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Green Synthesis of Purslane Seeds-Selenium Nanoparticles and Evaluate its Cytokines Inhibitor in Lead Acetate Induced Toxicity in Rats

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Lead is a heavy metal that produces toxicity in both humans and animals due to oxidative stress. The aim of this study was to synthesize through green channel and evaluate hepatoprotective effect of purslane seeds-selenium nanoparticles (PS-Se NPs) against lead acetate-induced liver toxicity. The PS-Se NPs were made and their particle size and zeta potential were measured. In addition, the IC_{50} of SP-Se NPs against the HepG-2 liver cancer cell line as well as and LD_{50} in rats were determined. The PS-Se NPs had a mean particle size of around 54.70 ± 4.55 nm, a negative zeta potential of +17.25 and the observed shape of the nanoparticle was spherical. Also, the IC_{50} of PS-Se NPs against HepG-2 liver carcinoma cell line is 92.66 µg/mL and the LD_{50} is 1695 mg/kg b.w. Daily oral administration of PS-Se NPs at concentrations of 33.9 and 84.75 mg/kg b.w. for 30 days to rats treated with lead acetate (0.6 mg/kg b.w.) resulted in significant improvements in ALT, AST, ALP, TNF-α, Bcl-2, P53 and MDA. In contrast, oral administration of PS-Se NPs increased the levels of SOD, GPx and GSH in the liver tissue of lead acetate-treated rats. Furthermore, PS-Se NPs almost normalized these effects in histoarchitecture and ultrasound scanning examination of liver tissue. The PS-Se NPs have protective activity against lead acetate-induced liver tissue injury in rats, according to the biochemical, histological and ultrasound scanning findings.

Keywords: Purslane seeds, PS-SeNPs, Lead acetate, Liver tissue toxicity, Cytokine storm.

INTRODUCTION

Lead is one of the most persistent hazardous contaminants in the environment, posing a serious public health risk [1]. It is a multi-organ toxin that affects the hematopoietic tissues, liver, kidney, brain and testis, among other organs [2]. It has been reported in the literature and also demonstrated, which lead causes oxidative stress [3], alters the glutathione system [4] and induces apoptosis [5]. Human occupational exposures may also occur during the application and removal of protection leading lacquers [6], during the grinding [7], welding [8] and cutting of materials with plumbing lacquers, for example, in the construction of ships, demolition industries, the manufacturing of heavy plumb glass and crystal and in crystal carving industry. Extensive sources of exposure can also be mining, smelting, informal processing and waste-recycling. Another important source of exposure is the use of fuel oil [9]. Several plants and their products have been screened for their protective efficacy against metal toxicity in various animal models. Some of the most recent investigations that yielded positive results include cranberry [10], resveratrol [11-13], *Verbascum nubicum* [14], astaxanthin [15], cucurbitacin-e-glycoside [16] and purslane [17].

Some herbal extraction compounds may be antioxidant to mitigate complications linked to obesity, such as atherosclerosis and some cancers [18-21]. Plants like purslane contain high concentrations of unsatsurated fatty acids and polyphenols, which represent excellent scavengers and promise anti-obesity effects of reactive oxygen-species [19]. The purslane leaves were tested *in vivo* in order to determine, for example, its hepatoprotective activity [22], hypolipidemic activity, hypoglycemic activity and antioxidation [17,23].

No reports about hepatoprotective, antioxidant and cytokines inhibitory activity of purslane seeds-selenium nanoparticles (PS-Se NPs) against lead acetate induced liver injury in rats. As an extension of our interested research program in the extraction and therapeutic evaluation of rare medicinal plants [24,25], we report herein, a facile route to explain the liver protective and antioxidant effects of the PS-SeNPs against lead acetate induced liver injury in rats, in which may pave the way for possible therapeutic application.

EXPERIMENTAL

Prof. Heba A Elgizawy, Department of Phytochemistry, College of Pharmacy, October 6 University, Giza, Egypt, authenticated the purslane seeds obtained from a local market. Sigma Chemical Co. provided vitamin C and lead acetate (St. Louis, USA). All of the other compounds used in this experiment were of analytical grade.

Preparation of aqueous extract: Aqueous extract of purslane seeds was prepared by stirring 50 g powdered purslane seeds in 500 mL warm distilled water for 45 min at 60-70 ºC with a magnetic stirrer at 60-70 ºC, filtered, dried under reduced pres-sure and weighed (3.5 g). An aqueous suspension, which is the most prevalent form of folk medicine, was created to make handling easier.

Phytochemical screening: Alkaloids, cardiac glycosides, flavonoids, anthraquinones, saponins, fixed oil, polyphenols, ω-3 and ω-6 were all found in purslane seeds [26].

Preparation of purslane seeds-selenium nanoparticles (PS-Se NPs): A freshly prepared 20 mM ascorbic acid solution was prepared by dissolving 35.2 g in 10 mL deionized water. Aqueous extract of purslane seeds was dissolved in deionized water and then diluted in deionized water (90 mL) in a conical flask as follows: The ruby red SeNPs were suspended and characterized by TEM after dissolving selenious acid (H_2SeO_3, 0.013 g, 0.01 mmol) in 10 mL deionized water and heating at 60 °C for 10 h with constant stirring.

Characterization of PS-Se NPs: The crystal-line characteristics and grain dimensions of PS-Se NPs were determined by the X-ray diffraction pattern at 25-28 ºC with nickel (D8 Advance X-ray diffractometer) filtered using Cu*K*α (β = 1.54184 Å) radiation as X-rayed source. Scanning electron microscope and field transmission microscope at an accelerating voltage of 15 and 200 kV have investigated the morphology and size of the PS-Se NPs.

Determination of PS-Se NPs cytotoxicity on cells: To generate a full monolayer sheet, the 96 well tissue culture plate was injected with 1×10^5 cells/mL (100 µL/well) and incubated at 37 ºC for 24 h. Growth medium was decanted from 96 well micro titer plates after creating a confluent sheet of cells and the cell monolayer was washed twice with wash media. In RPMI medium with 2% serum, two-fold dilutions of the tested material were prepared (maintenance medium). Three wells served as controls and received simply maintenance medium, while 0.1 mL of each dilution was evaluated in each well. After incubation at 37 ºC, the plate was examined.

Any physical symptoms of toxicity, such as partial or total loss of the monolayer, rounding, shrinkage or cell granulation, were examined in the cells. MTT solution (5 mg/mL in PBS) was prepared (BioBasic, Canada, Inc.). Each well received a 20 µL MTT solution. The MTT was mixed into the media completely at 150 rpm for 5 min. To allow the MTT to be metabolized, incubate for 1-5 h at 37 ºC and 5% CO_2. Remove the media from the equation. In 200 µL DMSO, resuspend formazan (MTT metabolic product). The formazan was also mixed into the solvent at 150 rpm for 5 min. Subtract b from the optical density was measured at 560 nm.

Animals: Adult albino rats weighing approximately 150 ± 5 g (90 rats; 60 for LD_{50} estimation and 30 rats for estimation of PS-SeNPs liver protective activity) were obtained from the animal house of Cairo University, Giza, Egypt. At the National Cancer Institute Animal House, they were housed in plastic cages with stainless steel covers. In a light-controlled room, the animals were kept at a temperature of 21 ± 2 ºC and a humidity of 55-60%. The animals were kept for one week to acclimate and fed a standard diet and given unlimited water.

Animal ethics: The Research Ethics Committee at Egypt's October 6 University's Faculty of Applied Medical Sciences granted the ethical approval for data collection (No. 20201110). In the studies that served as the foundation for this research, no human subjects were used; instead, rats were used in an *in vivo* study.

Determination of LD_{50} of PS-Se NPs: Four-rat groups were used in the preliminary testing. The PS-Se NPs were given orally in various dosages to establish the range of dosage that cause animals to die from 0-100%. The LD_{50} was measured by giving resveratrol nanoemulsion at different doses of 1200, 1400, 1600, 1800, 2000 and 2300 mg/kg orally to groups of 10 rats. Following delivery of the tested PS-Se NPs, animals were monitored individually every hour for the first day and every day for the next 5 days. Animal behaviour and clinical symptoms were tracked during the investigation. The LD_{50} was calculated using Abal *et al.* [27] method using the following formula:

$$LD_{50} = Dm - \left(\frac{\Sigma(Z.d)}{n} \right)$$

Dm = The largest that kill all animals; Σ = The sum of (Z × d); Z = Mean of dead animals between 2 successive groups; d = The constant factor between 2 successive doses; n = Number of animals in each group.

Experimental setup: The purpose of this study was to investigate if PS-Se NPs could protect against lead acetate-induced liver damage. The Animal Care and Use Committee of October 6th University developed rules for this experiment, which were followed. Six groups of adult albino rats were formed, each with six animals. Table-1 summarizes the treatment groups.

After 30 days of treatment, blood samples were drawn from each animal's retro-orbital vein and collected in heparin-containing tubes. The heparinized blood samples were centrifuged for 20 min at 1000 ×g. The separated plasma was used to estimate glutamic-oxaloacetic transaminase (GOT) [29], glutamic-pyruvate transaminase (GPT) [29] and alkaline phosphatase (ALP) [30] levels. Also, plasma TNF-α [31], Bcl-2 [32] and P53 [33] levels using ELISA kits from RayBiotech,

TABLE-1
DESCRIPTION OF TREATMENT GROUPS

Group	Group name	Treatment description
I	Normal control A	3 mL of distilled water, orally for 30 days
II	Lead acetate (LA)	Was given lead acetate orally 0.6 mg/kg b.w. in distilled water in a single daily dose for 30 days [28].
III	PS-Se NPs + lead acetate	Oral suspension of 1/50 LD_{50} (33.9 mg/kg b.w. PS-Se NPs) in water + oral administration of 0.6 mg/kg b.w. lead acetate in distilled water in a single daily dose for 30 days.
IV	PS-Se NPs + lead acetate	Oral suspension of 1/20 LD_{50} (84.75 mg/kg b.w. PS-Se NPs) in water + oral administration of 0.6 mg/kg b.w. lead acetate in distilled water in a single daily dose for 30 days.
V	Vitamin C + lead acetate	Was simultaneously given vitamin C (1.0 g/kg b.w) and lead acetate (0.6 mg/kg b.w.) for 30 days [23].

Inc., USA, Quest Diagnostics Nichols Institute, San Juan, California and R&D Systems Inc., Minneapolis, USA, respectively was evaluated.

Preparation of liver samples: Cervical dislocation was used to kill the animals and then the mammary tissues were quickly removed. To prepare a 25% w/v homogenate, a portion of each liver was weighed and homogenized in a glass homogenizer (Universal Lab. Aid MPW-309, Mechanika Precyzyjna, Poland) with ice-cold saline. Also, homogenized suspension was filtrated and the supernatant was used to evaluate the liver superoxide dismutase (SOD) [34] and glutathione peroxidase (GPx) [35], malondialdehyde (MDA) [36] and reduced glutathione (GSH) [37].

Histological assessment: The mammary tissue was cut into pieces and fixed in a 10% buffered formaldehyde solution for histological study. An automated tissue processing machine was used to process the fixed tissues. Tissues were embedded in paraffin wax using standard techniques. Sections of 5 mm thickness were prepared and stained with hematoxylin and eosin for light microscopy analysis using the Attia *et al.* method [38]. The sections were then examined under the microscope for histopathological changes and photomicrographs were taken.

Ultrasound scanning procedure: The ultrasound procedure was applied 3 rats from each experimental groups using linear transducer with power 7 MHz in US machine model (Philips CV650). Animals were successfully anesthetized by inhalation using a piece of cotton saturated with 30 mL diethyl ether, the rats were prepared by prevent feeding for 8 h before the study to prevent vomiting, aspiration during anesthesia and gases from the abdominal area during the procedure, the abdominal area of rats were shaved to reduce imaging artifact. A conducting gel was applied to the rat's abdominal area during the procedure.

The rats were positioned in a supine recumbence position on the scanning table. The liver was examined by placing the probe in longitudinal and transverse positions to the upper hypochondrium, in longitudinal position the probe moved from left to right, while in transverse position the probe was angled its beam cranially, obtaining multiple longitudinal and transverse images.

Statistical analysis: For each of the eight separate determinations, the results were expressed as mean ± SD. SPSS/18 Software was used to perform statistical analysis on all of the data [39]. One-way analysis of variance was used to test hypotheses, followed by the least significant difference test. The *p* values of 0.05 are considered statistically significant.

RESULTS AND DISCUSSION

TEM analysis shows that purslane seeds-selenium nanoparticles (PS-Se NPs) had size of around 54.70 ± 4.55 nm with negative zeta potential of +17.25 (Fig. 1). Table-2 shows that incubation of PS-Se NPs at different concentrations (31.25, 62.50, 125, 250, 500 and 1000 µg mL^{-1}) with HepG2 cells

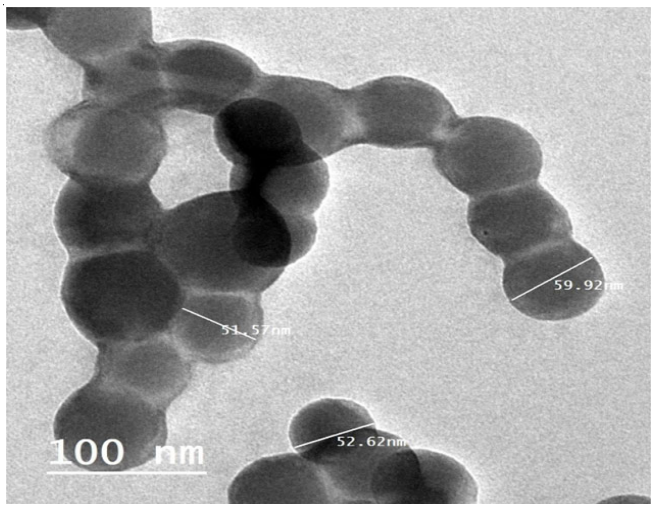

Fig. 1. TEM image (x200) of PS-Se NPs with a mean size of 54.70 ± 4.55 nm. The PS-SeNPs exhibit spherical shape and smooth surfaces

TABLE-2
IC_{50} OF PS-Se NPs AGAINST LIVER CARCINOMA (Hep-G2) CELL LINE

ID	Conc. (µg/mL)	Optical density			Mean optical density	Standard error	Viability (%)	Toxicity (%)	IC_{50}
	DMSO (0.1%)	0.383	0.376	0.399	0.386	0.006807	100	0	
PS-Se NPs	1000	0.018	0.019	0.017	0.018000	0.000577	4.663212435	95.33678756	92.66
	500	0.035	0.042	0.026	0.034333	0.004631	8.894645941	91.10535406	
	250	0.046	0.073	0.069	0.062667	0.008413	16.23488774	83.76511226	
	125	0.102	0.116	0.109	0.109000	0.004041	28.23834197	71.76165803	
	62.5	0.263	0.251	0.248	0.254000	0.004583	65.80310881	34.19689119	
	31.25	0.386	0.374	0.396	0.385333	0.006360	99.82728843	0.172711572	

resulted in viability of 99.82, 65.80, 28.23, 16.23, 8.89 and 4.66%, respectively and toxicity % of 0.17, 34.19, 71.76, 83.76, 91.10 and 95.33, respectively. The IC_{50} value of HepG2 liver carcinoma cell line was 92.66 μg mL^{-1}.

Table-3 shows that oral administration of PS-Se NPs in doses of 1200, 1400, 1600, 1800, 2000 and 2300 mg/kg b.w. resulted in mortalities of 0, 2, 4, 7, 9 and 10, respectively. The dose of PS-Se NPs that killed half of the rats (LD_{50}) was 1695 mg/kg b.w.

Table-4 shows the plasma AST, ALT and ALP levels. Oral administration of lead acetate led to significant increase of AST, ALT and ALP when compared with the normal control group ($p < 0.05$), indicating acute liver injury. Treatment of animals with PS-SeNPs at 33.9 and 84.75 mg/kg b.w., as well as vit.C (1 g/kg b.w.) significantly reduced the level of AST, ALT and ALP as compared with the lead acetate treated group. The acute or chronic exposure to lead damages the liver severely. Chronic lead injection was observed to generate a considerable increase in the primary marker enzymes for liver function and hepatic integrity, ALT, AST, and ALP, in the current investigation.

In present study, the concentration of PS-Se NPs was found to increase with increasing extract concentration, *i.e.*, increasing concentration of reducing agent [40]. Also, attributed increased PS-Se NPs concentration to the availability of more reducing biomolecules for the reduction of Se-2. The *in-vitro* determination of anticancer activity of various concentrations of PS-Se NPs showed the IC_{50} against Hep-G2 liver carcinoma cell line is 92.66 μg/mL.

Table-5 revealed a significant elevation in mammary tissue TNF-α, Bcl-2 and P53 levels ($p < 0.05$) in lead acetate treated rats when compared with control group. The administration of PS-SeNPs 33.9 and 84.75 mg/kg b.w., as well as vitamin C (1 g/kg b.w.) showed significantly decreased in TNF-α, Bcl-2 and P53 levels relative to lead acetate treated the group of rats after 30 days ($p < 0.05$). These findings suggested that the antifibrotic effect of PS-Se NPs is linked to the inhibition of mitogenic and/or fibrogenic signalling.

Table-6 shows a significantly ($p < 0.05$) decreased of mammary tissue antioxidant parameters SOD, GPx and GSH while significantly increasing mammary tissue MDA, were observed in the lead acetate treated rats as compared with the normal control group ($p < 0.05$), indicating acute mammary tissue damage. The PS-Se NPs 28.7 and 71.75 mg/kg b.w. as well as vitamin C (1 g/kg b.w.) treatment significantly enhanced the liver enzymes activities SOD, GPx and GSH in rats and decrease MDA level, as compared to the lead acetate treated group ($p < 0.05$).

TABLE-3
DETERMINATION OF LD_{50} OF PS-Se NPs GIVEN ORALLY IN ADULT RATS

Group number	Dose (mg/kg)	Number of animals/group	Number of dead animals	(Z)	(d)	(Z.d)
1	1200	10	0	1.0	200	200
2	1400	10	2	3.0	200	600
3	1600	10	4	5.5	200	1100
4	1800	10	7	8.0	200	1600
5	2000	10	9	8.5	300	2550
6	2300	10	10	0	00	6050

TABLE-4
EFFECT OF PS-Se NPs ON PLASMA ASPARTATE AMINOTRANSAMINASE (AST), ALANINE AMINOTRANSAMINASE (ALT), ALKALINE PHOSPHATASE (ALP) IN NORMAL AND EXPERIMENTAL GROUPS OF LEAD ACETATE-TREATED RATS

Groups	Treatment description	AST (U/L)	ALT (U/L)	ALP (U/L)
I	Normal control A	21.87 ± 3.76^{a}	38.95 ± 5.32^{a}	43.10 ± 4.10^{a}
II	LA (0.6 mg/kg b.w.)	74.66 ± 8.55^{d}	96.73 ± 6.40^{d}	81.22 ± 5.76^{d}
III	PS-Se NPs (33.9 mg/kg b.w.) + LA (0.6 mg/kg b.w.)	29.64 ± 2.90^{b}	43.44 ± 5.78^{b}	52.48 ± 6.11^{b}
IV	PS-Se NPs (84.75 mg/kg b.w.) + LA (0.6 mg/kg b.w.)	22.25 ± 4.16^{a}	39.64 ± 4.00^{a}	46.16 ± 4.64^{a}
V	Vit. C (1 g/kg b.w.) + LA (0.6 mg/kg b.w.)	36.90 ± 4.90^{c}	51.76 ± 3.27^{c}	63.77 ± 5.22^{c}

Data shown are mean ± standard deviation of number of observations within each treatment. Data followed by the same letter are not significantly different at $p \leq 0.05$.

TABLE-5
EFFECT OF PS-Se NPs ON LEVELS OF PLASMA TUMOR NECROSIS FACTOR ALPHA (TNF-α), B-CELL LYMPHOMA 2 (Bcl-2) AND TRANSFORMATION-RELATED PROTEIN 53 (P53) IN NORMAL AND EXPERIMENTAL GROUPS OF LEAD ACETATE-TREATED RATS

Groups	Treatment description	TNF-α (pg/mL)	Bcl-2 (ng/mL)	P53 (pg/mL)
I	Normal control A	2.65 ± 0.20^{a}	0.87 ± 0.16^{a}	74.3 ± 5.87^{a}
II	LA (0.6 mg/kg b.w.)	5.37 ± 0.42^{d}	3.66 ± 0.52^{c}	143.90 ± 12.38^{e}
III	PS-Se NPs (33.9 mg/kg b.w.) + LA (0.6 mg/kg b.w.)	3.18 ± 0.27^{b}	1.53 ± 0.18^{b}	110.76 ± 8.42^{c}
IV	PS-Se NPs (84.75 mg/kg b.w.) + LA (0.6 mg/kg b.w.)	2.53 ± 0.28^{a}	0.99 ± 0.13^{a}	84.50 ± 5.09^{b}
V	Vit. C (1 g/kg b.w.) + LA (0.6 mg/kg b.w.)	3.74 ± 0.30^{c}	1.76 ± 0.15^{b}	127.48 ± 10.57^{d}

Data shown are mean ± standard deviation of number of observations within each treatment. Data followed by the same letter are not significantly different at $p \leq 0.05$.

TABLE-6
EFFECT OF PS-Se NPs ON LEVELS OF LIVER SUPEROXIDE DISMUTASE (SOD) AND GLUTATHIONE PEROXIDASE (GPx), MALONDIALDEHYDE (MDA) AND REDUCED GLUTATHIONE (GSH) IN NORMAL AND EXPERIMENTAL GROUPS OF LEAD ACETATE-TREATED RATS

Groups	Treatment description	SOD	GPx	MDA (nmol/mg protein)	GSH (mg%)
I	Normal control A	22.70 ± 2.06 [d]	15.08 ± 0.81 [d]	1.25 ± 0.06 [a]	19.48 ± 1.64 [c]
II	LA (0.6 mg/kg b.w.)	7.44 ± 1.02 [a]	4.26 ± 0.44 [a]	3.64 ± 0.36 [b]	7.20 ± 0.83 [a]
III	PS-Se NPs (33.9 mg/kg b.w.) + LA (0.6 mg/kg b.w.)	15.55 ± 1.85 [b]	10.18 ± 0.74 [b]	1.40 ± 0.11 [a]	15.86 ± 1.54 [b]
IV	PS-Se NPs (84.75 mg/kg b.w.) + LA (0.6 mg/kg b.w.)	20.28 ± 2.55 [c]	15.40 ± 1.02 [c]	1.17 ± 0.19 [a]	20.00 ± 1.26 [c]
V	Vit. C (1 g/kg b.w.) + LA (0.6 mg/kg b.w.)	13.54 ± 0.68 [b]	9.75 ± 0.43 [b]	1.28 ± 0.16 [a]	17.42 ± 1.08 [b]

Values are given as mean ± SD for groups of six animals each. Values data followed by the same letter are not significantly different at $p \leq 0.05$. SOD: one unit of activity was taken as the enzyme reaction, which gave 50% inhibition of NBT reduction in 1 min/mg protein; GPx: µg of GSH consumed/min mg protein.

Histopathological test of the mammary tissue sections of the normal group (I) showed normal liver from control negative group showing normal hepatic parenchyma; note the normal polyhedral hepatocytes (h), blood sinusoids and central vein (c), (H&E X400) (Fig. 2a). On the other hand, liver from control positive group showing changes in the portal area; note the congested hepatoportal blood vessel (arrow head), hyperplastic bile duct (arrow) and leucocytic cells infiltration (*), (H&E X200) (Fig. 2b).

Histopathological test also showed showing liver from control positive group showing multi-focal mononuclear cells infiltration (arrows), (H&E X400). Lead acetate induced liver toxicity by PS-Se NPs exhibited as 33.9 and 84.75 mg/kg b.w. as compared with the lead acetate treated group and showed almost the same records as Groups III and IV (Fig. 2c&d). Group V all samples of lead acetate treated rats from treatment administered group showing apparently healthy and organized hepatic cords with slight congestion in the central vein (arrow), (H&E X400) by treatment with vitamin C (Fig. 2e).

Ultrasound scanning (US) examination of rats liver of the normal group I showed the liver measures from 3.5-3.6 cm in transverse diameter and the portal vein diameter between 1.5-1.8 mm and the parenchyma was homogenous with normal echogenicity (Fig. 3a). Also, the liver of lead acetate treated control group (II), US showed hepatic parenchyma contains variable sized circular and tubular anechoic anatomical structure that represent the hepatic and portal veins (Fig. 3b).

Ultrasound scanning also showed an abnormal well defined hepatic lesion was found in the right hepatic lobe, segment 8, with heterogenous texture and irregular borders, measures (2.1 mm × 1.4 mm) of lead acetate-treated rats with PS-Se NPs 33.9 mg/kg b.w., as compared with the lead acetate-treated rats (group III) (Fig. 3c). In addition, US liver examination of lead acetate-treated rats showed a marked improvement without inflammatory cells when treated with PS-Se NPs 84.75 mg/kg b.w., group (IV) (Fig. 3d). In addition, all samples of lead acetate-treated rats showed moderate lesion was regressed by treatment with vitamin C 1 g/kg b.w. group V (Fig. 3e).

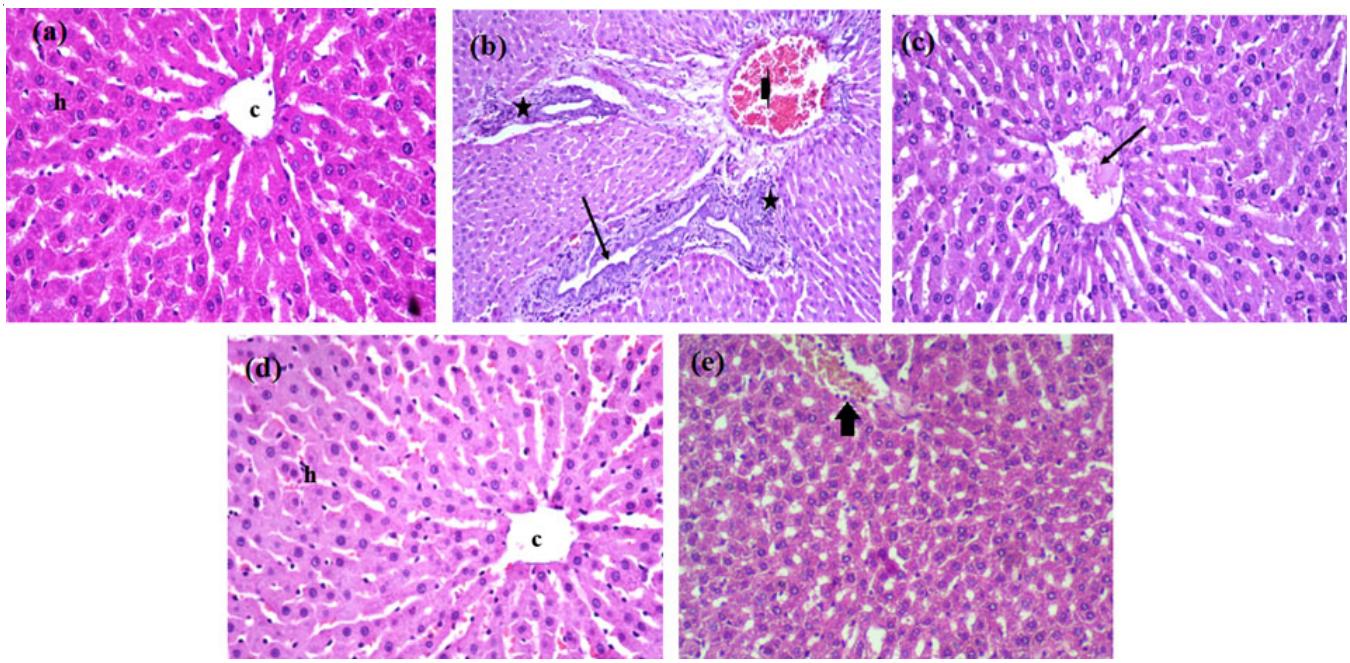

Fig. 2. Sections stained with hematoxylin and eosin (H&E; 400 X) histological examination of rats' liver tissue of different groups compared to control group; (a), Group I: Normal control; (b), Group II: lead acetate (0.6 mg/kg b.w.) (c); Group III: Was administrate PS-Se NPs (33.9 mg/kg b.w.) + PL (0.6 mg/kg b.w.) (d); Group IV: Was administrate PS-Se NPs (84.75 mg/kg b.w.) + PL (0.6 mg/kg b.w.); (e), Group V: Was administrate vitamin C (1 g/kg b.w.) + lead acetate (0.60 mg/kg b.w.)

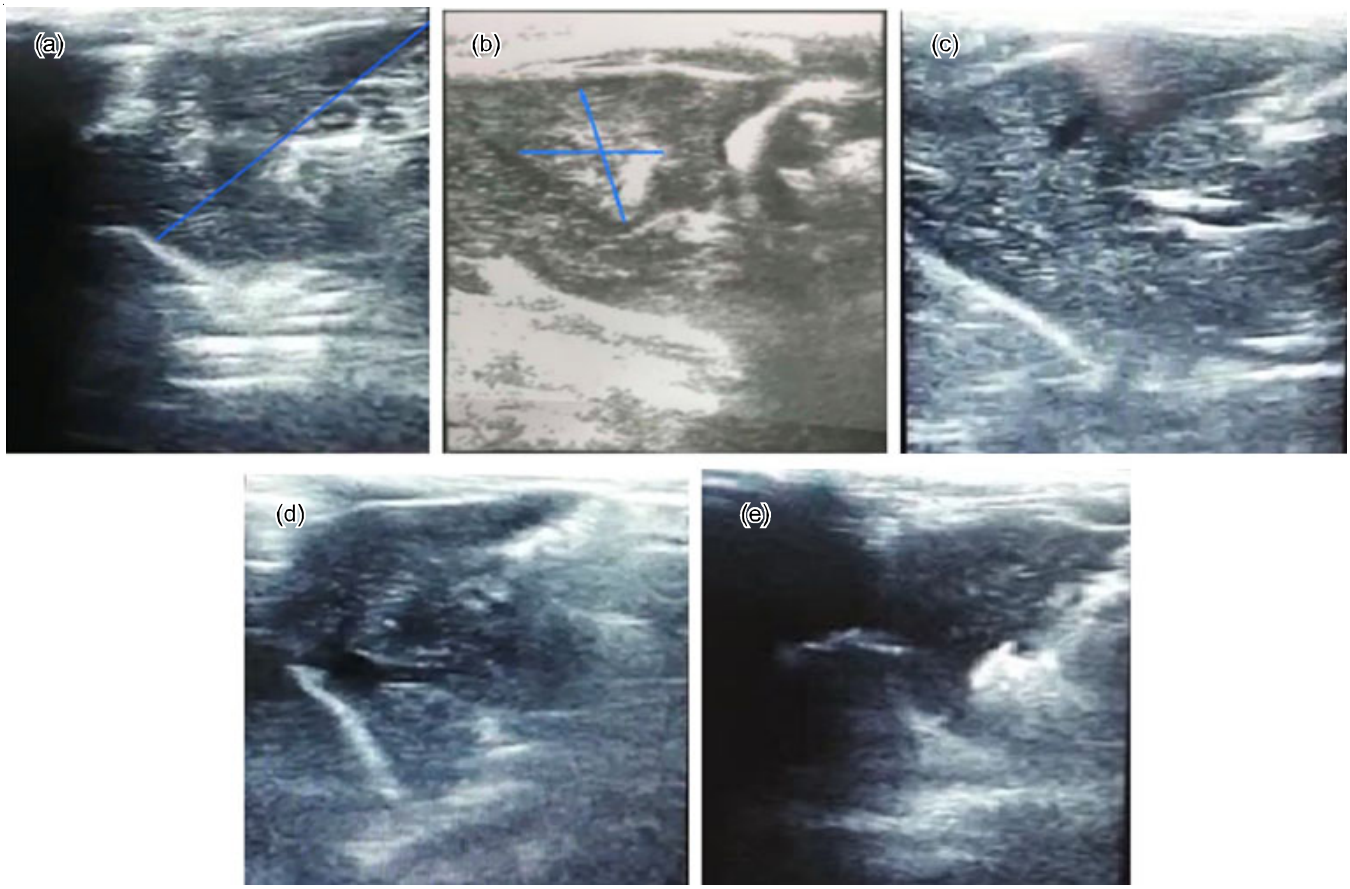

Fig. 3. Ultrasound scanning examination of rats liver tissue of different groups compared to control group; (a), Group I: Normal control; (b), Group II: lead acetate (0.6 mg/kg b.w.) (c); Group III: Was administrate PS-Se NPs (33.9 mg/kg b.w.) + PL (0.6 mg/kg b.w.) (d); Group IV: Was administrate PS-Se NPs (84.75 mg/kg b.w.) + PL (0.6 mg/kg b.w.); (e), Group V: Was administrate vitamin C (1 g/kg b.w.) + lead acetate (0.60 mg/kg b.w.)

The observed depletion of cytokines levels and increase in the activities of these antioxidant enzymes in liver tissues of PS-Se NPs treated rats compared to untreated rats reflects PS-Se NPs' antioxidant and anti-inflammatory potential. Several studies [22,23] have shown that PS-Se NPs containing flavonoids, tannins and other polyphenolic compounds, triterpenoids and a variety of other secondary plant metabolites have analgesic, antioxidant and anti-inflammatory properties in various animal models .

According to histological studies and US examinations, PS-Se NPs have a liver tissue-protective effect. Because liver tissue proliferation is an early event in toxicity-related changes, PS-Se NPs could be associated with a reduction in inflammatory response and induction of endogenous antioxidant enzymes in rats. To the best of my knowledge, no one has reported the prophylactic effect of PS-Se NPs against lead acetate-induced liver toxicity and this study may be the first of its kind.

Conclusion

The current study found that purslane seeds-selenium nanoparticles (PS-Se NPs) have potent anticancer activity against HpeG-2 breast carcinoma cell line and produce the protective activity against lead acetate-induced liver injury by normalizing the levels of oxidative stress biomarkers and inflammatory mediator gene expression.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interests regarding the publication of this article.

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