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Aluminum-induced epididymal damage and infertility in adult male rats: A nanotechnology-based perspective on nanoscale toxicity and oxidative mechanisms

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Epididymis damages are one of the most important causes of male infertility, described as post-testicular causes. Moreover, oxidative stress and inflammation are greatly implicated in these pathologies. The real point of this study is to look at what happens to the epididymis of adult male rats that are given 34 mg/kg of body weight of aluminum chloride ($AlCl_3$) by mouth for 30 days. There is a big drop in serum testosterone (0.31±0.26 ng/ml) and a rise in malondialdehyde (0.16±0.015 μM/g)

and superoxide dismutase activity in epididymal tissue homogenates (9.28±0.17 U/mg protein) after exposure to aluminum. Histological examination of the epididymis showed multiple abnormalities, namely the presence of apoptotic cells, testicular cellular debris (spermatocytes or spermatids) in the lumen, infiltration of immune cells, and the formation of an inflammatory focus inducing the appearance of sperm granuloma following the extraversion of spermatozoa in the extracellular matrix. To our knowledge, this study is the first to document the occurrence of rat sperm granuloma caused by subacute exposure to aluminum. In addition, nanotechnology-based perspectives provide deeper insight into aluminum-induced toxicity by examining nanoscale interactions, oxidative stress pathways, and cellular damage mechanisms, which may contribute to the development of advanced diagnostic and therapeutic strategies for male infertility

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Keywords: Aluminum; Epididymis; Inflammation; Oxidative stress.

1. INTRODUCTION

There are numerous examples of medications and environmental contaminants that cause alterations in spermatogenesis and/or epididymal sperm in experimental animals in the toxicology literature [1,2]. Until shown otherwise, it's safe to presume that humans exposed to these compounds will experience similar effects [3,4]. The potential association between chronic exposure to heavy metals, the alteration of antioxidant metabolism, and oxidative stress is well established [5,6]. Arsenic, lead, cadmium, and mercury are positively associated with pro-inflammatory cytokines and oxidative status [7,8]. Elevated plasma aluminum (Al) levels are linked to altered essential metal concentrations, increased oxidative stress, and increased inflammation [9,10]. Aluminum is an omnipresent environmental and industrial metal with increasing human exposure through food, water, pharmaceuticals, and occupational settings [11,13]. Despite its widespread distribution, aluminum's reproductive toxicity—especially its impact on post-testicular structures such as the epididymis—remains understudied compared to other heavy metals [14-16]. Given that the epididymis plays a fundamental role in sperm maturation, any disruption in its physiological integrity has significant implications for male fertility and reproductive health [17,18]. The epididymis is the site of sperm maturation, and it is critical for the development of mature, mobile spermatozoa with fertilization potential [19-21]. Proper epididymal function ensures that sperm acquire motility, membrane stability, and the capacity to undergo the acrosome reaction [22]. Consequently, damage to this organ can severely compromise male fertility even when testicular spermatogenesis remains intact [23-25]. Inflammation of the epididymis, which leads to the production of sperm granulomas, is the most common chemically caused epididymal lesion [26]. The blood-epididymal barrier (BEB), formed by occlusive connections between the apical surfaces of the epididymal epithelium, is not as effective as the blood-testis barrier [27-30]. Immune cell infiltration following recognition of antigenically foreign spermatozoa due to vacuolation and rupture of the epididymal epithelium is occasionally seen as a primary chemical-induced consequence and can lead to progressive granulomatous inflammation and the creation of a sperm granuloma [31].

Although previous toxicological studies have investigated aluminum's impact on testicular structure and endocrine function, relatively few have examined its effects on epididymal morphology, oxidative balance, and inflammatory cascades. Moreover, existing research rarely documents specific pathological outcomes such as sperm granuloma formation following sub-acute aluminum exposure [32,33]. Given the central role of the epididymis in sperm maturation and the increasing concern about environmental aluminum exposure, understanding how aluminum chloride affects epididymal integrity is crucial [34]. There is a distinct lack of studies that evaluate simultaneous changes in oxidative stress

markers, inflammatory responses, structural pathology, and circulating testosterone levels in a single experimental model [35,36]. The BEB not only protects developing sperm from the immune system but is also impacted by cytokines generated during inflammation, which can cause barrier disintegration. Inflammation may be the cause of barrier function loss [37,38]. This loss is linked to an immunological response and a reduction in sperm function, and it appears to play a role in post-testicular male infertility [39-41]. A lot of the time, changes in the epididymal epithelium that happen when there is less androgen stimulation are linked to vacuolation [42,43]. But, after 35 days, mice that are implanted with a lot of testosterones had sperm granulomas [44]. It has been suggested that high doses of testosterone damage the integrity of the epididymis [45-47]. Epididymis injury or surgical treatment, such as vasectomy in men, can also lead to the occurrence of an inflammatory granulomatous process [48]. Sperm granulomas are made up of a mass of spermatozoa surrounded by epithelioid macrophages, granulation tissue, lymphocytes, and plasma cells [49,50]. In this regard, oxidative stress and inflammation are thought to be major mechanisms underlying male infertility [51,52]. To our knowledge, no previous research has documented the formation of sperm granulomas in rats following sub-acute oral exposure to aluminum chloride. This study is therefore the first to provide histopathological evidence linking aluminum exposure to epididymal rupture and granulomatous reactions [53-55]. The primary objective of this study is to evaluate the effects of sub-acute exposure to aluminum chloride on epididymal tissue structure, oxidative stress biomarkers, inflammatory indicators, and circulating testosterone levels in adult male rats. This research investigates biochemical markers such as malondialdehyde (MDA), superoxide dismutase (SOD), and serum testosterone; histopathological alterations of the epididymis; and the presence of immune cell infiltration and sperm granuloma formation. Adult male rats received 34 mg/kg body weight of aluminum chloride orally for 30 days, providing a controlled model to assess aluminum-induced reproductive toxicity [56,57].

By combining biochemical, endocrine, and histological evaluations, this study contributes to a deeper understanding of how aluminum affects male reproductive health beyond the testes [58]. The findings have implications for toxicology, occupational health, environmental safety, and reproductive medicine, offering new insight into potential mechanisms of post-testicular infertility [59,60]. In this paper, we present comprehensive evidence demonstrating that sub-acute aluminum chloride exposure induces oxidative stress, inflammation, epithelial disruption, and sperm granuloma formation in the epididymis of adult male rats, thereby highlighting a previously undocumented pathway of aluminum-associated male infertility. Recent advances in nanotechnology have enabled a better understanding of toxicological mechanisms at the nanoscale level, particularly in relation to metal-induced oxidative stress and cellular damage. Nanotoxicology focuses on how nanoparticles and metal ions interact with biological systems, leading to oxidative imbalance, inflammation, and tissue disruption. Applying nanotechnology concepts to aluminum toxicity allows for deeper insight into molecular and cellular alterations affecting reproductive health.

2. MATERIALS AND METHODS

2.1. Ethical approved

The samples studied are acquired according to ethical approval committee, approval number (7693 at 25/11/2023), in the Al-Shirqat General Hospital, Salah Al-Deen Directorate of Health Government, Ministry of Health, Iraq

2.2. Animals and experimental design

Twelve male Wistar rats (average weight 193.9 ± 26.46 g) are used in this study. The animals are maintained under standard conditions of humidity, temperature (22±2 °C), and light/dark cycle (12 hours:12 hours). They had free access to the laboratory diet and water. The animals are then assigned to the following two groups (n = 6): Group I: received orally (gavage) aluminum chloride ($AlCl_3$) at a dose of 34 mg/kg b.w. (body weight), and Group II: served as a control (without any treatment).

2.3. The collection and analysis of tissue samples

Following a duration of four weeks, the animals are euthanized while under the influence of profound anesthesia. The epididymis from each rat specimen is harvested and measured in weight. One portion is stored in a freezer at a temperature of -20 °C, while the other portion is preserved in a solution of 10% Formol-salin and then embedded in paraffin. Sections with a thickness of five micrometers (µm) are acquired in order to conduct morphophysiological and histochemical analyses.

2.4. Bodyweight gain and epididymis index

Upon the conclusion of the experimental procedure, the researchers proceeded to measure the body weight of the rat and the weight of the epididymis. These measurements are used to determine the body weight increase and the epididymis index (Ei) by applying the following formula [58-60]:

$$Ei = \frac{Organ\ mass\ (g)}{Body\ mass\ (g)} \times 100 \quad (1)$$

2.5. Tissue biochemical analysis

Following defrosting, the epididymal tissue is homogenized to 10% (w/v) in cold phosphate buffer (pH 7.4) and centrifuged for 10 minutes at 3000 rpm. Malondialdehyde (MDA) and superoxide dismutase (SOD) tests are then done on the supernatant [12].

2.6. Hormonal analysis

Blood samples are taken from the inferior vena cava in dry tubes. The blood has been centrifuged to obtain serum, which is then stored in the refrigerator until used for hormone assays. All tests are performed within 24 hours of specimen collection. Radioimmunoassay methods with commercial kits (VIDAS Assays, BIOMERIEUX) are used to measure testosterone levels in serum samples (Abubakar and Ang, 2020).

2.7. Histological studies

After the histological procedure is completed, paraffin blocks of the tissues are prepared and sectioned at a thickness of 5 µm. For the morphophysiological examination, a subset of the samples underwent staining with hematoxylin-eosin. In contrast, the remaining sections are prepared for histochemical analysis using toluidine blue (Sigma, T3260) staining to ascertain the apoptosis state of the cells and estimate the degree of inflammation via mast cell recording [25].

2.8. Statistical analysis

The results are utilized to calculate the standard error (SE) of six replicates. A comparison of multiple samples is conducted utilizing the GraphPad Prism 9.5.1 for Windows software. There is an ANOVA, and then there is a Tukey's multiple comparison tests. P0.05 represented the level of significance.

2.9. Nanotechnology-based toxicological assessment

Nanotechnology-based approaches can be applied to enhance the evaluation of aluminum toxicity through nanoscale analysis of tissue damage and oxidative stress markers. Advanced analytical tools such as nanoscale imaging, high-resolution microscopy, and nano-biomarker detection can provide more detailed characterization of cellular alterations and inflammatory responses in epididymal tissue

3. RESULTS AND DISCUSSION

3.1. Bodyweight gain and epididymis index

The results in Table 1 show a significant diminution in body weight gain in Al-exposed groups (group I) compared to the control group of rats (group II). In contrast, the epididymis index did not differ significantly between the two groups.

Table 1 Effects of Al on bodyweight evolution and epididymis index (E_i).

Groups	Initial bodyweight(g)	Final body weight (g)	Bodyweight difference (g)	Ei
I	202.30±45.22	226.33±24.65	24.00±49.71[a]	0.31±0.03 [a]
II	189.60±04.00	275.00±01.40	87.50±07.00[b]	0.35±0.06 [a]

I: $AlCl_3$-exposed group; II: control group. Data are expressed as means ± SE (n = 6). The Tukey test is employed to identify and compare groups. Different columns devoid of a common letter (a–b) are significantly different at $p < 0.05$.

3.2. Oxidative stress biomarker evaluation

Figure 1 displays the findings of measuring tissue lipid peroxidation in the homogenates of the epididymides that are studied. This is done by using spectrophotometry to find the level of malondialdehyde (MDA) and superoxide dismutase (SOD) activity. The amount of MDA in the tissue homogenates of rats that are exposed to Al is significantly higher ($p < 0.001$) than in the tissue homogenates of rats that are not exposed to Al (0.36±0.04 μM/g tissue). While the levels of enzyme activity are significantly higher ($p < 0.05$) in the epididymal homogenates of Al-exposed rats (9.28±0.17 U/mg protein) compared to control rats (8.59±0.07 U/mg protein),

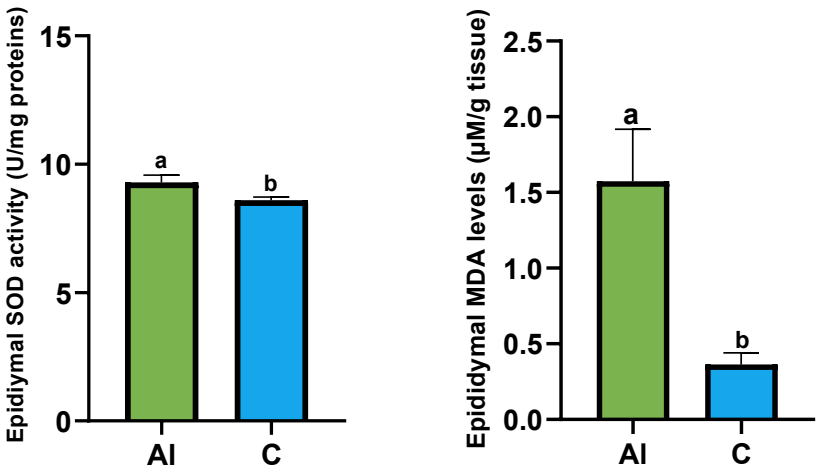

Figure 1 Oxidative stress biomarker evaluation in epididymal tissue A: malondialdehyde (MDA) levels; B: superoxide dismutase activity (SOD). Al: aluminum chloride exposed group; C: control group. Data are expressed as means ± SD (n = 6). The Tukey test is employed to identify and compare groups. Different columns devoid of a common letter (a–b) are significantly different at $p < 0.05$.

3.3. Serum testosterone level

The results in Figure 2 show a significant variation in serum sex hormone levels between the two different experimental groups. Indeed, a very significant decrease ($p < 0.001$) in the serum testosterone level is noted in the group of rats exposed to Al (0.31±0.15 ng/ml) compared to the control group (1.24±0.35 ng/ml).

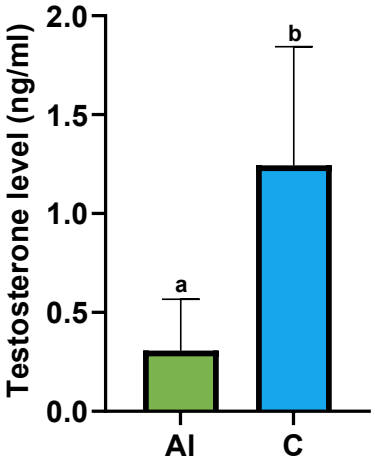

Figure 2 Testosterone-level evaluations Al: aluminum chloride exposed group; C: control group. The data are presented as means with standard deviations (n = 6). The categories are compared using the Tukey test. Bars denoted by letters other than a–b differ substantially ($p < 0.05$).

3.4. Epididymis histotology

In the control groups, the epididymal canal's histological sections (Figure 3) show a typical structure, with a cubical to prismatic epithelium filled with stereocilia and a light densely packed with spermatozoa. In addition, observation of epididymal histological sections in Al-exposed rats reveals multiple disturbances, namely the presence of testicular germ cells or cellular debris (often recognizable as round spermatids or spermatocytes) in the lumen of the epididymal duct, indicating disruption of spermatogenesis in the testis; inflammation in response to tissue injury; and disruption of the hemato-epididymal barrier, resulting in infiltration of immune cells and the formation of a granulomatous reaction surrounding a sperm nucleus (Sperm granuloma).

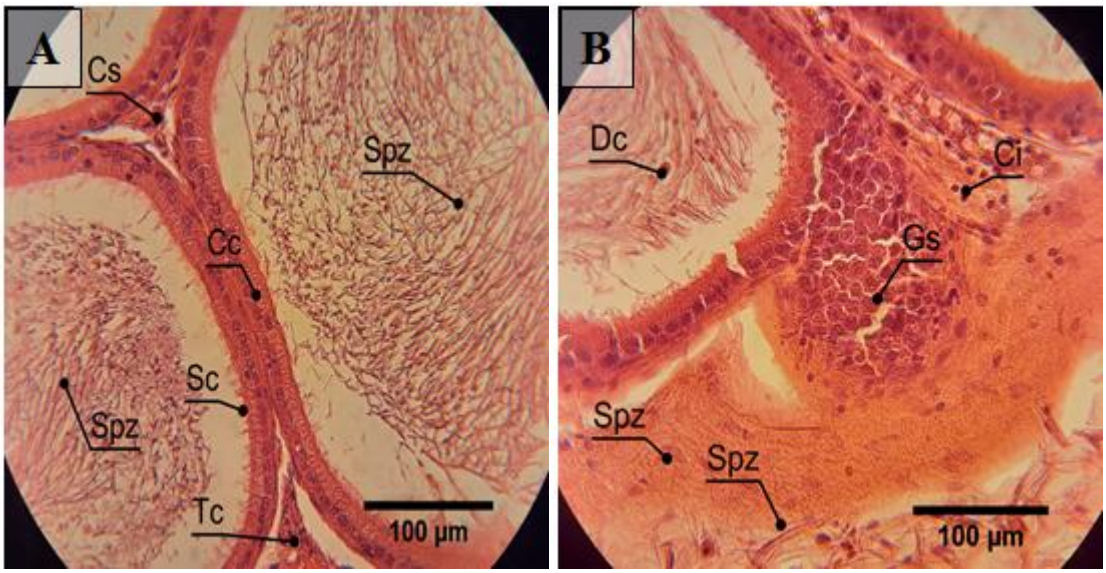

Figure 3 Microscopic observation of the histological structure of the epididymal canal. H&E staining (G×400). A: represents the epididymal segment of the control group composed of cubic to prismatic cells (Cc) provided with stereocilia (Sc). B: Al exposed group showing multiple abnormalities, namely the presence of apoptotic cells (Apo), testicular cellular debris (Dc) (spermatocytes or spermatids) in the lumen, infiltration of immune cells (Ci) in the epithelium, and formation of inflammatory focus inducing the appearance of sperm granuloma (Gs) following the extravasation of spermatozoa (Spz) in the extracellular matrix.

3.5. Epididymis histochemistry

The exploration of histological sections of the epididymis stained with toluidine blue (Figure 4), to estimate the state of the cells and the degree of inflammation due to the infiltration of immune cells, showed in the control group of rats a normal architecture of the epididymis tissue free of any apoptotic cells or immune cells, with tubules filled with spermatozoa, indicating the good progress of spermatogenesis. However, exposure to Al at 34 mg/kg b.w. for 30 days induced an apoptotic state of the epididymal cells, as shown by a positive stain with toluidine blue showing cells with dense nuclei typical of a state of chromatin condensation, which is indeed the first sign of cell death. Toluidine blue staining also allowed the visualization of mast cell-type immune cells in the interstitial tissue, showing an inflammatory state of the epididymal tissue.

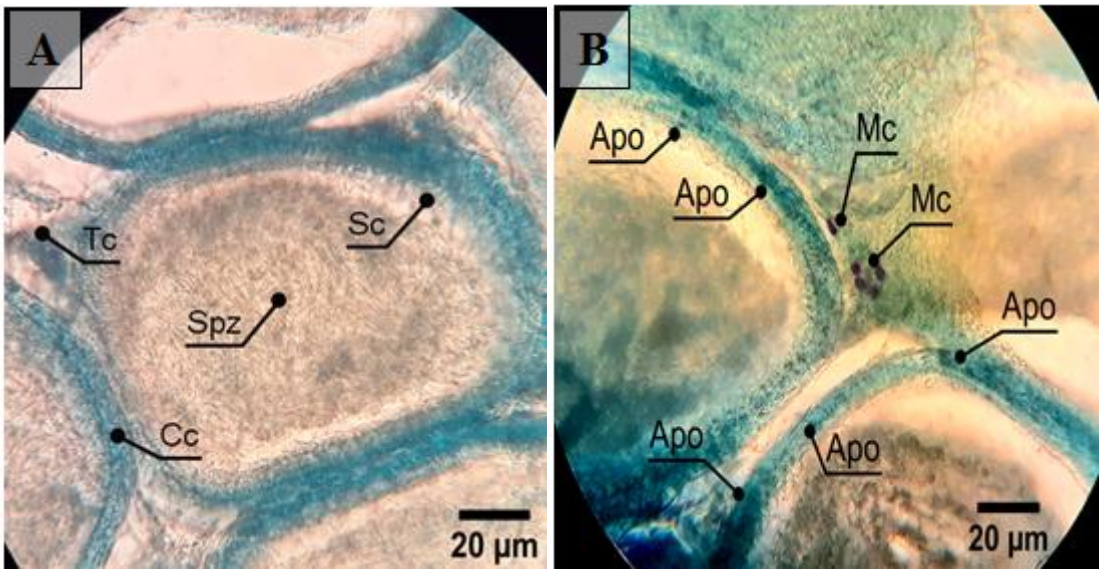

Figure 4 Looking at histological sections of the epididymis through a microscope after they have been stained with borated toluidine blue (G□400). A represents the observation of the control group showing a normal appearance of epididymal tissue composed of cubic to prismatic cells (Cc) provided with stereocilia (Sc) free of any apoptotic cells or immune cells. B represents the observation of the Al group showing apoptosis (Apo) and mast cell infiltration (Mc).

This study represents the first documented occurrence of rat sperm granuloma caused by subacute exposure to aluminum chloride (AlCl3). Epididymal damage is a significant contributor to male infertility, often attributed to post-testicular factors [11, 61-65]. The study results show that short-term exposure to AlCl3 has serious negative effects on the epididymis [66-70]. These effects include oxidative stress, inflammation, lower serum testosterone levels, and the growth of sperm granulomas. Heavy metals, including aluminum, have been associated with alterations in spermatogenesis and epididymal function in both animal studies and humans [20, 71-75]. Chronic exposure to heavy metals is known to disrupt antioxidant metabolism and induce oxidative stress [22, 76-80]. Elevated levels of Al in the plasma have been linked to increased oxidative stress and inflammation [15]. The epididymis is very important for sperm development. When chemicals damage the epididymis, they often cause inflammation in this area, which then creates sperm granulomas [14, 24]. While BEB is designed to protect sperm from the immune system, it is not as robust as the blood-testis barrier [21]. Chemical exposure can disrupt the BEB, allowing immune cell infiltration, inflammation, and granuloma formation [81-85]. This, in turn, can impair sperm function and contribute to male infertility [9]. The results of this study demonstrate that subacute exposure to $AlCl_3$ leads to several significant consequences. Serum testosterone levels are significantly reduced in the Al-exposed group, indicating disruption of endocrine function [8]. The decline in serum testosterone is a concerning finding, as testosterone is essential for male reproductive health and normal sperm production [27]. Oxidative stress biomarkers, specifically malondialdehyde (MDA) and superoxide dismutase (SOD), are evaluated in the epididymal tissue. [18] say that the significant rise in MDA levels in the Al-exposed group suggests higher lipid peroxidation and oxidative stress. [23] say that the rise in SOD activity may be an adaptive response to counteract the oxidative damage [86]. These findings highlight the oxidative stress that exposure to Al causes and its potential role in epididymal damage. A close look at

the epididymal tissue through a microscope showed that the Al-exposed group had a number of problems, such as cells that had died, testicular cell debris, and immune cells infiltrating the tissue [87]. These changes indicate disruption of spermatogenesis in the testis and inflammatory processes. Additionally, the development of sperm granulomas, which are spermatozoa encircled by inflammatory cells [10], suggests a significant alteration of epididymal function due to aluminum exposure. The histochemical toluidine blue staining showed that the epididymal tissue of the group that had been exposed to Al is also in an inflammatory state [88]. The staining showed that epididymal cells had gone through apoptosis, which is a sign of cell death [6], and mast cells had moved in, which is a sign of inflammation [25]. These findings confirm the impact of aluminum exposure on the histological and inflammatory status of the epididymis.

3.6. Effect of nanotechnology on understanding aluminum-induced toxicity

The application of nanotechnology concepts provides a deeper understanding of aluminum-induced toxicity at the cellular and molecular levels. Nanoscale interactions between aluminum ions and biological structures contribute to oxidative stress, membrane damage, and inflammatory responses. These effects lead to disruption of epididymal function and sperm maturation, highlighting the importance of nanotoxicological approaches in studying reproductive toxicity. Table 2 presents a comparison between conventional toxicological methods and nanotechnology-based approaches, demonstrating improved sensitivity and accuracy in detecting aluminum-induced damage

Table 2 Comparison between conventional and nanotechnology-enhanced approaches in evaluating aluminum-induced reproductive toxicity.

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 5 presents the nanoscale mechanisms of aluminum toxicity, highlighting oxidative stress pathways, cellular damage, and inflammatory responses contributing to male infertility.

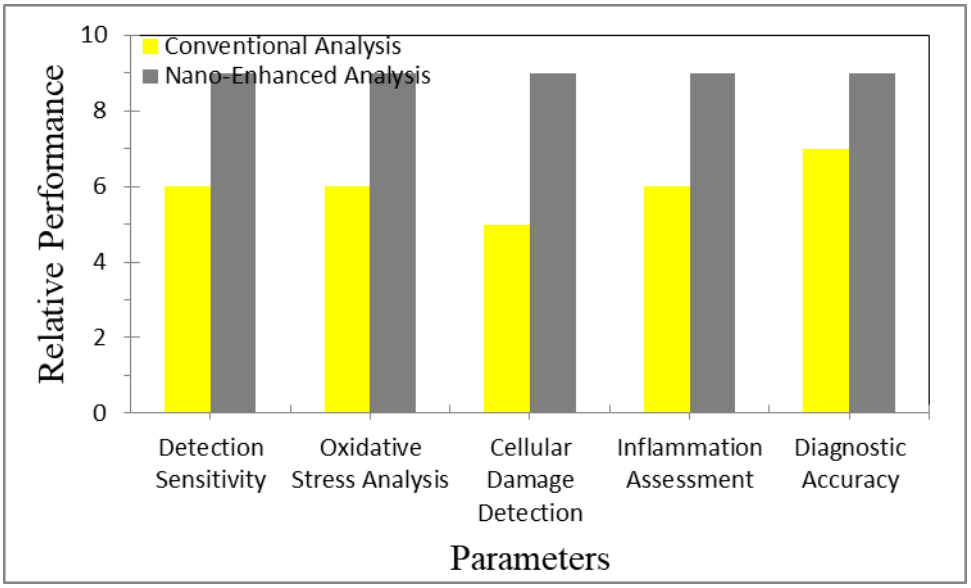

Figure 5 Nanotechnology-enhanced evaluation of aluminum toxicity, showing improved detection sensitivity, oxidative stress analysis, cellular damage detection, inflammation assessment, and diagnostic accuracy compared with conventional analysis.

4. CONCLUSIONS

In conclusion, this study is the first to report the occurrence of rat sperm granuloma resulting from subacute exposure to aluminum chloride. The findings underscore the reproductive toxicity of aluminum and its potential to disrupt male fertility through oxidative stress, inflammation, and disruption of normal epididymal function. This research contributes to our understanding of the adverse effects of Al exposure on male reproductive health and highlights the importance of further investigations to elucidate the underlying mechanisms and explore potential interventions to mitigate these effects. The integration of nanotechnology into toxicological studies provides a powerful framework for understanding aluminum-induced reproductive toxicity at the nanoscale level. Nanotechnology-based approaches enhance the detection of oxidative stress, inflammation, and cellular damage, offering new opportunities for early diagnosis and targeted therapeutic strategies in male infertility.

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