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RESEARCH ARTICLE

Histo-Chemical Effects of Iraqi-Camel Colostrum Extracts on Some Liver Biomarkers in Albino Rats

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Abstract

This study was intended to investigate the histo-chemical effects of Iraqi camel (*Camelus dromedaries*) colostrum on liver tissues and enzymes of albino rats, *Rattus norvegicus*. Colostrum was collected at 30ml at post-parturition times of 0, 6, and 12hr. The colostrum was subjected to an extraction procedure to obtain a filtered fluid that contained bioactive substances. These extracted fluids were given to 4 groups/5 rats each. Group1 (0G), Rats were given 0.1ml of 0hr-colostrum extract orally for 1 week. Group2 (6G), Rats were given 0.1ml of 6hr-colostrum extract orally for 1 week. Group3 (12G), Rats were given 0.1ml of 12hr-colostrum extract orally for 1 week. Group4 (control group, CG), Rats were given tap water only. Body and liver weights were measured every week for 4 weeks. Blood samples and liver samples were collected after 30 days to perform assays of measuring serum proteins and liver enzymes. The results indicate richness of colostrum with protein and fat. The results also revealed increases the levels of liver enzymes and changes in the tissues of the livers. These levels increased especially at 30 days of the experiment for the groups that were given camel colostrum extracts. These results indicate important effects of camel colostrum to prepare and protect body against various environmental intruders such as pathogens. Our results generate important information for future work to thoroughly study colostrum extracts on infections in humans and animals.

Keywords: *Camel, Colostrum, Liver enzymes, Rats.*

Introduction

In the Arab countries, the one-humped camel (*Camelus dromedaries*) was considered as a ship of desert. They were utilized for moving between far places. Milk, meat, and wool are good sources that are provided by camels. In the last decade, many researchers focused on camel products, and one of the most important products was the post-parturition biological fluid, colostrum.

According to our knowledge, there have been no studies that were focused on the histo-chemical effects of camel colostrum on liver enzymes. Many researchers studied the histo-chemical effects of many different chemical materials on liver enzymes [1-5]. However, [6-10] studied the effects of camel milk on these enzymes. Camel colostrum contains high levels of proteinous, non-proteinous, non-protein nitrogenous, vitamin, ash, and mineral materials [11]. It has major milk proteins such as a-Lactalbumin, lactophorin, serum albumin, and whey protein [12, 13]. Camel colostrum is rich in lactoferrin contents at an amount of 2.3 gL^{-1} in comparison to 0.5 gL^{-1} in bovine colostrum. This substance is responsible for antimicrobial activity of camel colostrum [14].

Immunoglobulin-based agents of camel colostrum are three main sub-classes which are IgG, IgG2, and IgG3 [15]. In addition, [16] reported that IgG2 and IgG3 devoid of light chains and had molecular masses of 42 and 45 KDa, respectively. These features of proteinous contents of the camel colostrum might reflect a particular biological activity

against various harmful substances of exogenous- or endogenous-based origins. Few studies have mentioned that colostrum of camels contains high amounts of bioactive molecules such as antioxidant and antihypertensive peptides that are released after enzymatic hydrolysis via fermentation of milk [17]. The two essential enzymes are AST and ALT that are released from the liver following tissue damages. The activities of AST are increased in serum [18].

ALT is found in the cystosol of the liver cells only. On the other hand, AST is found in both mitochondria and cystosol. ALT is used in liver damages more than AST level. The levels of these enzymes might also be increased as a result to deficiencies or diseases in other body organs [19]. When rats are fed buffalo colostrum [20], significant decreases of AST and ALT were seen, as indications for improvement of liver dysfunction.

Also [21] studied the effects of camel milk on liver functions and found significant development in liver function parameters, e.g. ALT and AST activities. For the big value of our camels, this study was conducted to test the histo-chemical effects of camel colostrum on the liver enzymes, ALT and AST, Total protein, and LIC.

Material and Methods

The study was performed in the College of Veterinary Medicine, University of Al-Qadisiyah (October-November, 2016).

Experimental Design

Iraqi camel, *Camelus dromedarius*, colostrum at 30ml was collected during post-parturition times of 0, 6, and 12hr in sterile containers and sent directly in ice to a laboratory in the mentioned college. Then, the samples were centrifuged twice at 5000rpm for 10min. After that, the supernatants were filtrated twice using microfiltration system containing microfiltration membrane, 0.22µm, and the extracts were kept at 4℃ waiting for the next process.

Twenty albino rats, *Rattus norvegicus*, (54.3±2.06gm) that were obtained from the animal care unit, Department of Physiology, College of Veterinary Medicine, University of Al- Qadisiyah were subjected to this study.

Gross Composition Analysis

The pH of the samples was measured using pH meter. Viscosity was determined via applying a shear stress of 0.1 to 100rpm at oscillation frequency of 1Hz for 1min with a viscometer (DV-E, USA). Lipid profile was determined utilizing acid-butyrometric-Gerbar method using [21]. Crude proteins of colostrum were detected using Kjeldahl method, NX 6.38, [21]. Dry matter was calculated via the remaining residues after samples had been exposed to 105℃ for 24hr, g/L of colostrum. Ash content was determined after dry_mineralization at 505ºC had been done, gL¯1 of colostrum [21].

The animals were randomly categorized into 4 groups/ 5 rats each. Group1 (0G), Rats were given 0.1ml of 0hr-colostrum extract orally for 1 week. Group2 (6G), Rats were given 0.1ml of 6hr-colostrum extract orally for 1 week. Group3 (12G), Rats were given 0.1ml of 12hr-colostrum extract orally for 1 week. Group4 (control group, CG), Rats were given tap water only.

Collection of Blood Samples and Preparation of Liver Homogenate

After 30 days, all the rats were sacrificed. Blood samples were immediately collected and spun down using a centrifuge at 5000rpm for 5 min to generate serum. After dissecting the carcasses, livers were quickly excised, weighed, and transferred into tubes preloaded with 0.25M ice-cold sucrose solution. The weights of the bodies and livers were recorded at 1, 2, 3, and 4 weeks. The livers were then homogenized in ice-cold 0.25M sucrose solution using pestle and mortar.

Enzyme Assay

In the livers of the rats, total protein (TP), ALP, AST, and ALT were measured.

Statistical Analysis

The results were analyzed using one-way ANOVA via SPSS software, and any significant result was recognized at $p \leq 0.05$.

Results

Table, 1, shows that the Mean±SE of the pH was significant ($p \leq 0.05$) different between groups. The lipids recorded significant ($p \leq 0.05$) increases especially in 12G, and proteins recorded significant ($p \leq 0.05$)

decreases in all groups. The viscosity, dry matter, and ash did not face significant (p>0.05) differences.

Table 1: Gross composition of camel colostrum

No.	Gross composition	Colostrum		
		0G	6G	12G
1	pH	6.56± 0.08 c	6.43± 0.13 b	6.30± 0.09 A
2	Viscosity (c P)	385± 3.01 a	341± 1.51 b	322± 0.99 B
3	Fats (gL^{-1})	1.65± 0.02 c	1.82± 0.11 b	1.93± 0.03 A
4	Proteins (gL^{-1})	152.51± 3.52 a	143.26± 4.31 b	132.17± 1.98 C
5	Dry matter (gL^{-1})	196.11± 3.56 a	195.32± 6.32 a	194.67± 5.40 A
6	Ash (gL^{-1})	9.82± 0.06 a	9.87± 0.12 a	9.75± 0.28 A

The results showed that the Mean±SE of the body weights of 12G were significantly ($p\leq$ 0.05) higher during 1st, 2nd, and 3rd week. The Mean±SE of the liver weights (LW) of 12G were significantly ($p\leq$ 0.05) higher. Table, 2, shows the gradual increases of the Mean± SE of the body and LW. Figure, 1, shows the differences in the weights of livers for all treatments.

Table 2: The mean± SE of the body and LW (g) of the experimental groups

Groups	1st week			2nd week			3rd week			4th week		
	Age (days)	Body weights (gm.)	LW (gm)	Ages (days)	Body weights (gm)	LW (gm)	Ages (days)	Body weights (gm.)	LW (gm)	Ages (days)	Body weights (gm.)	LW (gm)
0G	38± 0.23 b	274± 1.20 b	6.67± 0.05 c	45± 1.01 b	285± 1.44 b	6.87± 0.01 b	52± 0.13 b	392± 3.10 a	6.94± 0.09 a	59± 0.12 b	410± 2.91 a	7.36± 0.08 a
6G	36± 0.41 b	259± 0.92 b	6.63± 0.14 c	43± 0.99 b	264± 2.21 b	6.78± 0.91 b	50± 0.90 b	287± 2.09 a	6.84± 0.11 a	57± 0.14 b	297± 3.67 b	6.97± 0.17 b
12G	42± 0.31 a	293± 1.55 a	7.65± 0.92 a	49± 1.52 a	299± 1.56 a	7.72± 0.09 a	56± 0.82 a	336± 1.98 b	7.78± 0.25 b	63± 0.21	375± 1.98 b	7.84± 0.54 a
CG	37± 0.11 b	262± 1.22 b	6.50± 1.50 b	44± 1.22 b	267± 2.51 b	6.57± 0.87 b	51± 0.79 b	272± 3.92 c	6.62± 0.67 c	58± 0.23 b	284± 3.90 b	6.68± 0.98 b

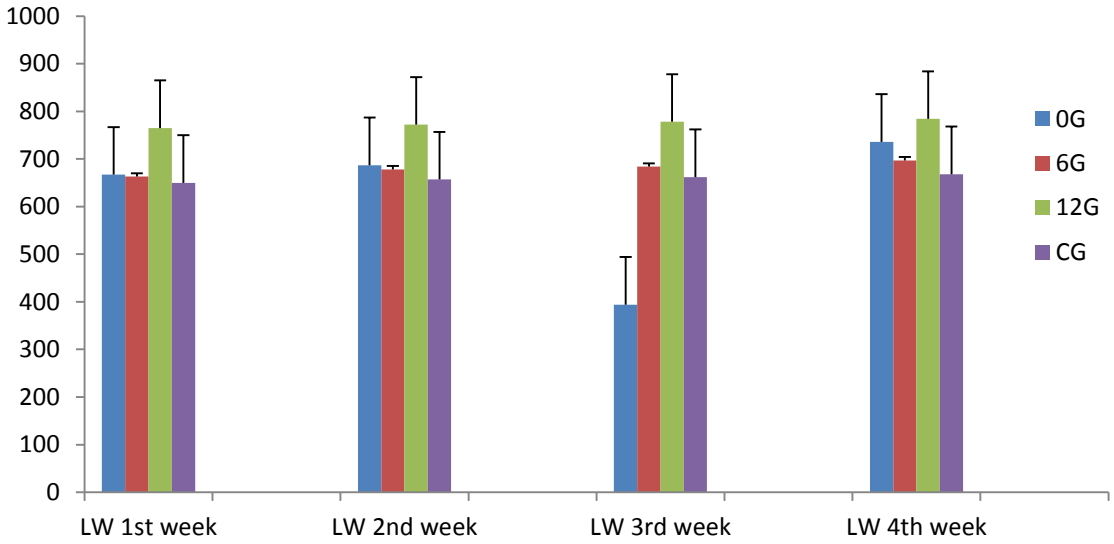

Figure 1: shows the correlation between LW and different colostrum therapies at all time points

Table, 3, shows that all enzymes except, TP, recorded significant ($p \leq 0.05$) increases in 0G. The increases were apparent at 30 days in ALT and AST. However, ALP recorded a decrease at all time-points of the experiment.

Table 3: the mean±SE of the liver enzymes of the 0 G

No	Enzyme values	(10) days	(20) days	(30) days
1	ALT(U/L)	45± 0.51 c	56± 0.32 b	76± 0.25 a
2	AST(U/L)	205±0.6.01 c	225± 5.11 b	232± 4.99 a
3	TP(g/ d L)	5.43± 0.02 a	5.37± 0.03 a	5.31± 0.06 a
4	ALP(U/L)	210± 3.41 a	197± 5.20 b	183± 6.22 c

Table, 4, reveals that all enzymes recorded significant ($p \leq 0.05$) decreases in 6G. ALT and AST were significantly decreased at 30 days. TP and ALP were decreased at 20 and 30 days.

Table 4: the mean ± SE of the liver enzymes of the 6 G

No	Enzyme values	(10) days	(20) days	(30) days
1	ALT(U/L)	88± 0.51 a	85± 0.02 a	79± 0.03 b
2	AST(U/L)	259± 5.11 a	247± 6.30 b	220± 3.44 c
3	TP(g/ d L)	5.23± 0.31 a	5.18± 0.21 b	5.14± 0.09 b
4	ALP(U/L)	172± 3.31 a	167± 4.20 b	161± 5.33 b

Table, 5, declares that ALT and AST showed significant ($p \leq 0.05$) increases at 20 and 30 days. TP and ALP recorded significant ($p \leq 0.05$) decreases at the same time-points mentioned above.

Table 5: the mean± SE of the liver enzymes of the 12 G

No	Enzyme values	(10) days	(20) days	(30) days
1	ALT(U/L)	143± 3.22 c	168± 5.12 b	189± 0.99 a
2	AST(U/L)	280± 7.11 c	312± 3.14 b	346± 4.41 a
3	TP(g/ d L)	5.59± 0.01 a	5.18± 0.02 b	5.10± 0.06 b
4	ALP(U/L)	148± 3.21 a	112± 2.14 b	103± 0.3.39 c

Table, 6, reveals that ALT and AST were increased significantly ($p \leq 0.05$) at 20 and 30 days. TP and ALP didn't show significant differences.

Table 6: the mean± SE of the liver enzymes of the CG

No	Enzyme values	(10) days	(20) days	(30) days
1	ALT(U/L)	264± 6.11 c	278± 5.20 b	294± 3.44 a
2	AST(U/L)	295± 2.22 c	368± 4.11 b	395± 5.64 a
3	TP(g/ d L)	5.10± 0.02 a	5.08± 0.12 a	5.09± 0.03 a
4	ALP(U/L)	148± 5.41 a	146± 2.99 a	145± 1.29 a

Histopathological Changes

Interestingly, changes have been occurred in the livers of the tested rats. These changes are presented in Figures (1-16), and they reveal the following:

- Week1-CG: Presence of normal-radially arrangement of hepatocytes surrounding the central vein is shown. There is congestion of the central vein and infiltration of inflammatory cells mainly macrophages and lymphocytes. There are vacuolation and hydropic degeneration of hepatocytes. Hyperplasia and congestion of bile duct are induced. There is dilation of sinusoids.
- Week1-0G: It reveals the presence of normal-radially arrangement of hepatocytes. There are mild congestion of central vein with dilation of sinusoids and normal bile duct with infiltration of inflammatory cells.
- Week1-6G: There are normal hepatic architecture with mild dilation of sinusoids and proliferation of kupffer cells with normal central vein. Normal bile ducts and mild infiltration of inflammatory cells are shown. The hepatocytes are normal; although, some cells show bi-nucleation.
- Week1-12G:
- Week2-CG: Extreme congestion, infiltration of mainly macrophages, and fatty change with hepatocytes shown as signet-like shape (peripheral nuclei) are presented.
- Week2-0G: There are vacuolation of hepatocytes with dilation of sinusoids, mild-congested bile-duct, loss of hepatic architecture around the congested-central vein, and mild-fatty degeneration which occur within hepatocyte.
- Week2-6G: There are sever hyperplasia of bile duct with infiltration of inflammatory cells, sever dilation of sinusoids, and proliferation of kupffer cells.
- Week2-12G: There are sever peripheral-nuclei-based fatty degeneration of hepatocytes that look like signet like shapes, dilation of sinusoids with proliferation of kupffer cells, and hyperplasia of bile duct.
- Week3-CG: There are hyperplasia of the congested-bile, fatty degeneration with dilation of sinusoids, and some binucleus-hepatocytes.
- Week3-0G: Normal-radially arrangement of hepatocytes that surround the normal-central vein is revealed. Mild-fatty degeneration and proliferation of kupffer cells, and mildly-congested bile-duct are present.
- Week3-6G: There are congestion of blood vessels, sever dilation of sinusoids, proliferation of kupffer cells, vacuolation with hydrobic degeneration within hepatocytes, hyperplasia of bile duct, and sever hemorrhage between hepatocytes with sever fatty degeneration.
- Week3-12G: There is mild hyperplasia of the congested-bile duct. There is mild dilation of sinusoids. Radially arrangement of hepatocytes around the normal-central vein is found. There is mild proliferation of kupffer cells. There are normal bile duct and infiltration of inflammatory cells.
- Week4-CG: There are mild infiltration of inflammatory cells and mild hemorrhage between hepatocytes. Hepatocytes are radially arranged around the normal-central vein. There are mild proliferation of kupffer cells and normal-bile duct.

- Week4-0G: There are infiltration of inflammatory cells and congestion of bile duct. Hepatocytes are radially arranged around the normal-central vein with mild dilation of sinusoids. Some proliferations of kupffer cells are present.
- Week4-6G: There is mild-fatty degeneration of radially-arranged hepatocytes around the normal-central vein with high proliferation of kupffer cells. Inflammatory cells are scattered between hepatocytes.
- Week4-12G: Extreme congestion of bile duct and mild hyperplasia are found. There are some changes of mild dilation of sinusoids, radially-arranged of cells around the normal-central vein, and binucleated hepatocyte with some kuffer cells present in the sinusoids.

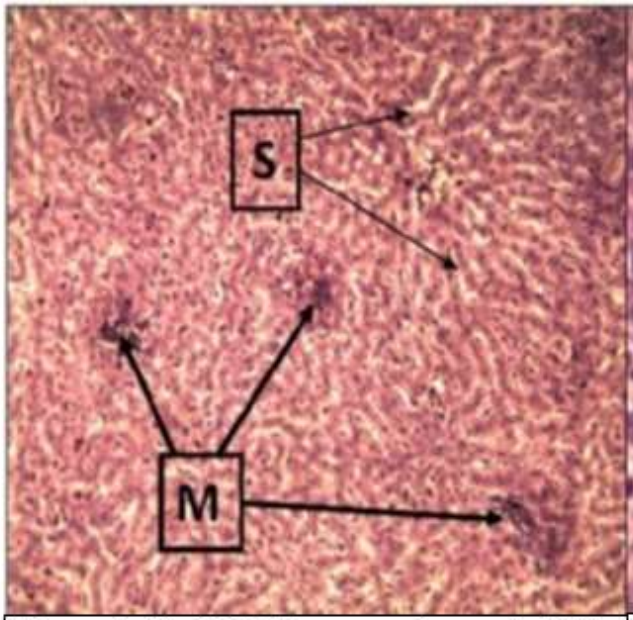

Figure 1: Week1-CG: Presence of normal-radially arrangement of hepatocytes. surrounding the central vein is shown

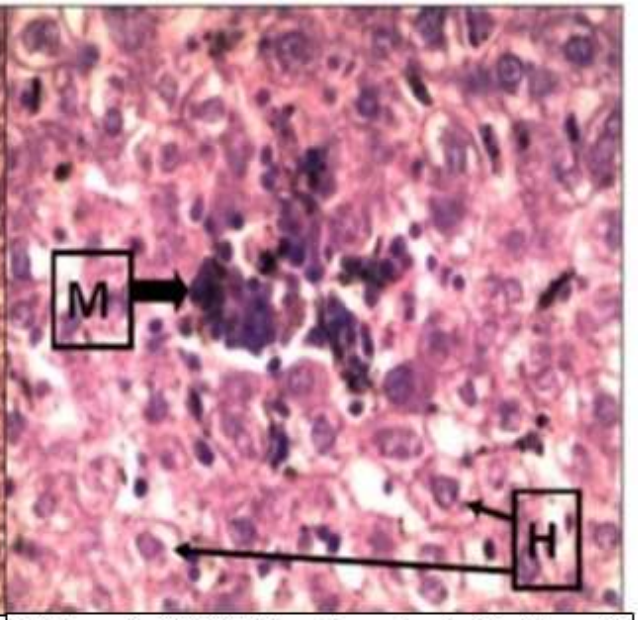

Figure 2: Week1-CG. There is infiltration of inflammatory cells mainly macrophages (M), also vacuolation and hydropic degeneration of hepatocytes (H), with dilation of sinusoids. 40X H&E

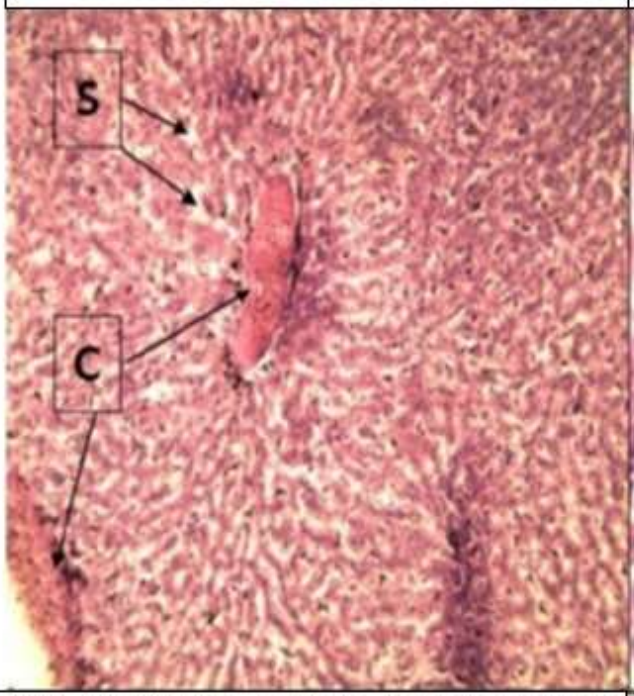

Figure 3: Week1-CG. Presence of normal-radially arrangement of hepatocytes around the central vein. There are congestion of central vein (C) and dilation of sinusoids (S). 10X H&E

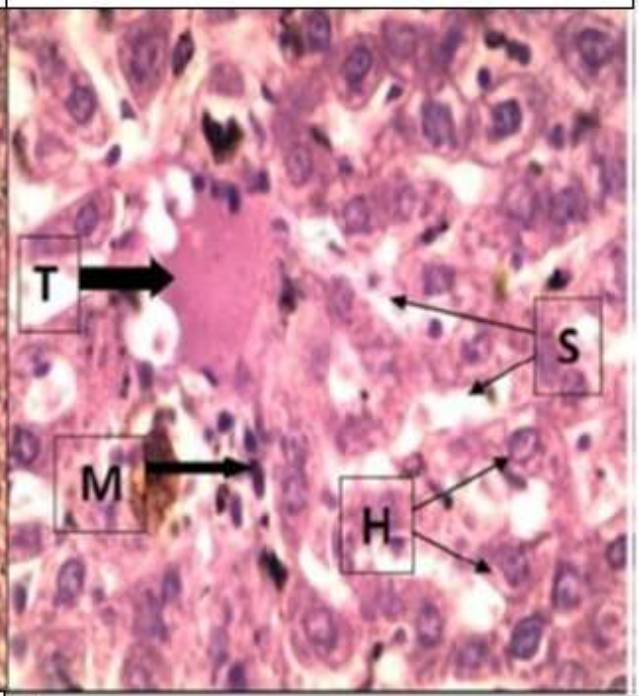

Figure 4: Week1-CG. Presence of large thrombus within the central vein (T), mild infiltration of inflammatory cells mainly macrophage (M), hydropic degeneration of hepatocytes (H), and dilation of sinusoids (S). 40X H&E

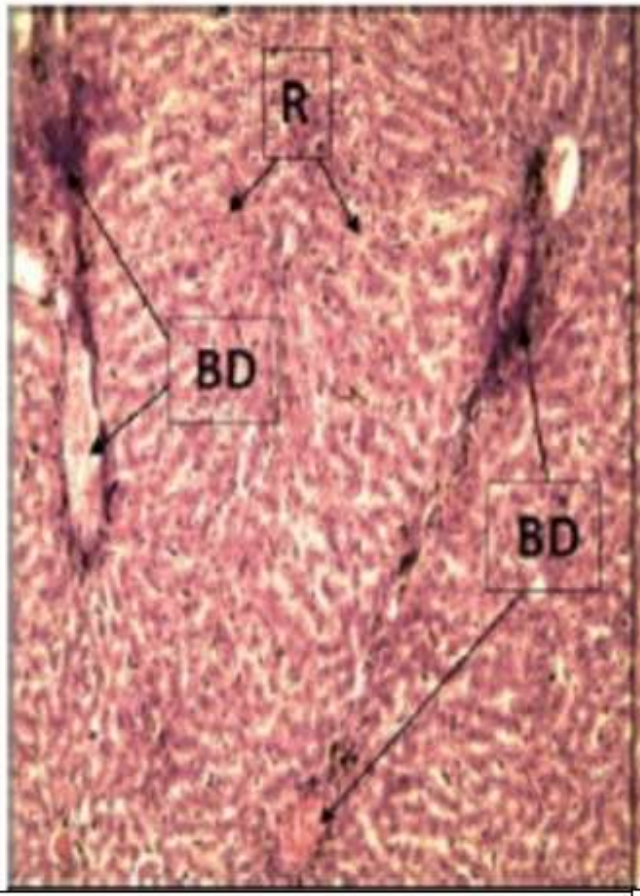

Figure 5: Week1-CG. Presence of normal radially arrangement of hepatocytes around the central vein (R) and hyperplasia and congestion of bile duct (BD). 10X H&E

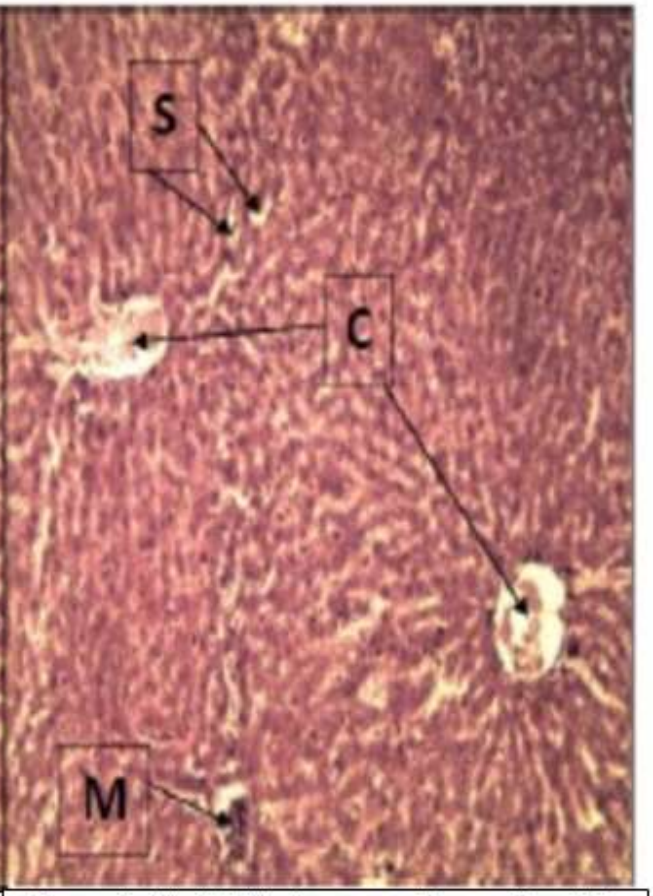

Figure 6: Week1-CG. Presence of normal radially arrangement of hepatocytes, mild congestion of central vein (C) with dilation of sinusoids (S) and infiltration of inflammatory cells (M). 10X H&E

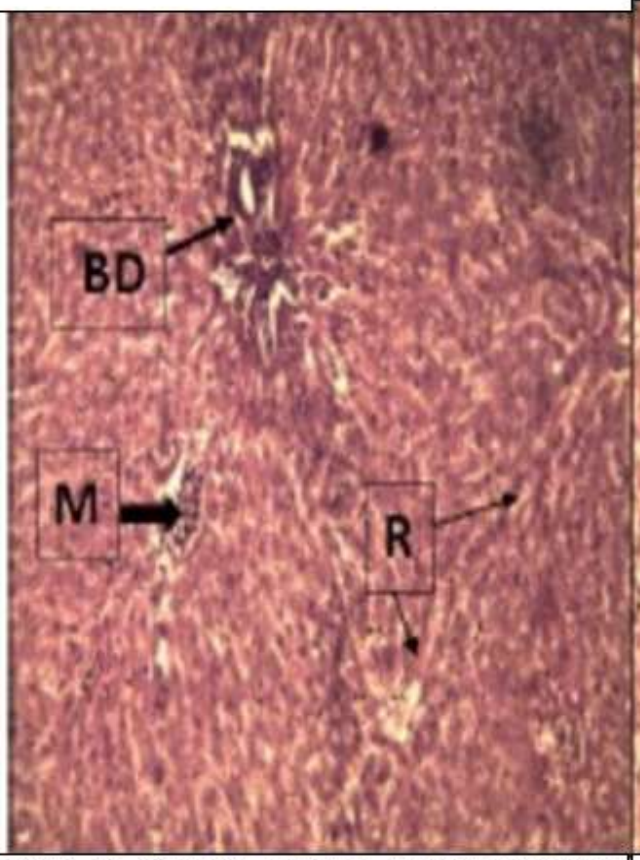

Figure 7: Week1-0G. Presence of normal-radially arrangement of hepatocytes (R) with normal bile duct (BD) and infiltration of inflammatory cells (M). 10X H&E

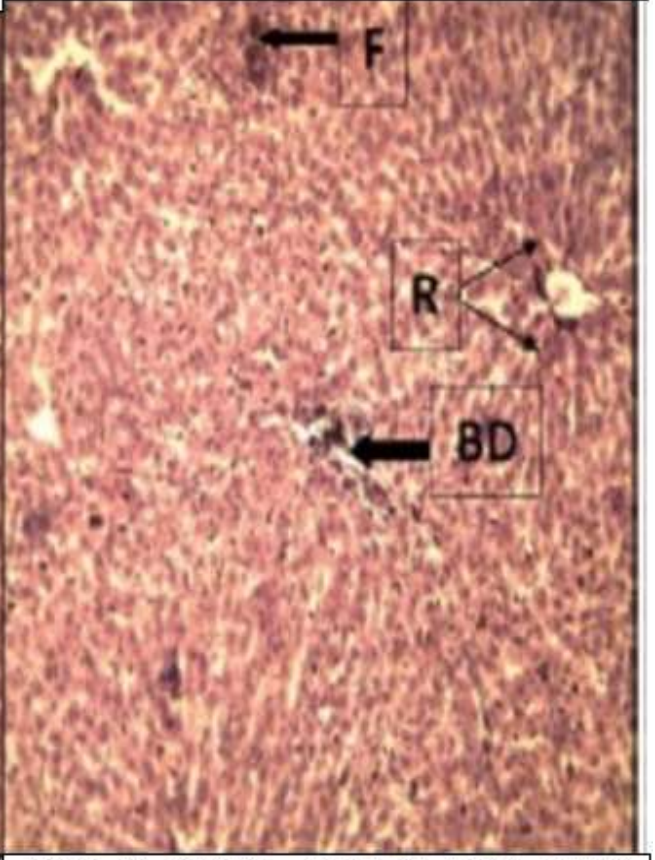

Figure 8: Week1-6G. There is normal hepatic architecture (R) with normal central vein. Normal bile ducts (BD) and mild infiltration of inflammatory cells (M). 10X H&E

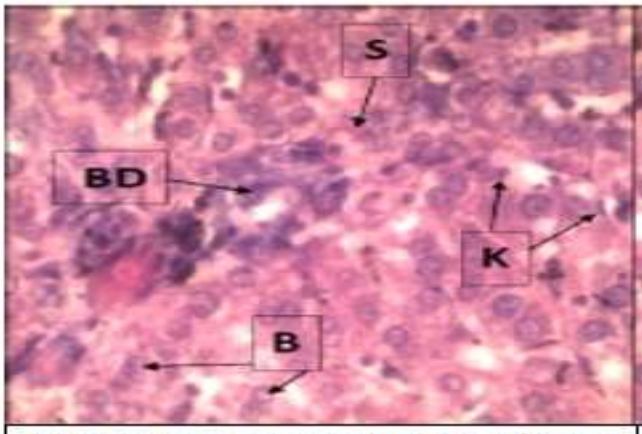

Figure 9: Week1-6G. There are mild dilation of sinusoids (S) and proliferation of Kupffer (K). Normal bile ducts (BD) and the hepatocytes show normal but some show binucleation (B). 40X H&E

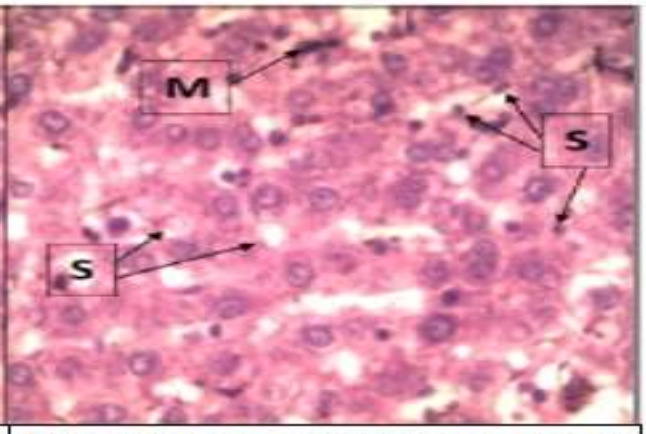

Figure 10: Week1-6G. There are mild dilation, some congestions of sinusoids (S), proliferation of Kupffer cells (K), and scattered inflammatory cells (M). 40X H&E

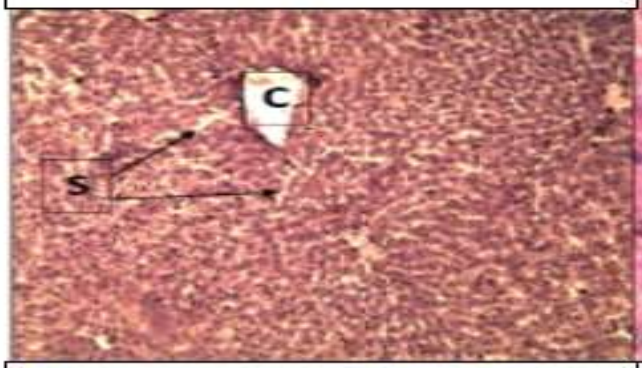

Figure 11: Week1-12G. There is normal hepatic architecture around normal-central vein (C). There is dilation of sinusoids (s). 10X H&E.

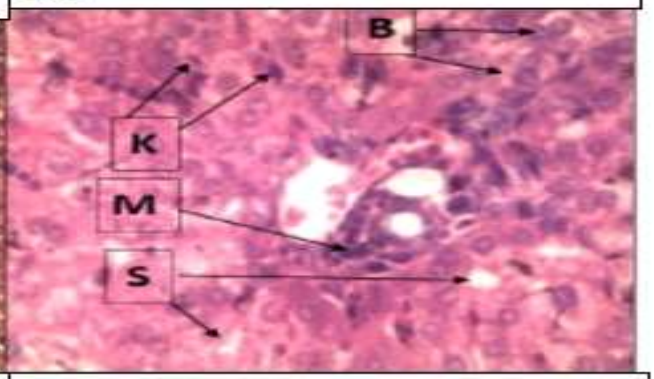

Figure 12: Week1-12G. There is mild dilation of sinusoids (S), proliferation of Kupffer cells (K), normal bile ducts, scattered inflammatory cells (M), normal hepatocytes with some binucleated ones (B). 40X H&E

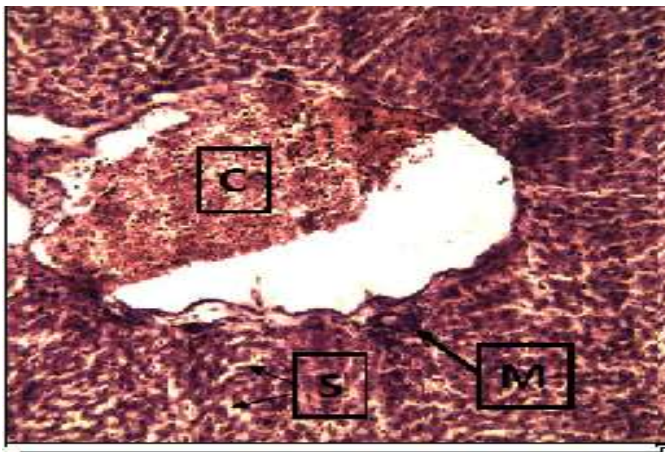

Figure 13: Week2-CG. there is severe congestion in hepatic tissue (C), infiltration of inflammatory cells (M) with dilation of sinusoids (S). 10X H&E

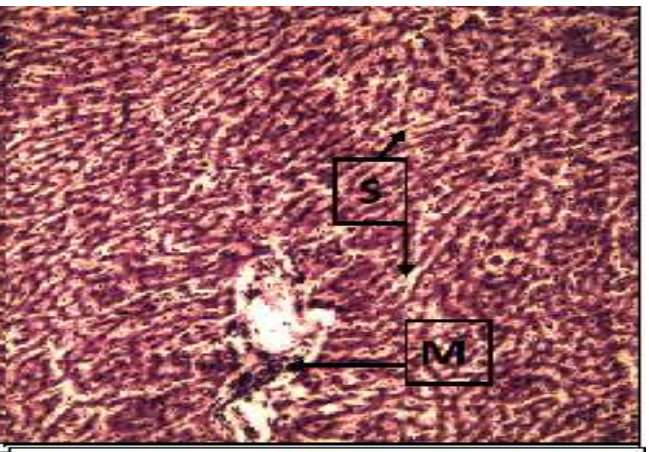

Figure 14: Week2-CG. loss of hepatic architecture, dilation of sinusoids (S) with infiltration of inflammatory cells (M). 10X H&E

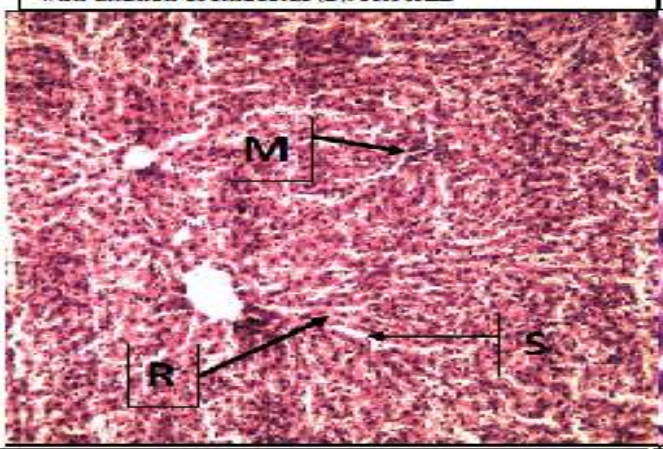

Figure 15: Week2-CG. loss of radially arrangement of hepatocytes around central vein (R). Also there is infiltration of inflammatory cells (M) with marked dilation of sinusoids (S). 10X H&E

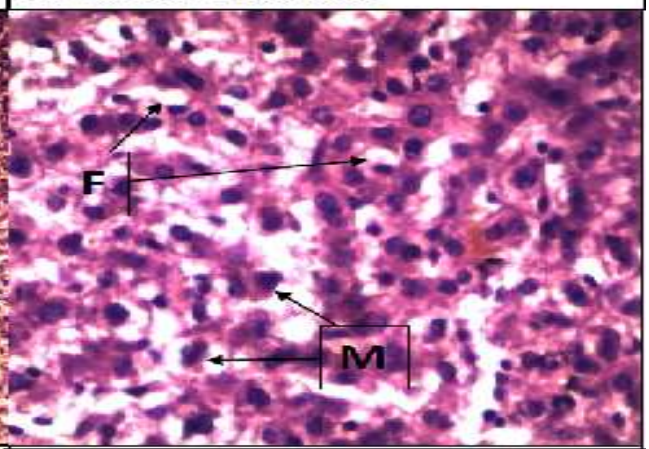

Figure 16: Week2-CG. There is infiltration of inflammatory cells mainly macrophages (M), presence of fatty infiltration (fatty change) in the hepatocytes in which hepatocytes showed as signet-like shape (peripheral nuclei) (F). 40X H&E

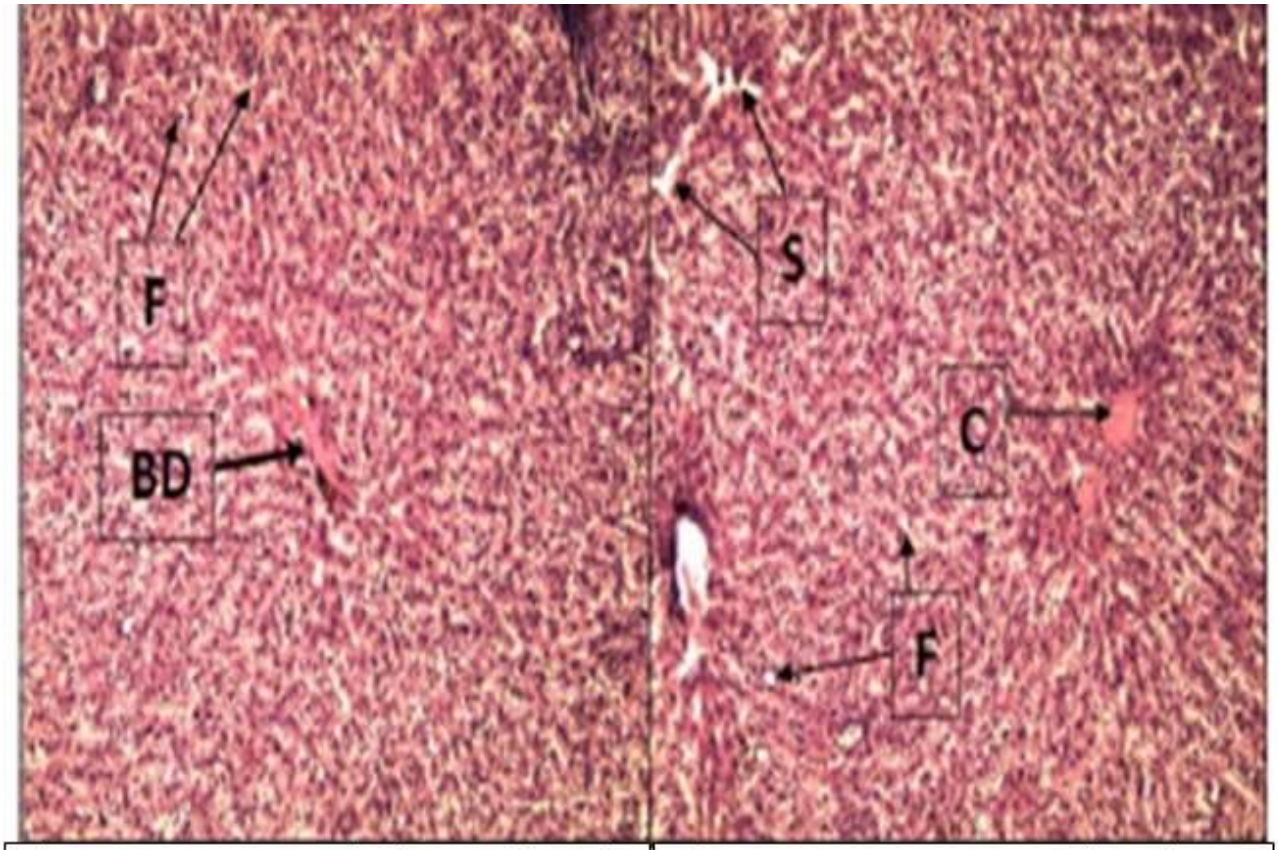

Figure 17: Week2-0G. There is mild congestion of bile duct (BD), loss of hepatic architecture and mild fatty degeneration which occur within hepatocytes (F). 10X H&E

Figure 18: Week2-0G.< Loss of hepatic architecture around congested central vein (C) with dilation of sinusoids (S). Also mild fatty degeneration which occurs within hepatocyte. 10X H&E.

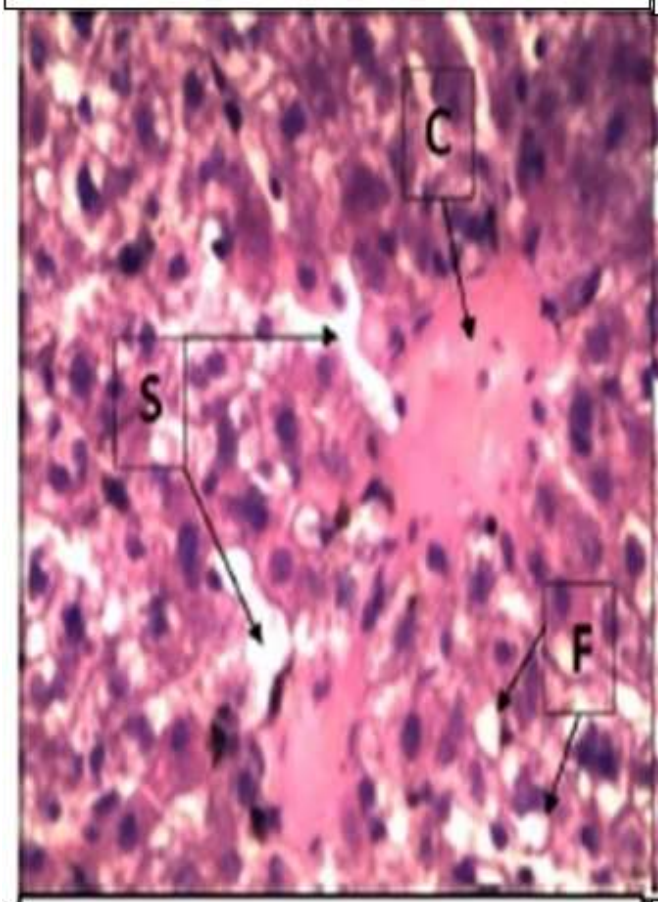

Figure 19: Week2-0G. There is dilation of sinusoids, congestion of central vein, mild fatty degeneration which occurs within hepatocytes. 40X H&E

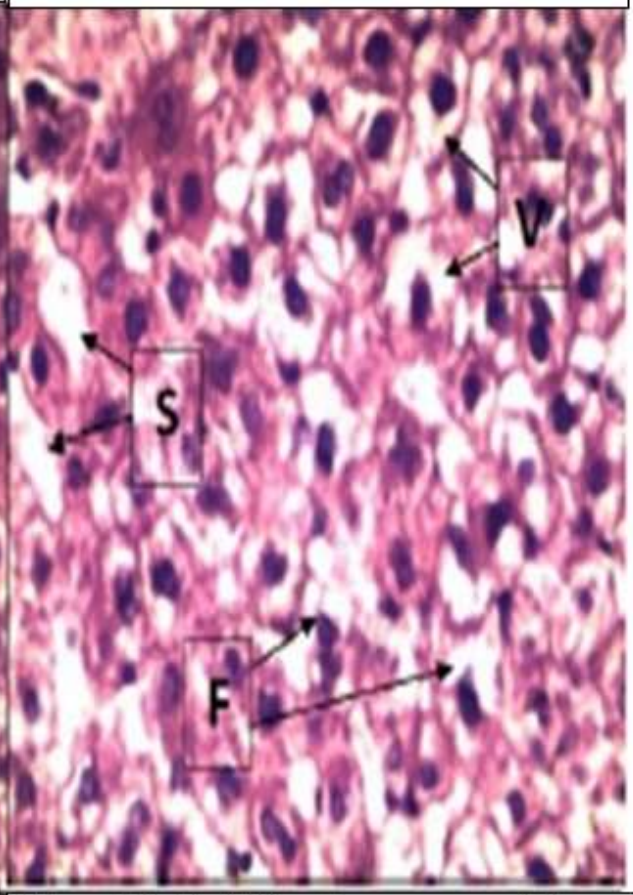

Figure 20: Week2-0G. There are vacuolation of hepatocytes with dilation of sinusoids with fatty degeneration which occur within hepatocyte. 40X H&E

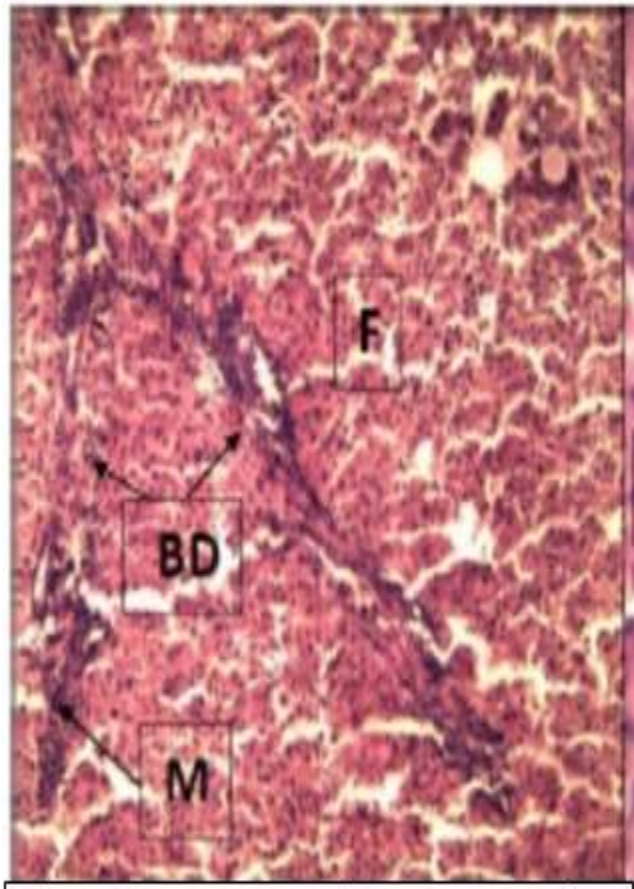

Figure 21: Week2-6G. There is severe hyperplasia of bile duct (BD) with infiltration of inflammatory cells (M), severe dilation of sinusoids. 10X H&E

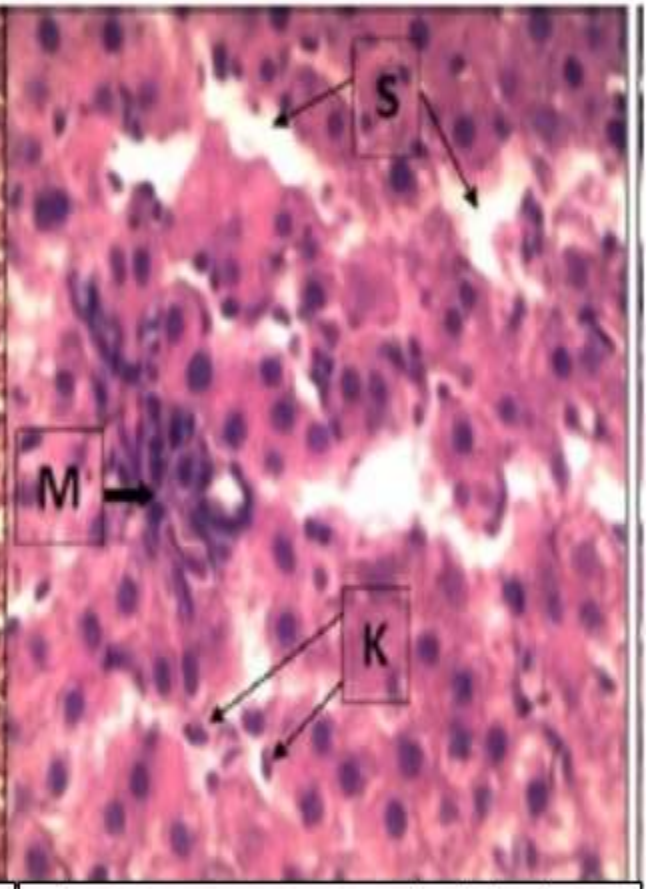

Figure 22: Week2-6G. There is infiltration of inflammatory cells mainly macrophages (M), severe dilation of sinusoids (S) and proliferation of Kupffer

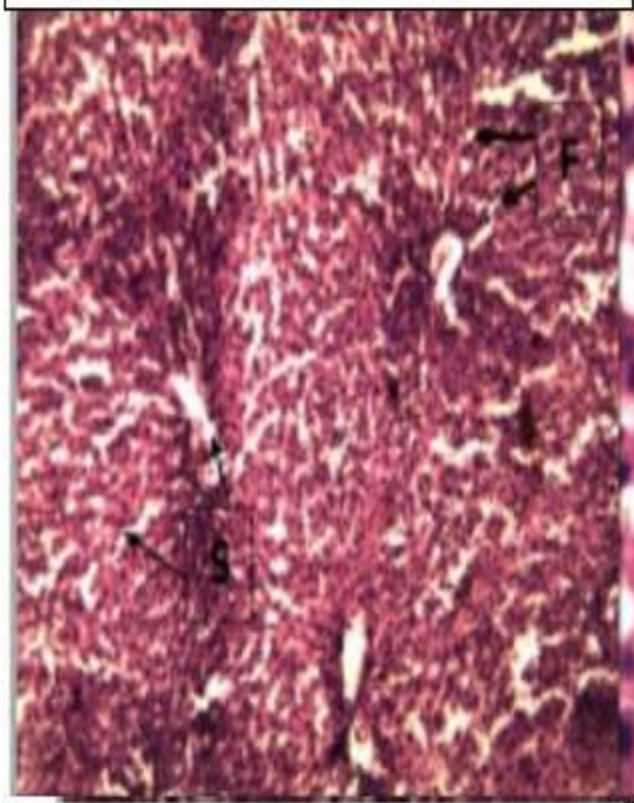

Figure 23: Week2-12G. there is loss of hepatic architecture with marked dilation of sinusoids, severe fatty degeneration (F) with dilation of sinusoids (S), with proliferation of Kupffer cells. 10X H&E.

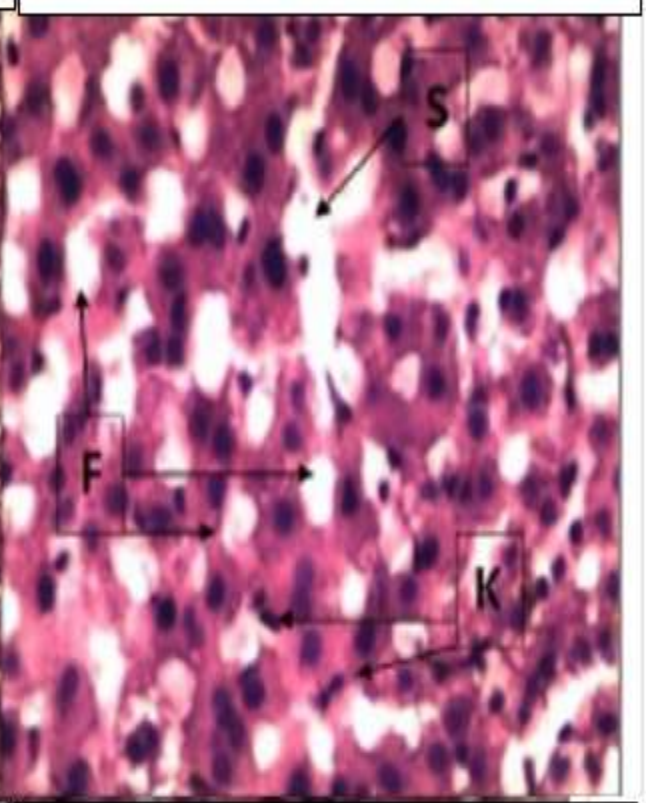

Figure 24 Week2-12G. Severe fatty degeneration which characterized by presence of peripheral nuclei of hepatocytes (hepatocytes showed as signet like shape) (F), dilation of sinusoids (S), with proliferation of Kupffer cells (K). 40X H&E

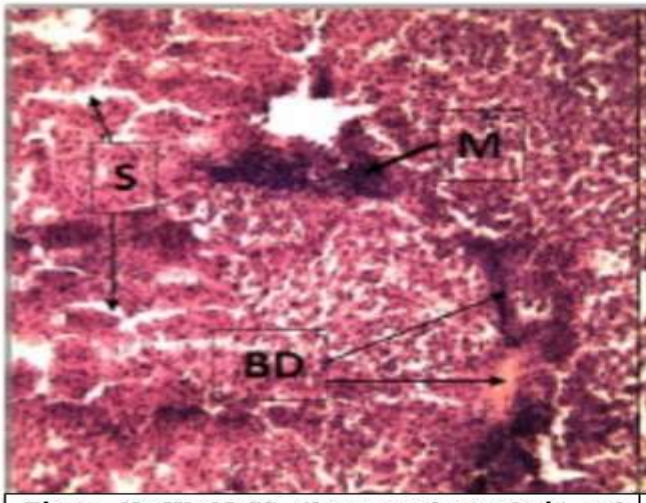

Figure 25: Week3-CG. there are hyperplasia and congestion of bile duct (BD) with infiltration of inflammatory cells (M) and dilation of sinusoids (S). 10X H&E

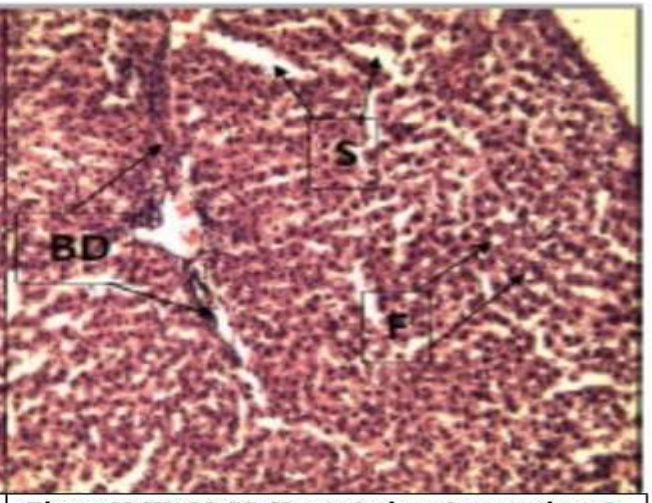

Figure 26: Week3-CG. Hyperplasia and congestion of bile duct (BD) and fatty degeneration (F) with dilation of sinusoids (S). 10X H&E

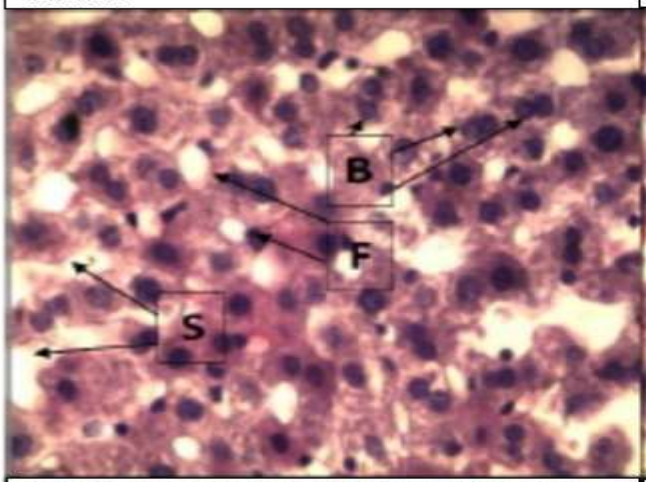

Figure 27: Week3-CG. There is fatty degeneration with dilation of sinusoids, some hepatocytes with binucleated. 40X H&E

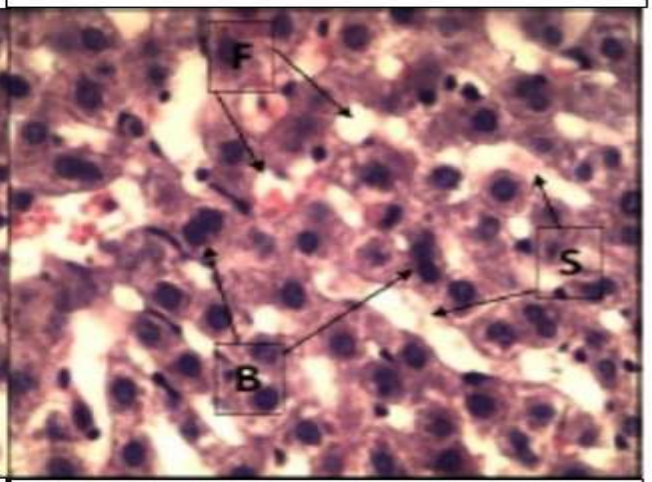

Figure 28: Week3-CG. Dilation and congestion of sinusoids. Also there is fatty degeneration of hepatocytes, some hepatocytes showed binucleated. 40X H&E

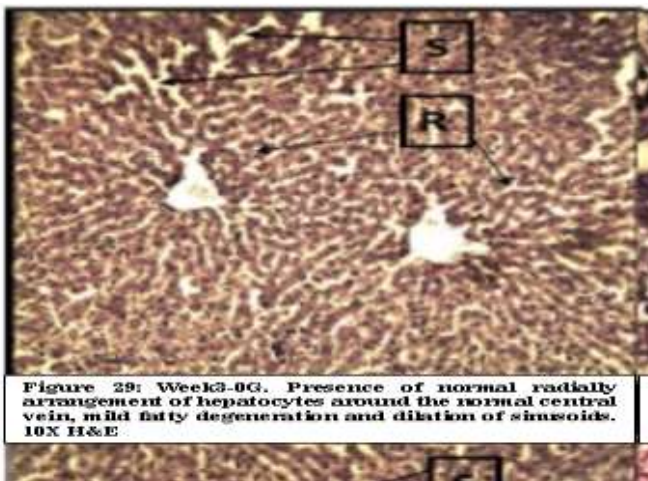

Figure 29: Week3-0G. Presence of normal radially arrangement of hepatocytes around the normal central vein, mild fatty degeneration and dilation of sinusoids. 10X H&E

Figure 30: Week3-0G. Mild fatty degeneration (the hepatocytes showed as signet-like shape) and with proliferation of Kupffer cells. 40X H&E

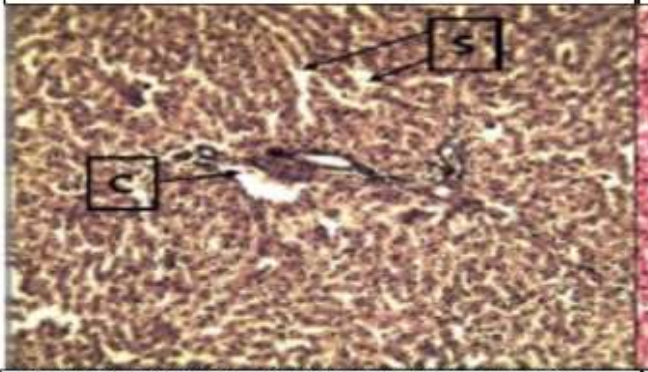

Figure 31: Week3-0G. the bile duct showed normal but slightly congested with marked dilation of sinusoids. 10X H&E.

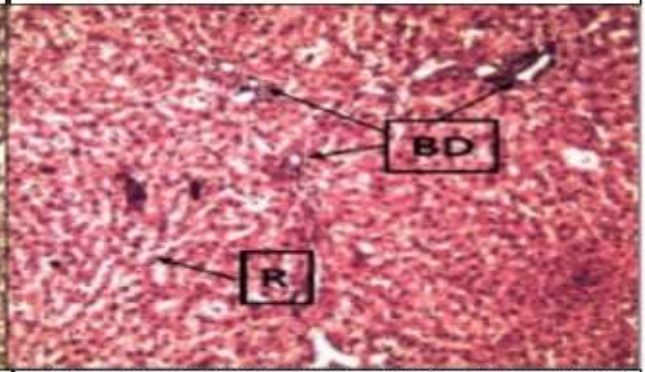

Figure 32: Week3-0G. Presence of normal radially arrangement of hepatocytes around the normal central vein and normal bile ducts. 10X H&E

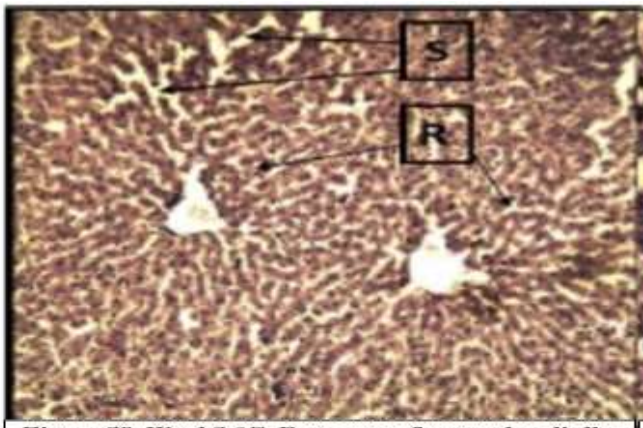

Figure 29: Week3-0G. Presence of normal radially arrangement of hepatocytes around the normal central vein, mild fatty degeneration and dilation of sinusoids. 10X H&E

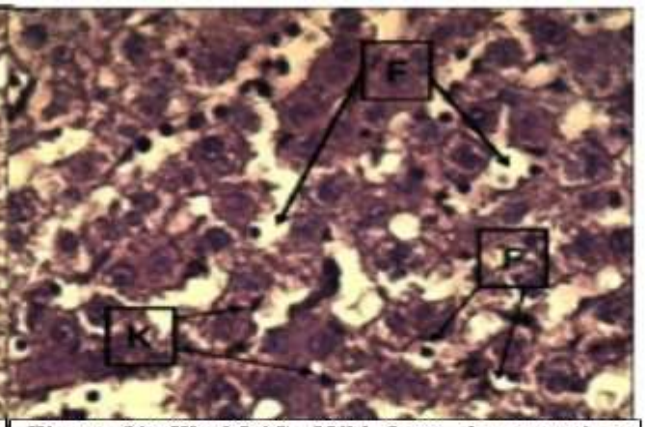

Figure 30: Week3-0G. Mild fatty degeneration (the hepatocytes showed as signet-like shape) and with proliferation of Kupffer cells. 40X H&E

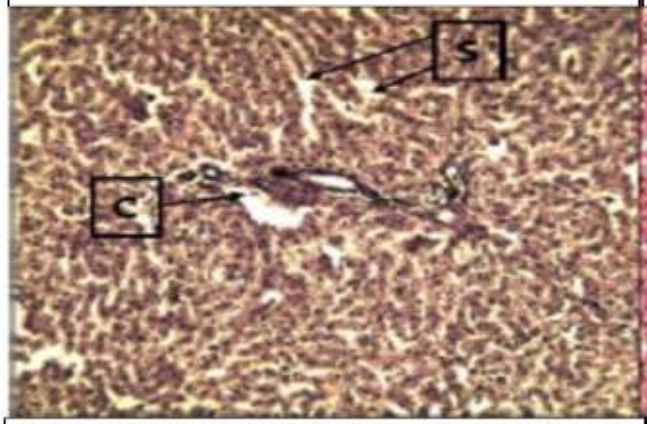

Figure 31: Week3-0G. the bile duct showed normal but slightly congested with marked dilation of sinusoids. 10X H&E.

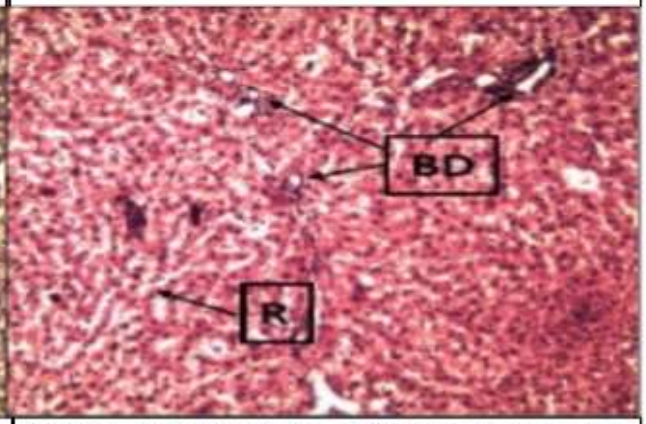

Figure 32: Week3-0G. Presence of normal radially arrangement of hepatocytes around the normal central vein and normal bile ducts. 10X H&E

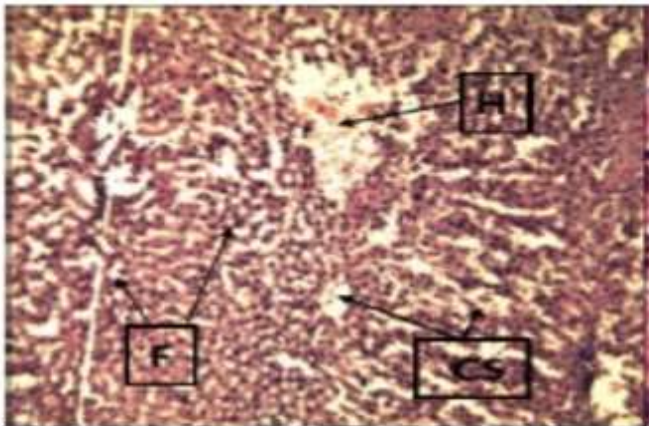

Figure 33: Week3-12G. Severe hemorrhage in the hepatic tissue (H), severe dilation of sinusoids (S), congestion of sinusoids (CS) with severe fatty degeneration (F). 10X H&E

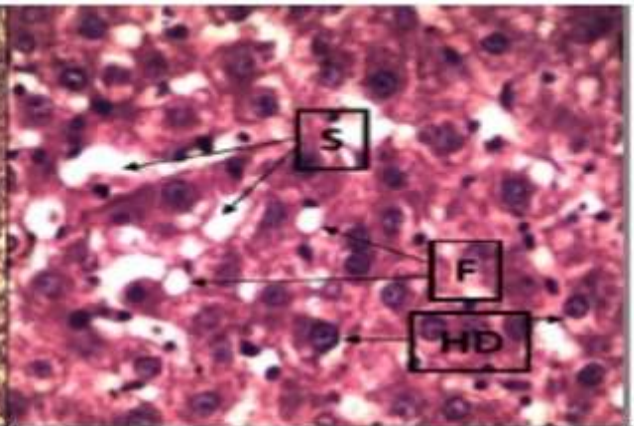

Figure 34: Week3-12G. Vacuolation and hydropic degeneration of hepatocytes (HD), severe dilation of sinusoids (S) with proliferation of Kupffer cells, and severe fatty degeneration (F). 40X H&E

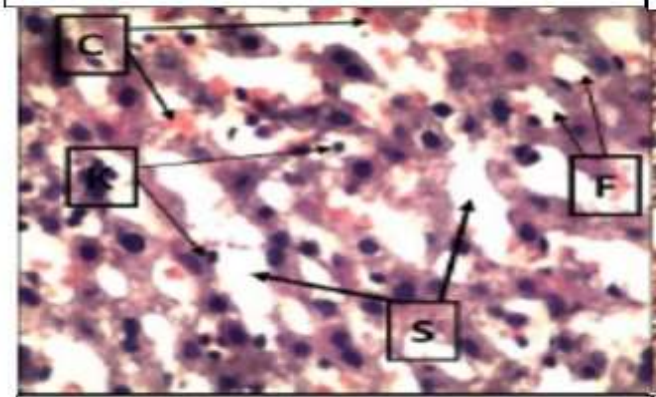

Figure 35: Week3-12G. Severe dilation of sinusoids (S), congestion of sinusoids (C) with proliferation of Kupffer cells (K), and severe fatty degeneration (F). 40X H&E

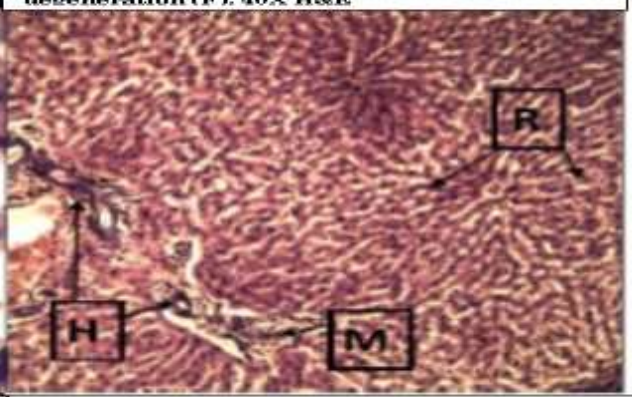

Figure 36: Week3-12G. There is mild hyperplasia and congestion of bile duct (H), with infiltration of inflammatory cells (M), mild dilation of sinusoids. Presence of radially arrangement of hepatocytes around the normal central vein (R). 10X H&E

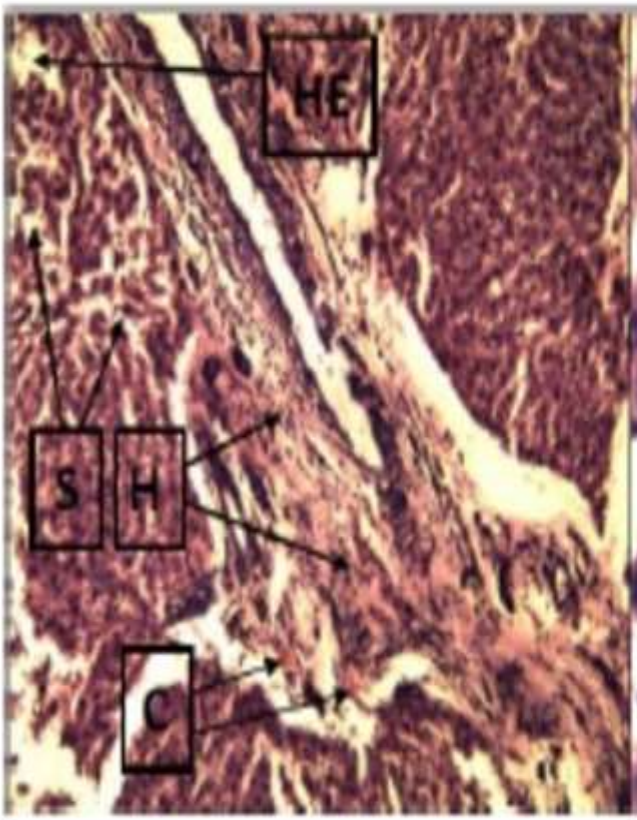

Figure 37: Week4-CG. There is marked hyperplasia (H) and congestion of bile duct (C), mild dilation of sinusoids (S) with hemorrhage in the hepatic tissue (HE). 10X

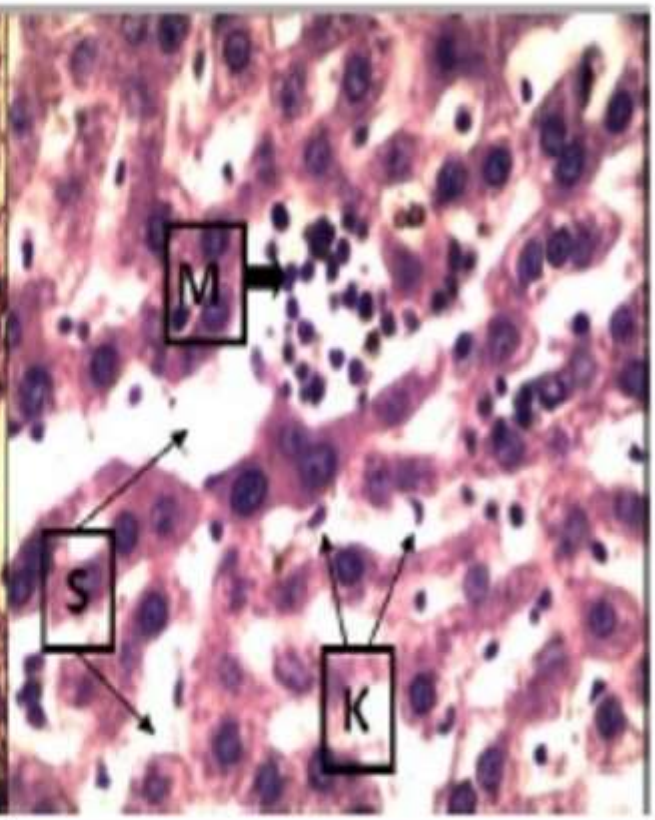

Figure 38: Week4-CG. Dilation of sinusoids (S), mild proliferation of Kupffer cells (K), with infiltration of inflammatory cells mainly macrophages (M). 40X H&E

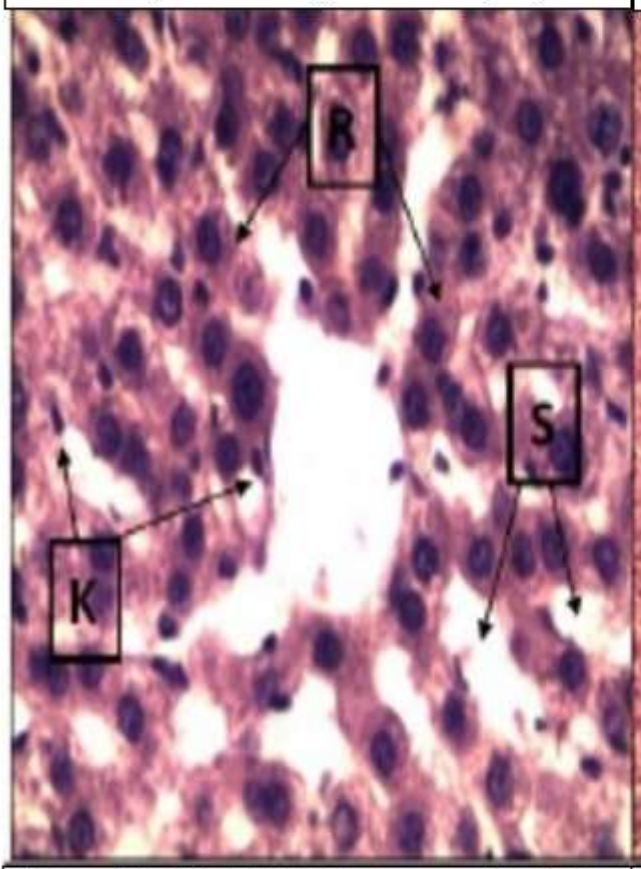

Figure 39: Week4-CG. Presence of radially arrangement of hepatocytes around normal central vein (R) and dilation of sinusoids (S) with mild proliferation of Kupffer cells (K). 40X H&E

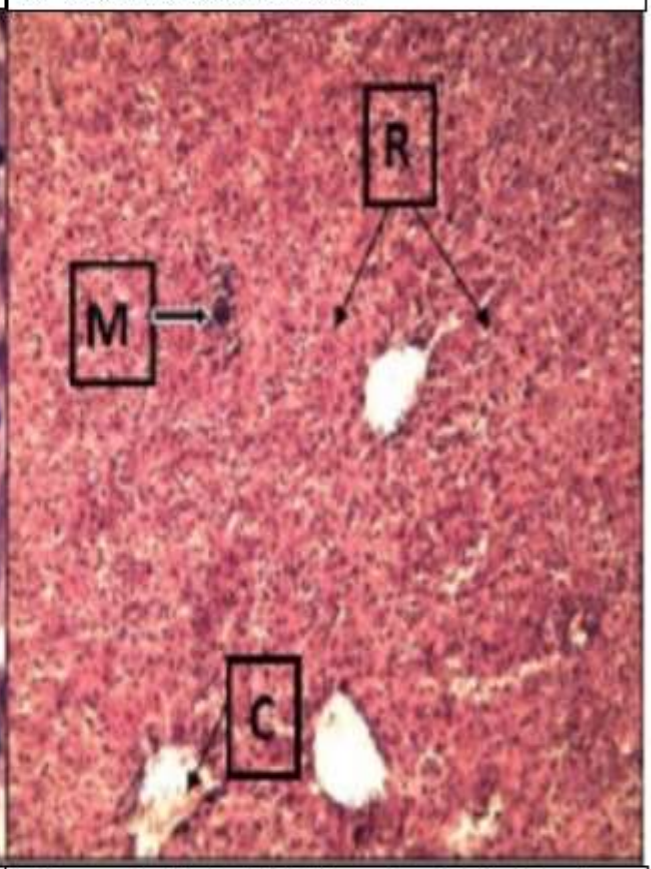

Figure 40: Week4-CG. There's mild infiltration of inflammatory cells (M) with mild congestion in the hepatic tissue (C). Presence of radially arrangement of hepatocytes around normal central vein (R). 10X H&E

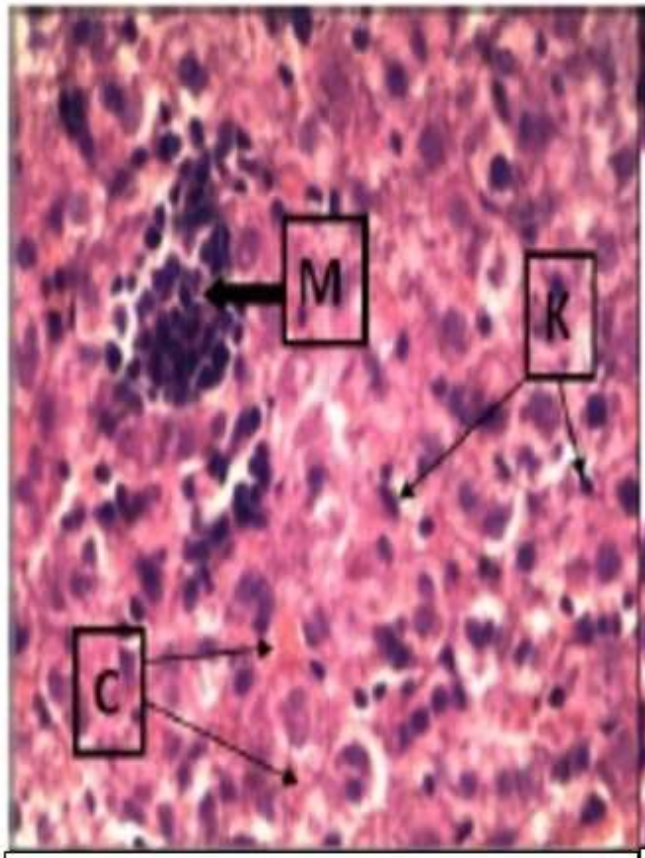

Figure 41: Week4-CG. There's mild infiltration of inflammatory cells mainly macrophages (M) with mild congestion of sinusoids (C), mild proliferation of Kupffer cells (K). 40X H&E

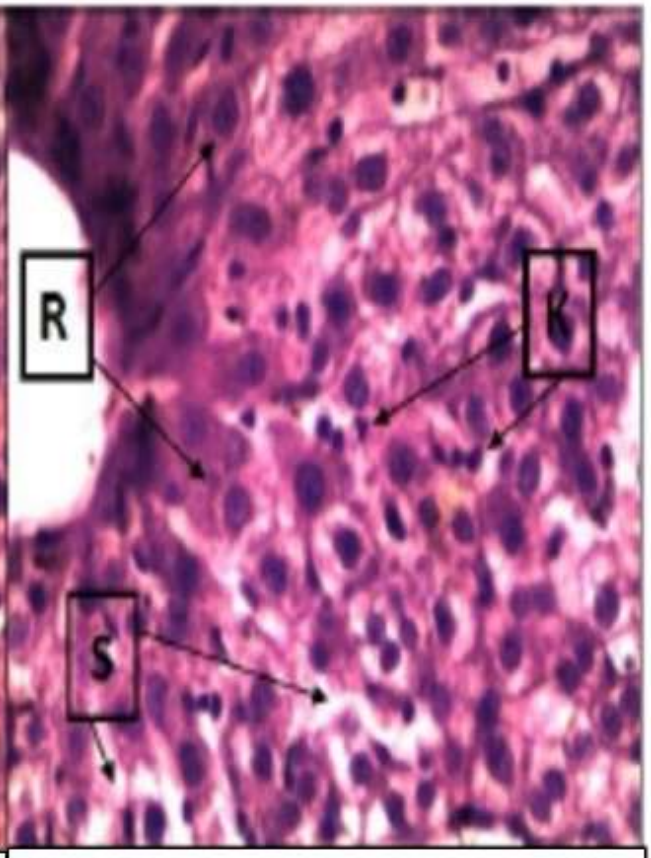

Figure 42: Week4-0G. there is radially arrangement of hepatocytes around the normal central vein with mild dilation of sinusoids. Also a few proliferation of Kupffer cells. 40X H&E

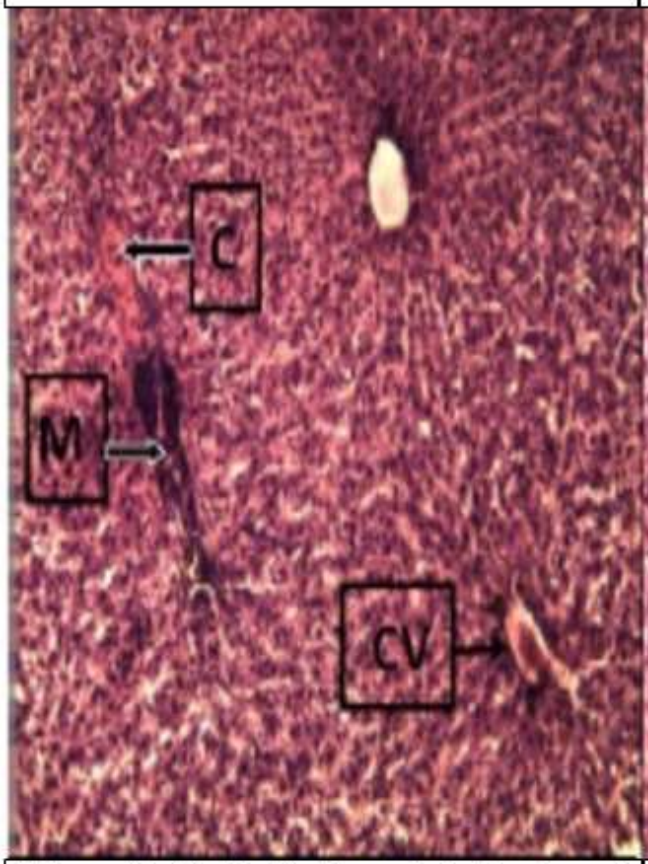

Figure 43: Week4-0G. There is infiltration of inflammatory cells (M) with congestion of bile duct (C). Also presence of radially arrangement of hepatocytes around central vein which showed slightly congested (CV), mild dilation of sinusoids. 10X H&E.

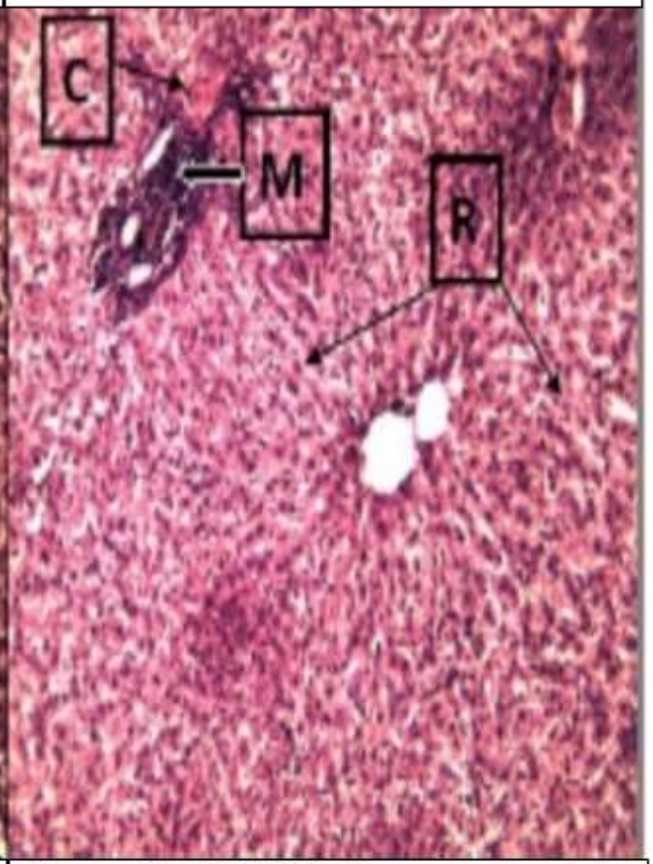

Figure 44: Week4-0G. Presence of radially arrangement of hepatocytes around the central vein (R). Also there is infiltration of inflammatory cells (M) with congestion of bile duct (C). 10X H&E

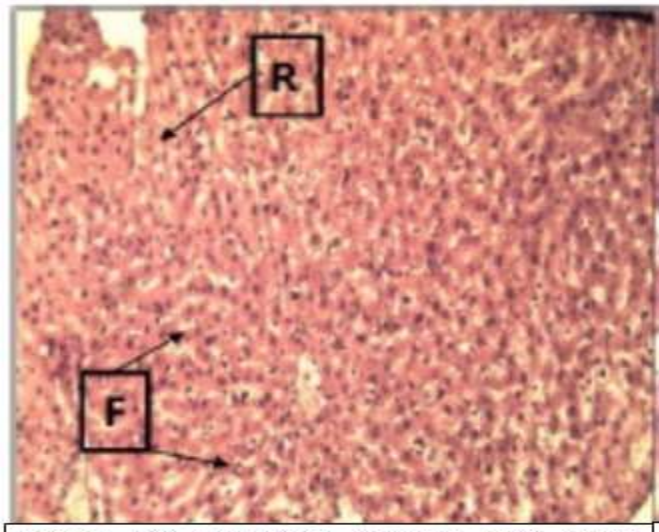

Figure 45: Week4-6G. There's mild fatty degeneration (fatty change) of hepatocytes (F), presence of radially arrangement of hepatocytes around the normal central vein (R). 10X H&E.

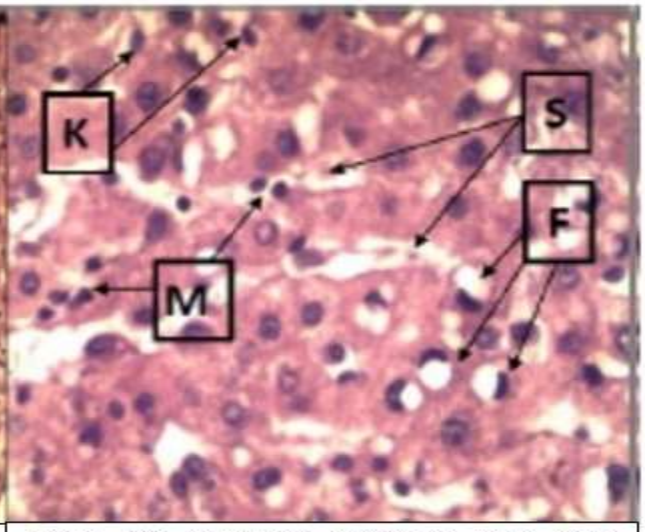

Figure 46: Week4-6G. There is mild fatty degeneration (fatty change) of hepatocytes (the hepatocytes showed as signet-like shape) (F), mild dilation of sinusoids (S) with high proliferation of Kupffer cells (K) and scattered of inflammatory cells in the hepatic tissue (M).

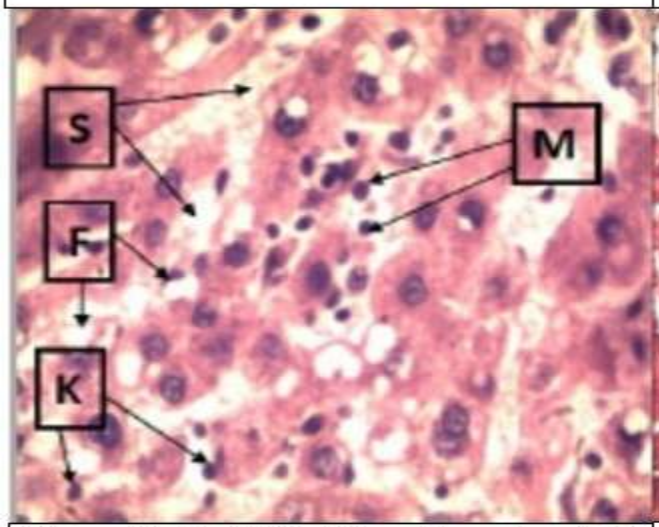

Figure 47: Week4-6G. There is scattered of inflammatory cells in the hepatic tissue (M) and presence of fatty change of hepatocytes (the hepatocytes showed as signet-like shape) (F), mild dilation of sinusoids (S) with high proliferation of Kupffer cells (K). 40X H&E

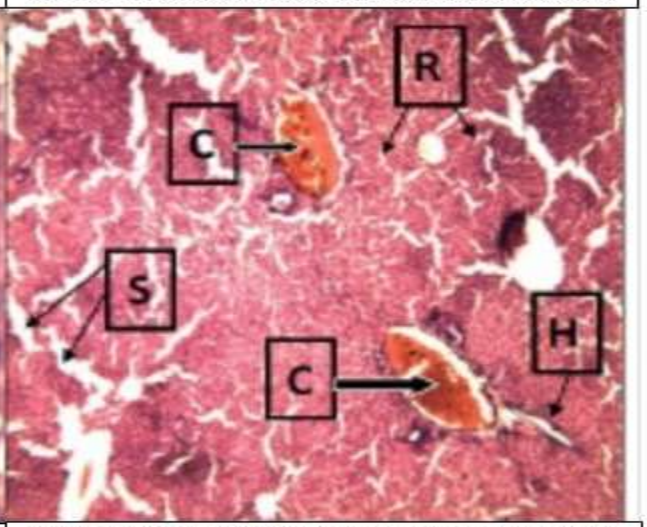

Figure 48: Week4-12G. Severe congestion of bile duct (C) with mild hyperplasia (H), dilation of sinusoids (S) and presence of radially arrangement around the normal central vein (R). 10X H&E.

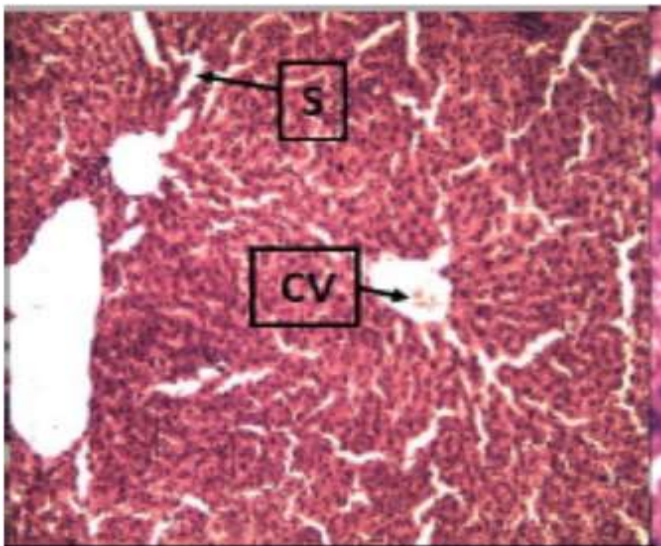

Figure 49: Week4-12G. Presence of radially arrangement around slightly congested central vein (CV), marked dilation of sinusoids (S). 10X H&E

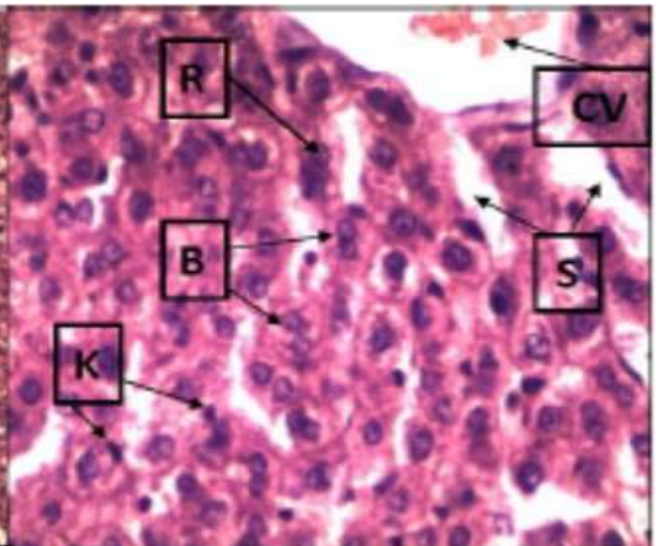

Figure 50: Week4-12G. Slightly congested central vein (CV) and radially arrangement of hepatocytes around it (R) and mild dilation of sinusoids (S). Presence of binucleated hepatocytes (B) with few Kupffer cells (K). 40X H&E

Discussion

The current study was conducted to evaluate the effects of camel colostrum extracts of different post-parturition time-points, 0, 6, and 12hr, on the liver biomarkers in rats. The camel colostrum is a nutrient-rich fluid that contained immune and bioactive growth factors. Table 1 shows the gross composition of the colostrum.

PH values were decreased gradually 6.56± 0.08, 6.43± 0.13, and 6.30± 0.09 respectively due to the high presence of proteins 152.51± 3.52, 143.26± 4.31, and 132.17± 1.98 g L^{-1} respectively especially immunoglobulins G (IgG1, IgG2, and IgG3) and camel serum albumin. These results agreed with [14] who detected the presence of high quantities of IgG1, 2, and 3 that decreased after 48h after parturition. Also the high contents of dry matters were decreased in the colostrum 196.11± 3.56, 195.32± 6.32, and 194.67± 5.40 respectively and gradually. We believe that this decrease was due to the decrease in the protein contents [22]. The results of the body and liver weights indicate increases in these weights, and this is could be due to colostrum richness with various nutrients such as fat and protein. The liver enzymes increases such as ALT and AST indicate responses to camel colostrum [23] who found using camel milk affected the responses of these enzymes in human patients.

The histopathological changes revealed variable responses in all treated groups, and this indicates that camel colostrum induces such slight changes for the body tissues to prepare themselves for future intruders such as pathogens [24]. These results indicate important effects of camel colostrum to prepare and protect body against various environmental intruders such as pathogens. Our results generate important information for future work to thoroughly study colostrum extracts on infections in humans and animals.

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