Hematological, Immunological and Histopathological Studies On The Effect Of Vanadium Pentoxide And Glyphosate In Albino Rats

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ABSTRACT

Two hundred clinically healthy female adult albino rats were divided into five main groups to evaluate the hematological and biochemical effects of both vanadium pentoxide and glyphosate . Gp. (1) contained 30 rats was kept as control. Gp. (2) contained 50 rats was orally given 1mg/kg B.W. vanadium pentoxide (1/10 LD_{50}) for 60 successive days. Gp. (3) contained 35 rats was orally given 0.5 mg/kg B.W. vanadium pentoxide (1/20 LD_{50}), for 60 successive days. Gp. (4) contained 50 rats was orally given 560 mg/kg B.W. glyphosate (1/10 LD_{50}) for 60 successive days. Gp. (5) contained 35 rats was orally given 280 mg/kg B.W. glyphosate (1/20 LD_{50}), for 60 successive days. The obtained blood samples which collected in a clean EDTA tube were used for determination of erythrogram and leukogram, beside collection of 3ml venous blood containing 150 IU heparin for phagocytosis. The liver, kidneys and spleen were immediately fixed in 10 % neutral buffered formalin for histopathological studies.

The results declared that the vanadium induce blood loss anemia as a result of erythrocyte destruction. The leukogram showed leukocytosis, lymphopenia and neutrophilia in rats treated with 1mg/kg B.W. vanadium pentoxide at the 60 days PT, while in rats treated with glyphosate the picture was leukopenia, lymphopenia and neutrophilia. Vanadium pentoxide1mg/ kg B.W. after 60 days affecting the cellular immunity represented by the decrease in phagocytic % and phagocytic index. Also, glyphosate treated rats (gps. 4&5) at the 30 and 45 days PT respectively till the end of the experiment decrease cellular immunity. Both vanadium and glyphosate have immunosuppressive effect.

It could be concluded that application of environmental pollutants for long periods have effects on the cellular immunity, so precautions must be taken during its application to protect public health from their effects.

INTRODUCTION

Environmental pollutants are hazardous substances which contaminate the environment; such contamination can result in acute or chronic health hazard on the population *(1)*. Vanadium is extensively distributed in nature. It is a trace element which is present in almost all-living organisms, including man. It has the ability to inhibit membrane sodium pump and affect the activities of various other intracellular enzyme systems *(2)*. Glyphosate is perhaps the most important organophosphate herbicide, which contains glyphosate (N-phosphonomethyl-glycine) as the active ingredient and polyoxyethyleneamine (POEA) as the surfactant agent. It is marketed as a non-selective, broad-spectrum, post-emergence herbicide due to its high water solubility and extensive usage. It is used to control weeds in emerged grasses, broad-leaf weeds, pastures and cultures such as rice, corn and soyabean *(3-4)*.

The aim of the present work was to investigate the hematological, immunological and histopathological effects of vanadium and glyphosate herbicide toxicity in albino rats.

MATERIAL AND METHODS

I-Material

Experimental rats

Two hundred clinically healthy female adult albino rats (120 g average body weight) were obtained from the Animal Laboratory House, Helwan, Egypt. The animals were kept in clean hygienic metal cages, divided into 5 main groups and acclimatized for 14 days. All animals were kept under hygienic conditions, given balanced free medication ration and drinking water were

allowed ad.libitum throughout the experimental period.

Chemicals

Vanadium (vanadium pentoxide) was obtained from Thomas Baker Chemical Industries PVT LTD, Mumbai, India.

Glyphosate 75% was obtained from Monsanto Agricultural Company.

I-Methods

Experimental design (Table 1)

Two hundred adult albino rats were divided into five groups. Gp. (1) was kept as control. Gp. (2) was orally given 1mg/kg B.W. vanadium pentoxide (1/10 LD_{50}) *(5)* for 60 successive days. Gp. (3) was orally given 0.5 mg/kg B.W. vanadium pentoxide (1/20 LD_{50}) *(5)* for 60 successive days. Gp. (4) was orally given 560 mg/kg B.W. glyphosate (1/10 LD_{50}) *(6)* for 60 successive days. Gp. (5) was orally given 280 mg/kg B.W. glyphosate (1/20 LD_{50}) *(6)* for 60 successive days.

Table 1.Experimental design

Group	*No of rats*	*Treatment*	*Dose*	*No & time of sacrified rats*
1	30	---------	-------	5 rats were sacrified at 7, 14, 30,45, and 60 day from the beginning of the experiment
2	50	Vanadium pentoxide	1 mg/kg	
3	35	Vanadium pentoxide	0.5 mg/kg	
4	50	Glyphosate	560 mg/kg	
5	35	Glyphosate	280 mg/kg	

Blood sampling

The blood samples were collected from the retro-orbital venous plexus of rats, in a clean EDTA tube and used for the hematological examinations. For phagocytosis, 3ml venous blood containing 150 IU heparin were collected.

Tissue specimens

The liver, kidneys and spleen were immediately fixed in 10 % neutral buffered formalin for histopathological studies.

Hematological techniques

The total erythrocytic count was performed using the improved Neubauer hemocytometer and Gower's solution. The hemoglobin was determined using Drabkin's solution *(7)*. The packed cell volume was determined by microhematocrit centrifuge. Blood indices (MCV and MCHC) were calculated. Blood films were stained with New methylene blue stain for the reticulocytic count *(8)*. The total leukocytic count (TLC) was done using improved Neubauer hemocytometer and Turkey's fluid. Blood films were stained by Giemsa stain and the differential leukocytic counts were performed and then the absolute counts were calculated for each type of cells *(9)*.

Evaluations of phagocytic activity & phagocytic index

This method is based on the uptake of C.albicans by monocytes over a certain period of time.Ten fields, each containing about 10 phagocytes, were examined under the oil-immersion lens of a light microscope. The total number of phagocytes which ingested yeast cells was determined to calculate the percentage of phagocytosis and phagocytic index *(10)*.

Phagocytic % is the number of monocytes ingesting C. albicans=

$$\frac{\text{Number of monocytes containing C. albicans}}{\text{Total number of counted monocytes}}$$

Phagocytic index (P.I) is the number of C.albicans ingested by 100 monocytes

$$=\frac{\text{Total number of C. albicans in 100 monocytes}}{100}$$

Histopathology

Rats were sacrificed and necropsied after 7, 14, 30, 45 and 60 day from the beginning of the experiment. The liver, kidneys and spleen were immediately fixed in 10 % neutral buffered formalin for histopathological studies *(11).*

Statistical analysis

Data was collected and then analyzed by using the computer program, the statistical method was one way ANOVA (F test), LSD (Least significant difference) *(12)* to estimate the effect of different treated groups on different parameters. Data were presented as mean ± SE and significance was declared at ($P \leq 0.05$). Means followed by different letters differed significantly and the highest value was represented with the letter a.

RESULTS AND DISCUSSION

Vanadium administered rats (gps.2&3), showed a decrease in RBC count, hemoglobin and packed cell volume (at the 30 days till the end of the experiment) (Table 2) due to hemolysis caused by pro-oxidative action of vanadium *(13)* which reduced the activity of Na^+, K^+ ATPase, leading to the disturbances of sodium and potassium gradient on both sides of the cell membrane *(14).* Furthermore, vanadium inhibits the plasma membrane Ca^{2+}ATPase activity *(15)* and may open an L type-like Ca^{2+} channel in red blood cells *(16).* This situation enhances erythrocytes swelling and leads to haemolysis. Macrocytic hypochromic anemia with reticulocytosis which observed at 30 days till the end of the experiment in rat received vanadium pentoxide (gps.2&3) may be due to the reduction of vanadate to vanadyle in the red blood cells *(17)* so lead to destruction of erythrocytes which in turn stimulate the bone marrow to push reticulocytes into the circulation. A decrease in erythrocytic-count and hemoglobin levels with an increase in reticulocytes were recorded in rats orally exposed to 1.18 mg vanadium/kg/day as ammonium metavanadate for 4 weeks *(18).* Aqueous solution of ammonium metavanadate (AMV) at different concentration in drinking-water (sole drink) for 4 weeks to Wistar rats of both sexes showed a decrease in the erythrocytic-count, hemoglobin concentration and hematocrit value *(19-22).* Rats administered aqueous solution of ammonium metavanadate (AMV) (0.15 mg V/ml) for 14 days showed no changes in the erythrocytic-count and hemoglobin concentration *(23).* Meanwhile, a slight increase in hematocrit value was observed. On the contrary rats exposed intratracheally once a month to 0.56 mg V_2O_5/kg B.W. for 12 months, no changes in the erythrocytic-count, hemoglobin level and the peripheral blood indices were recorded *(24).* Also, it has been demonstrate the absence of any significant changes in haematological indices *(25,26).* The differences between our results and the results that were obtained by the above cited authors may be due to of various chemical forms of vanadium, doses and different times of treatment.

Glyphosate administration to normal rats (gps.4&5) did not show any significant change in erythrogram (RBCs-count, hemoglobin, PCV and reticulocytes) throughout the experimental periods (Table 2). Our results are consistent with WHO report *(27).* Meanwhile, our results were disagree with *(28)* who found mild increase in hematocrit and RBC-count in male rats, 13 weeks after oral administration of 25000 and 50000 ppm glyphosate with significant increase in MCH, and MCV in female rats. Also *(29)* who found a decrease in the hematocrit, erythrocyte and hemoglobin-content in teleost fish, *Leporinus obtusidens* (piava), exposed to different concentrations of Round up , a glyphosate (acid equivalent) herbicide: 3, 6, 10, and 20 mg/L for 96 h (short-term).The

difference may be due to the dose of glyphosate and duration of the experiment.

The leukocytosis which observed in gp. 3 at 30 days , till the end of the experiment (Table 3) was due to lymphocytosis which may be due to an early antigenic stimulation with increased T-lymphocytes and neutrophilia could be due to hemolysis and toxicity by chemicals *(30).* Wister rats administered aqueous solution of ammonium metavanadate (0.15 mg V/ml) for 4 weeks showed an increase in the neutrophilic granules and lymphocytic-count *(17).* On the other hand, leukocytosis, observed in gp. (2) at 60 day PT (Table 3) was due to neutrophilia which may be due to tissue distruction *(8).* It was accompanied with lymphopenia which may be due to the immunosuppressive effect of the vanadium with necrosis of lymphoid tissues *(31).* Also, lymphopenia could be due to lymphocytic migration to the site of tissue to play their role in the defense mechanism. This result gets along with the histopathological lesions in the liver which showed interstitial lymphocytic aggregations (Fig.1). The kidneys showed focal replacement of renal parenchyma with lymphocytes (Fig.2). On contrary it has been recorded a significant decrease in leukocytic counts in both the 50 and 100 ppm vanadium-fed groups after 10 weeks. Also, no significant changes in WBC count in male Wistar rats that received vanadyl sulphate (0.5 mg/ml) in drinking water for 52 weeks *(25).* Male rats orally administered 9.7 mg ammonium metavanadate/ kg B.W./day, vanadyl sulphate 0.15 mmol V/kg B.W./day and bis (maltolato) oxovanadium 0.18 mmol V/ kg B.W./day in drinking water for 12 weeks showed a nonsignificant changes in leukocytic-count at 1, 2, 4, 8, and 12 weeks PT*(26).* Moreover, a non significant change in the leukocyic count was observed in rats orally treated with 0.05mg/kg B.W. vanadium pentoxide. The differences between our results and the results that were obtained by the above cited authors may be due to the difference in the dose of vanadium and duration of the experiment *(33).*

Glyphosate treated groups showed leukopenia, lymphopenia and neutrophilia at 30 days in gp.4 and at 45 day in gp.5 that continued till the end of the experiment in both groups (Table 3) which may be due to necrosis of lymphoid tissues *(31).*This result get along with the histopathological lesions in the spleen which showed necrosis and depletion of lymphoid elements and thickened splenic capsule reflected the encountered lymphopenia (Figs.3&4). The neutrophilia may be due to tissue destruction *(8).* Leukopenia, lymphopenia and monocytosis were recorded in all Round up poisoned rabbits receiving 10.000, 20.000 and 30.000 ppm in drinking water for 16 weeks. The difference may be due to the difference in the animal species, the dose of glyphosate and duration of the experiment. However, a significant increase in the lymphocytes at a dose ≥25,000 ppm and white blood cells (WBC) at a dose ≥ 12,500 ppm in 10 F344N was recorded in rats or B6C3F1 mice orally administered glyphosate in the diets for 13 weeks *(28).* The differences between our results and the results that were obtained by the above cited authors may be due to the difference in the dose of glyphosate and duration of the experiment.

The monocytes, eosinophils and basophils in both vanadium and glyphosate treated rats did not show any significant changes. Similarly a non significant change in the differential leukocyic count in rats orally treated with 0.05mg/kg B.W. vanadium pentoxide *(33).* On the other hand, it has been noticed monocytosis in all Round up poisoned rabbits receiving 10.000, 20.000 and 30.000 ppm of herbicide in drinking water for 16 weeks *(34).* The difference may be due to the difference in the animal species, the dose of glyphosate and duration of the experiment.

The decrease in phagocytic % and phagocytic index in (gp.2) at 60 days and in glyphosate treated rats (gps. 4&5) at 30 and 45 day PT respectively (Table 4) indicate the immunosuppressive effect of both vanadium and glyphosate. This effect may be due to the stress of toxic materials in addition to depletion of lymphocytes. All these factors may interfere with lymphocytic ability to proliferate or differentiate. Depression of phagocytosis was recorded *(35),* in the adult ICR mice treated with 6 mg/kg B.W. vanadium pentoxide orally by

gavage (5 days / week for 6 weeks) and in Wister rats given vanadium pentoxide in drinking-water (1 or 100 mg/L for 6 months). Also a short exposure to Roundup (10 min at concentration of 100,000 ug/L) in carp *(Cyprinus carpio)* and European catfish *(Silurus glanis)* caused a decrease in phagocytic activity *(36)*. Vanadium administration to rats at a dose of 0.5mg/ kg B.W. did not show any effects on the phagocytic % and phagocytic index. This may be due to the low dose used. Histopathological examination of spleen in the present study confirmed the result of phagocytosis, in which the spleen showed necrosis and depletion of lymphoid elements and edema of septa together with thickened splenic capsule (gp.2) at 60 days and in glyphosate treated rats (gps. 4&5) at 30 and 45 day PT respectively.

It could be concluded that application of environmental pollutants for long periods have effects on the cellular immunity, so precautions must be taken during its application to protect public health from their effects.

Table 2. Erythrogram after 7, 14, 30, 45 & 60 days from the beginning of the experiment (mean ±S.E.) (No=5).

Days& gps.	RBCs (10^6/µl)	Hb (gm%)	PCV (%)	MCV (fl)	MCHC (%)	MCH (pg)	Reticulocytes (%)
7 Gp.1	6.71±0.28	14.80±0.49	44.60±1.2	66.64±1.05	33.18±0.61	22.11±0.48	1.48±0.04
Gp.2	6.59±0.27	14.44±0.53	44.20±0.58	67.38±1.94	32.63±0.82	21.93±0.3	1.43±0.07
Gp.3	6.65±0.08	14.72±0.16	44.20±0.58	66.45±0.3	33.31±0.08	22.13±0.10	1.48±0.04
Gp.4	6.74±0.16	14.76±0.19	44.60±0.40	66.26±1.3	33.09±0.15	21.92±0.34	1.50±0.04
Gp.5	6.68±0.08	14.68±0.12	44.80±0.58	67.06±0.58	32.78±0.27	21.98±0.11	1.50±0.09
14 Gp.1	6.80±0.17	14.89±0.23	44.80±0.66	65.76±0.74	32.24±0.14	21.92±0.22	1.48±0.13
Gp.2	6.77±0.11	14.69±0.20	45.00±0.44	66.53±0.59	32.65±0.28	21.72±0.28	1.50±0.07
Gp.3	6.80±0.07	14.68±0.17	45.00±0.89	66.14±0.79	32.64±0.27	21.58±0.17	1.50±0.04
Gp.4	6.86±0.09	14.72±0.11	44.60±0.24	65.07±1.12	33.01±0.37	21.46±0.25	1.38±0.03
Gp.5	6.88±0.06	14.78±0.13	44.80±0.73	65.15 ±1.51	33.05±0.78	21.48±0.06	1.43±0.07
30 Gp.1	6.61±0.15 a	14.6±0.27 a	44.60 ±0.67 a	67.49 ±0.68 b	32.93±0.37 a	22.22±0.32	1.53 ±0.11 b
Gp.2	4.44±0.10 b	10.22±0.14 b	36.80 ±0.48 b	83.06 ±1.15 a	27.76 ±0.23 b	23.06±0.31	2.46 ±0.05 a
Gp.3	4.52±0.09 b	10.13 ±0.05 b	36.40 ±0.24 b	80.62±1.12 a	27.82±0.07 b	22.43±0.34	2.38 ±0.06 a
Gp.4	6.64 ±0.11a	14.59 ±0.16 a	44.40 ±0.24 a	66.96 ±0.81 b	32.86 ±0.20 a	22.00±0.18	1.48 ±0.09 b
Gp.5	6.66±0.10 a	14.67±0.11 a	44.80 ±0.96 a	67.25 ±0.67 b	32.78 ±0.49 a	22.03±0.19	1.50 ±0.08 b
45 Gp.1	6.85±0.08 a	14.64±0.24 a	44.80±0.73 a	65.43±0.43 b	32.67±0.13 a	21.37±0.12	1.67±0.08 b
Gp.2	4.57±0.16 b	10.34±0.10 b	37.40±0.40 b	82.20±2.25 a	27.64±0.11 b	22.72±0.62	2.48±0.08 a
Gp.3	4.71±0.24 b	10.41±0.09 b	37.80±0.37 b	80.90±3.29 a	27.55±0.06 b	22.28±0.91	2.40±0.11 a
Gp.4	6.80±0.07 a	14.71±0.09 a	44.60±0.67 a	65.58±0.48 b	33.00±0.31 a	21.63±0.13	1.60±0.11 b
Gp.5	6.87±0.07 a	14.72±0.10 a	44.80±0.73 a	65.16 ±0.43 b	32.88±0.56 a	21.42±0.24	1.57±0.06 b
60 Gp.1	6.77±0.11 a	14.72±0.22 a	44.80±0.58 a	66.23±0.78 c	32.86±0.23 a	21.75±0.16	1.50±0.10 b
Gp.2	4.34±0.04 b	10.28±0.08 b	37.60±0.74 b	86.65±1.03 a	27.38±0.36 b	23.71±0.17	2.56±0.08 a
Gp.3	4.67±0.20 b	10.35±0.10 b	37.80±0.80 b	81.25±1.92 b	27.40±0.35 b	22.28±0.74	2.48±0.08 a
Gp.4	6.70±0.08 a	14.78±0.28 a	45.00±1.04 a	67.12±0.89 c	32.85±0.29 a	22.04±0.21	1.47±0.08 b
Gp.5	6.75±0.11 a	14.88±0.27 a	45.00±1.22a	66.6±0.89 c	33.09±0.30 a	22.0±0.12	1.52±0.07 b

Means within the same column at the same period carrying different letters are significant at ($p \leq 0.05$)
Each value represents the mean ± SE

Table 3. Leukogram (x 10^3/µl) after 7, 14, 30, 45 & 60 day from the beginning of the experiment (mean ±S.E.) (No=5).

Days& gps.	TLC	Lymphocytes	Neutrophils	Monocytes	Eosinophils	Basophils
7 Gp.1	8.81±0.13	6.17±0.18	2.18±0.10	0.37±0.02	0.09±0.00	0
Gp.2	8.78±0.08	6.22±0.10	2.11±0.08	0.37±0.02	0.08±0.001	0
Gp.3	8.89±0.08	6.35±0.10	2.06±0.04	0.40±0.02	0.08±0.001	0
Gp.4	8.74±0.21	6.22±0.18	2.06±0.10	0.37±0.01	0.09±0.002	0
Gp.5	8.90±0.21	6.32±0.19	2.13±0.04	0.36±0.01	0.09±0.002	0
14 Gp.1	8.99±0.15	6.38±0.14	2.16±0.04	0.36±0.006	0.09±0.002	0
Gp.2	9.11±0.12	6.54±0.11	2.11±0.05	0.37±0.004	0.09±0.001	0
Gp.3	9.13±0.13	6.50±0.15	2.17±0.03	0.37±0.005	0.09±0.001	0
Gp.4	8.97±0.2	6.44±0.17	2.08±0.04	0.36±0.008	0.09±0.002	0
Gp.5	9.0±0.17	6.49±0.13	2.07±0.06	0.36±0.007	0.09±0.002	0
30 Gp.1	9.10±0.2 b	6.57±0.17 b	2.07±0.03 b	0.37±0.03	0.09±0.02	0
Gp.2	11.29±0.07 a	8.08±0.04 a	2.73±0.05 a	0.38±0.03	0.10±0.02	0
Gp.3	11.08±0.07 a	8.00±0.06 a	2.61±0.08 a	0.38±0.03	0.09±0.02	0
Gp.4	7.52±0.12 c	4.50±0.04 c	2.57±0.10 a	0.36±0.01	0.09±0.03	0
Gp.5	8.98±0.06 b	6.45±0.03 b	2.08±0.05 b	0.36±0.03	0.09±0.06	0
45 Gp.1	9.22±0.06 b	6.66 ±0.01 b	2.10±0.04 c	0.37±0.002	0.09±0.06	0
Gp.2	11.50±0.16 a	8.21±0.13 a	2.83±0.02 a	0. 37±0.03	0.09±.0.02	0
Gp.3	11.48±0.16 a	8.20±0.18 a	2.80±0.03 a	0.39±0.0	0.09±0.02	0
Gp.4	7.54±0.13 c	4.46±0.10 c	2.61±0.05 b	0.38±0.07	0.09±0.03	0
Gp.5	7.62±0.07 c	4.56±0.65 c	2.61±0.37 b	0.36±0.02	0.09±0.03	0
60 Gp.1	9.30±0.10 b	6.71±0.07 b	2.12±0.06 d	0.38±0.03	0.09±0.01	0
Gp.2	11.62±0.06 a	5.04±0.08 c	6.11±0.05 a	0.38±0.04	0.09±.0.02	0
Gp.3	11.54±0.09 a	8.24±0.04 a	2.84±0.04 b	0.37±0.05	0.09±0.02	0
Gp.4	7.54±0.10 c	4.52±0.07 d	2.55±0.05 c	0.38±0.05	0.09±0.03	0
Gp.5	7.55±0.11 c	4.50±0.07 d	2.58±0.04 c	0.38±0.06	0.09±0.03	0

Means within the same column at the same period carrying different letters are significant at ($p \leq 0.05$)
Each value represents the mean ± SE.

Table 4. Phagocytic % (P%) & Phagocytic index (PI) after 7, 14, 30, 45 &60 day from the beginning of the experiment (mean ±S.E.) (No=5).

Gps.	The days from the beginning of the experiment and the parameters									
	7		14		30		45		60	
	P%	PI	P%	PI	P%	PI	P%	PI	P%	PI
1 (Control)	52.40 ±0.92	0.87 ±0.02	52.40 ±0.92	0.87 ±0.02	52.4 ^{a} ±0.92	0.87^{a} ±0.02	52.4^{a} ±0.92	0.87^{a} ±0.02	52.40^{a} ±0.92	0.87 ^{a} ±0.02
2 V_2O_5 (1mg/kg B.W.)	54.40 ±0.74	0.83 ±0.01	55.60 ±0.74	0.81 ±0.01	49.80^{a} ±0.58	0.81^{a} ±0.008	48.00^{a} ±0.44	0.80^{a} ±0.009	35.60 ^{bc} ±0.92	0.70 ^{b} ±0.01
3 V_2O_5 (0.5mg/kg B.W.)	53.20 ±1.15	0.86 ±0.004	53.00 ±0.70	0.85 ±0.01	49.00^{a} ±1.18	0.86^{a} ±0.01	49.60^{a} ±1.63	0.86^{a} ±0.007	49.40 ^{a} ±1.36	0.85^{a} ±0.01
4 Glyphosate (560mg/kg B.W.)	50.00 ±1.0	0.85 ±0.007	54.40 ±0.92	0.81 ±0.01	37.80^{b} ±1.15	0.70^{b} ±0.007	36.00^{b} ±0.83	0.69^{b} ±0.01	33.40 ^{c} ±1.32	0.67^{b} ±0.007
5 Glyphosate (280mg/kg B.W.)	52.00 ±1.22	0.85 ±0.01	54.00 ±1.14	0.84 ±0.01	49.00^{a} ±1.78	0.81^{a} ±0.01	38.80^{b} ±2.00	0.68^{b} ±0.006	38.20^{b} ±2.1	0.65^{b} ±0.01

Means within the same column at the same period carrying different letters are significant at ($p \leq 0.05$)
Each value represents the mean ± SE

Histopathological finding

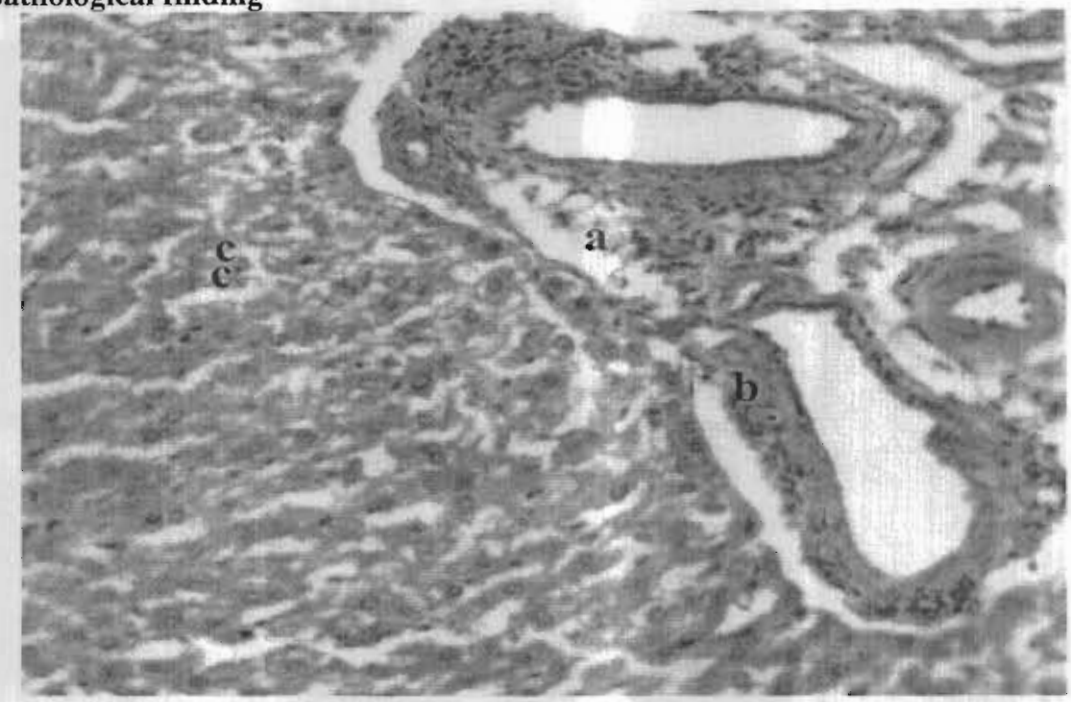

Fig. 1. Section of rat liver from group 2, sixty days post treatment showing portal edema (a), lymphocytes and fibroblast proliferation (b) and vacuolar degeneration of the hepatocytes (c) at 60 day PT, H & E. , X300.

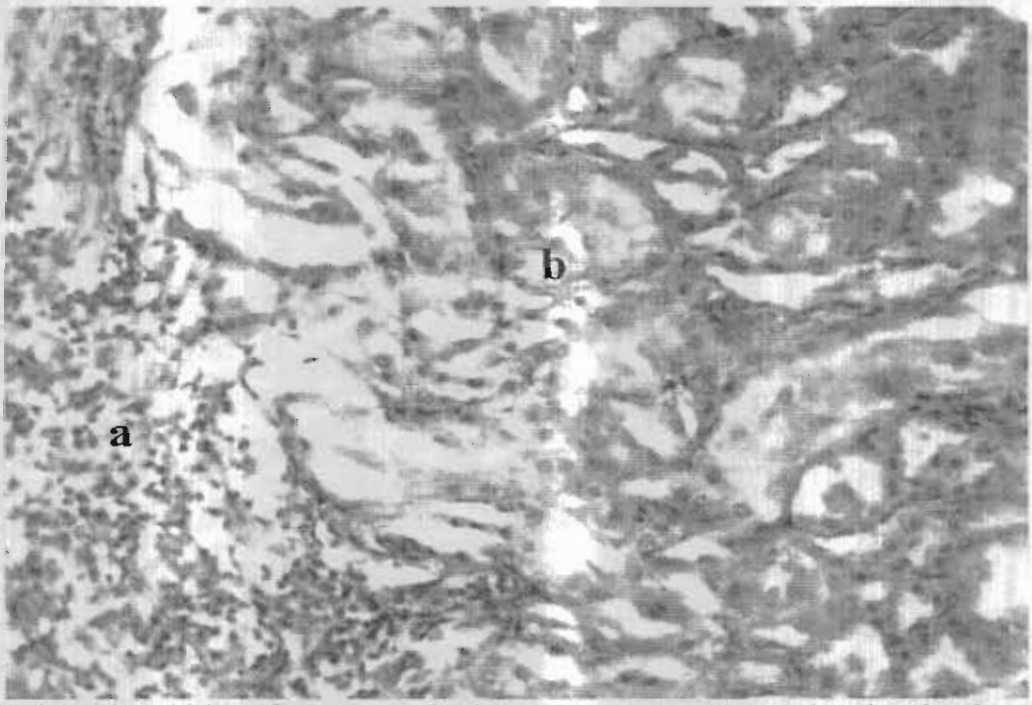

Fig. 2. Section of rat kidney from group 2, sixty days post treatment showing focal replacement of renal parenchyma with lymphocytes (a) and nephrotic changes in the renal tubules (b). H & E., X 300.

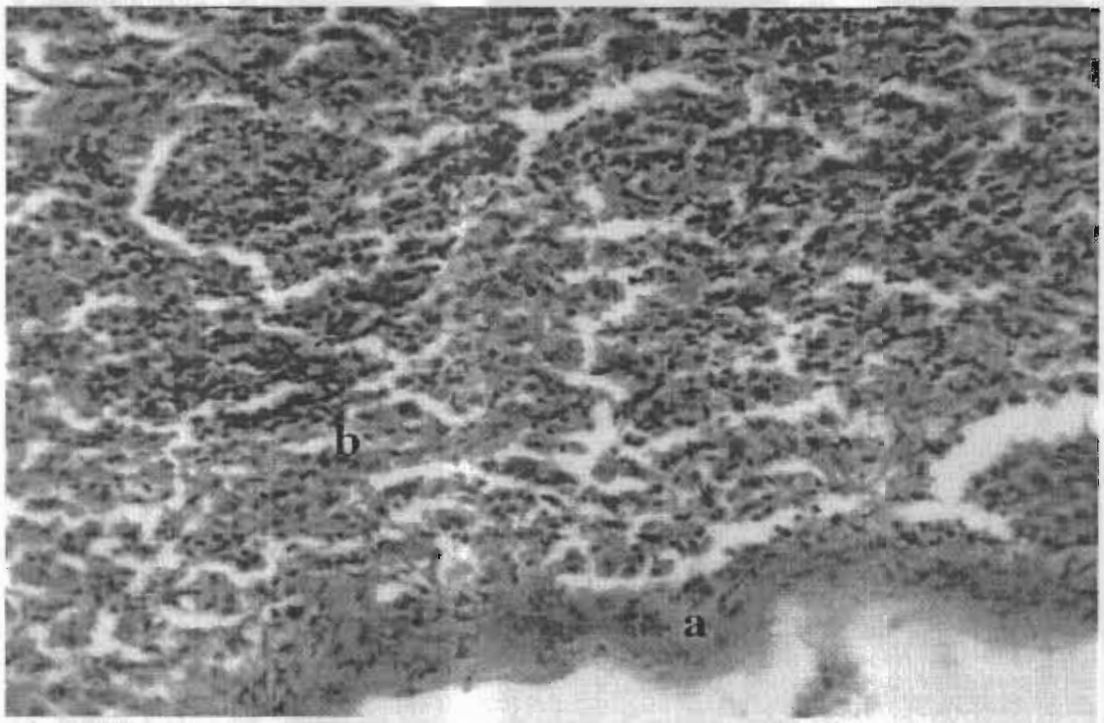

Fig. 3. Section of rat spleen from group 4, sixty days post treatment showing thickened splenic capsule (a) and lymphoid depletion (b). H & E. , X 300.

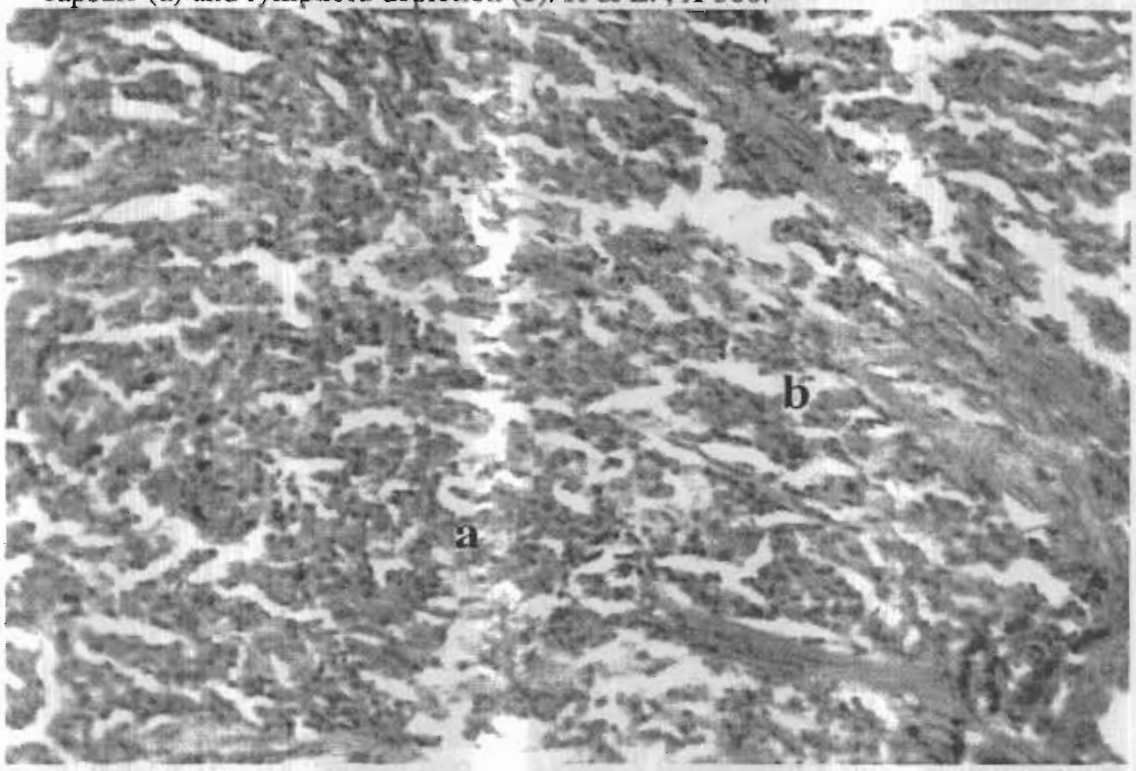

Fig. 4. Section of rat spleen from group 5, sixty days post treatment showing lymphoid depletion (a) and edema (b). H & E., X300.

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الملخـص العربي

دراسات هيماتولوجية ومناعية وهستوباثولوجية على تأثيرخامس أكسيد الفاناديم والجليفوسات فى الفئران البيضاء

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أجري البحث على 200 من إناث الفئران البيضاء وقد تم تقسيمها إلى خمس مجموعات:- المجموعة الأولى : مكونة من 30 فأرا تركت كضابط للتجربة.المجموعة الثانية : مكونة من 50 فأرا تم إعطاؤها خامس أكسيد الفاناديوم (1 ملجم / كجم من وزن الجسم) عن طريق الفم يوميا لمدة 60 يوما. المجموعة الثالثة : مكونة من 35 فأرا تم إعطاؤها خامس أكسيد الفاناديوم (0.5 ملجم / كجم من وزن الجسم) عن طريق الفم يوميا لمدة 60 يوما. المجموعة الرابعة: مكونة من 50فأرا تم إعطاؤها الجليفوسات (560 ملجم / كجم من وزن الجسم) عن طريق الفم يوميا لمدة 60 يوما.المجموعة الخامسة: مكونة من 35 فأرا تم إعطاؤها الجليفوسات (280 ملجم / كجم من وزن الجسم) عن طريق الفم يوميا لمدة 60 يوما. وتم تجميع عينات الدم من المجموعات الخمسة السابقة باستخدام أنابيب شعرية تم إدخالها فى التجمع الدموى العينى من الجهة الداخلية للعين على خمس فترات (بعد 7, 14, 30, 45,60 يوما) من بداية التجربة على مانع للتجلط (EDTA) لإجراء التحاليل الدموية لخلايا الدم كما تم تجميع عينات دم على هيبارين لقياس المناعة الخلوية . ثم ذبحت الفئران فى نفس المدد السابقة وتم أخذ عينات من كل من (الكبد- الكلية و الطحال) لإجراء الصفة التشريحية لهذه الفئران . و قد أظهرت تحاليل صورة الدم فى المجموعتين (2&3) وجود نقص معنوى فى كرات الدم الحمراء وتركيز الهيموجلوبين وحجم خلايا الدم الحمراء المضغوطة مع وجود زيادة فى الخلايا الشبكية عند 30 يوما. وزادت هذه التغيرات تدريجيا مع زيادة الوقت. أظهرت هاتان المجموعتان أنيميا ذات الحجم الكبير ونقص تركيز الهيموجلوبين. وقلت هذه التغيرات فى المجموعة (3) مقارنة بالمجموعة (2) . أما المجموعتين (4&5) فلم تتغير بهما هذه القياسات فى كل الفترات. كما ظهرت زيادة معنوية فى العدد الكلى والنوعى لكرات الدم البيضاء ممثلة فى زيادة عدد الخلايا متعادلة الصبغة والخلايا الليمفاوية فى المجموعتين(3,2) عند 30 يوم وحتى نهاية التجربة فيما عدا نقص معنوى فى الخلايا الليمفاوية فى المجموعة (2) عند 60 يوما من إعطاء الفاناديوم. أما المجموعتين (4&5) فأظهرتا نقص معنوى فى العدد الكلى لكرات الدم البيضاء والخلايا الليمفاوية مع وجود زيادة فى الخلايا متعادلة الصبغة عند 30 يوما فى المجموعة (4) وعند 45 يوما فى المجموعة (5) واستمرت حتى نهاية التجربة. كما لوحظ أيضا عدم وجود أى تغيير فى الخلايا حمضية الصبغة والخلايا قاعدية الصبغة والخلايا وحيدة النواة فى جميع المجموعات فى كل الفترات. وبالكشف عن المناعة الخلوية وجد حالة تثبيط مناعى ظهرت معنويا عند 60 يوما فى المجموعة (2) وفى المجموعتين (4&5) عند 30 و45 يوما على التوالى واستمرت حتى نهاية التجربة وذلك بناء على وجود نقص معنوى فى نسبة التهام العدوى والقدرة على قتل هذه العدوى.

ويتضح من هذه الدراسة مدى تأثير كلا من الفاناديوم والجليفوسات على مكونات الدم وتأثيرهم على المناعة الخلوية لذلك يجب توخى الحذرعنداستخدام هذه الملوثات البيئية وعدم استخدامها لفترة طويلة لما لها من تأثيرسلبى على صحة الإنسان.