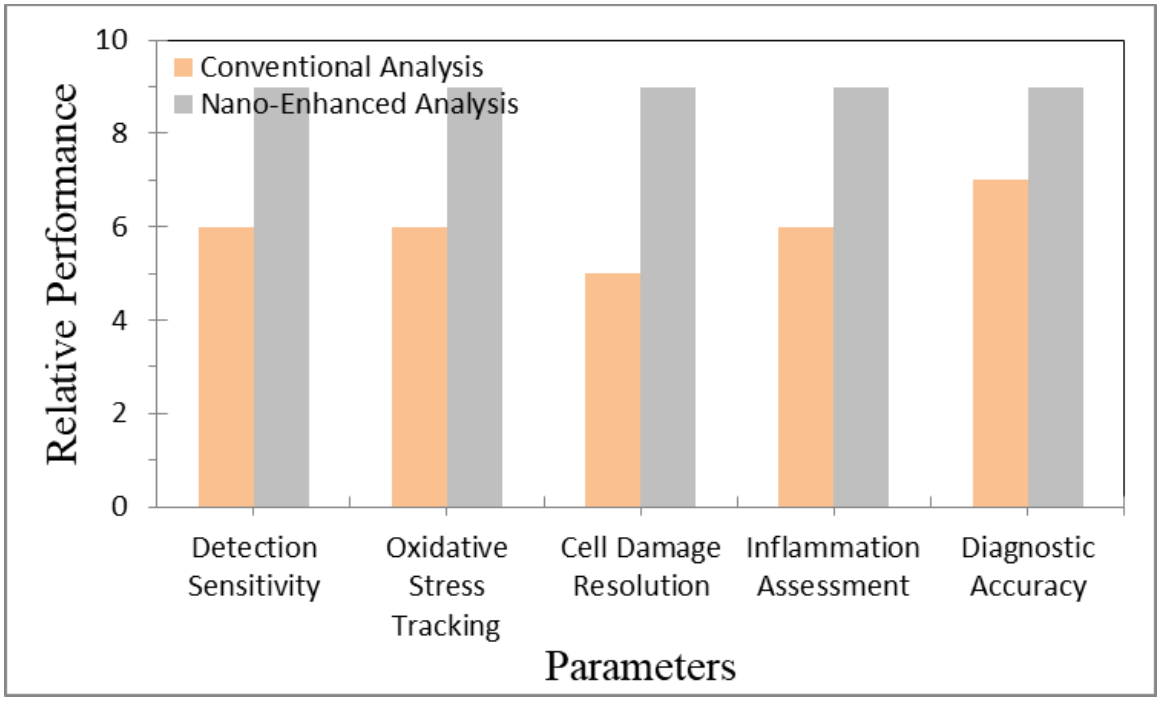

Figure 4 Schematic illustration of nanotechnology-based mechanisms underlying manganese-induced hematological alterations, including oxidative stress generation, blood cell damage, and inflammatory responses.

4. CONCLUSIONS

Chronic exposure to manganese chloride causes pronounced hematological disturbances in Wistar rats, including microcytic anemia, leukopenia, and thrombocytopenia, accompanied by marked erythrocyte morphological abnormalities. These alterations likely arise from the combined effects of oxidative stress, iron antagonism, and bone marrow suppression. Manganese competes with iron for intestinal absorption and transport, impairs erythropoiesis, and induces mitochondrial dysfunction in hematopoietic tissues. The results of this study provide compelling evidence for the hematotoxic potential of chronic Mn exposure and reinforce the importance of occupational health monitoring and environmental control of Mn contamination to safeguard public health. The integration of nanotechnology into toxicological studies provides a powerful framework for understanding manganese-induced hematotoxicity at the nanoscale level. Nanotechnology-based approaches enhance the detection of oxidative stress, inflammation, and blood cell damage, offering new opportunities for early diagnosis and improved toxicological monitoring of manganese exposure.

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