

Original Research

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Histological Evaluation of L-Carnitine's Ability to Restore Liver Architecture Following Ceftriaxone Toxicity

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ABSTRACT:

Background: Ceftriaxone, a commonly administered third-generation cephalosporin antibiotic, is generally regarded as safe but has been linked to few cases of hepatotoxicity. L-carnitine, a naturally occurring molecule, demonstrates significant antioxidant properties and indicates a potential hepatoprotective function.

Objective: This study aimed at establishing the hepatotoxicity of ceftriaxone in a rat model and the hepatoprotective effects of L-carnitine to counter the effects of the drug in causing hepatic damage. **Methods:** Forty adult male Wistar rats between 225 and 250 g weight were randomly stratified into four homogeneous groups (group n= 10): the untreated control, Group1 receiving ceftriaxone 250 1 kg/day -1 alone, Group2 receiving ceftriaxone 250 1 kg/day -1 at the same time receiving L-Carnitine 600 1 kg/day -1 at the same time, and Group3 receiving L-Carnitine 60 Treatments were delivered on a daily basis for a duration of 30 days. Hepatotoxicity was evaluated by quantifying serum concentrations of liver enzymes (AST, ALT, ALP), total serum bilirubin (TSB), and the muscle enzyme CPK. Histological changes of liver were conducted utilizing Hematoxylin & Eosin staining.

Results: Group 1 in the first cohort had severe histopathological changes characterized by hepatic toxicity induced by ceftriaxone treatment with the statistically significant increase ($p < 0.05$) of all the biochemical parameters compared the control. These hepatotoxic changes were significantly mitigated by the concomitant administration of L-carnitine with ceftriaxone (Group 2). Its safety was confirmed by the biochemical profile of the L-carnitine alone (G3) group, which was similar to that of the healthy control group.

Conclusion: that ceftriaxone causes considerable biochemical changes and hepatotoxicity in rats, predominantly via an oxidative stress mechanism. L-Carnitine demonstrates a substantial and significant protective effect against hepatotoxicity and associated histological structural damage.

1- INTRODUCTION

Cephalosporins are β -lactam antimicrobials used to manage various infections caused by both gram-positive and gram-negative bacteria. Cephalosporins are categorized into 5 generations based on their spectrum of coverage against gram-positive and gram-negative bacteria and their temporal discovery. (Bui, *et al.*, 2024). The third-generation class of cephalosporins is the most commonly prescribed group. These cephalosporins have semisynthetic mutations characterized by distinct replacements in acyl-amido group that is situated on C7, according to Arumugham *et al.* (2023). Ceftriaxone is a third-generation cephalosporin, so it has a good tradition of once-dose intake thus making it more applicable in the intravenous and intramuscular delivery in an outpatient. Its strong ability to resist β -Lactamases is the reason behind its extensive use as opposed to first- and second-generation cephalosporins. It is white to yellow-orange crystalline powder that is well dissolvable in water, insoluble to a limited extent in methanol, and slightly soluble in ethanol; it is resistant to various β -lactamases (Gelaw

et al., 2022). Ceftriaxone is broadly used in the treatment of pneumonia, sepsis, surgical prophylaxis, meningitis, and UTIs, and also used in about 39.216 per cent of surgical patients (Bishaw *et al.*, 2021). Ceftriaxone has been developed for clinical use for over 30 years (Duncan, *et al.*, 2021). ceftriaxone can lead to life-threatening conditions with serious CNS ADRs and even death (Lacroix, *et al.*, 2021). Ceftriaxone has a suppressive effect on the gallbladder and ileal contractility and has been attributed to the development of biliary sludge and pseudolithiasis. It significantly increases small-intestinal, large-intestinal, and total intestinal transit time (Arpacik and Yildiz, 2023). Ceftriaxone treatment showed a decrease in glial fibrillary acidic protein (GFAP) immunostaining and reductions in cerebral malondialdehyde (MDA) and tumor necrosis factor -alpha (TNF α) in patients with radiation induced cerebral injury (Cini *et al.*, 2025). The transient increase of the level of alanine aminotransferase (ALT), aspartate aminotransferase (AST), total bilirubin, albumin, and high-density lipoprotein (HDL) are the signs of 3- hepatocellular injury that is semi-transient in nature, as described by Mohammed (2020). L-Carnitine, a derivative, 3-hydroxy-4-N-trimethylaminobutyrate, is an amino-acid derivative and micronutrient which is involved in translocation of long-chain fatty acids into the mitochondrial matrix to be ingested as 2. Its auxiliary roles are to keep the integrity of membrane in check, to stabilize the CoA/acetyl-CoA ratio in the mitochondrion, and to suppress the lactate production (Gnoni *et al.*, 2020). According to Nazari *et al.* (2021), 4- Carnitine, which is an antioxidant found naturally in the seminal plasma of mammals, prevents lipid peroxidation, reactive oxygen species (ROS), and sperm membranes. Temporal occurrence of L-Carnitine has been observed in epididymal fluid under a secretory mechanism and high concentrations of L-acetyl -carnitine in sperm cells were found to activate sperm motility (Olugbodi *et al.*, 2021). L- Carnitine can have a dual mechanism in the pathogenesis and treatment of non-alcoholic fatty liver disease (NAFLD). Reduction in the L -carnitine levels may inhibit fatty -oxidative metabolism and induce a hepatic lipid deposition. In addition, L-carnitine also produces an anti-inflammatory effect through the up-regulation of the peroxisome proliferator-activated receptor- γ (PPAR- γ) in the liver tissue, according to Savic *et al.* (2020).

Cholestasis and hepatitis are known side effects of ceftriaxone. However, they are primarily mild, occur days to weeks after the drug initiation, and are self-limited in the majority. Severe acute hepatitis is a rarely reported side effect of this drug (Asif, *et al.*, 2023). Liver injury evoked by drugs spans various clinical manifestations ranging from mild biochemical abnormalities to acute liver failure. Due to having a longer half-life and deep tissue penetration, wider spectrum, and an overall positive safety profile, ceftriaxone is an essential part of clinical practice. There is a low likelihood of hepatotoxic effects in most cases, but there are isolated reports of ceftriaxone-induced hepatic injury, which highlights the importance of attention especially in the elderly, where the likelihood is enhanced (Guarino *et al.*, 2022). Close evaluation of a series of biochemical signs is crucial to early identify the emergence of liver dysfunction (Feng *et al.*, 2024). Ceftriaxone requires a longer time, which clinically causes neutropenia, thrombocytopenia, and elevated hepatic enzymes (Mistry *et al.*, 2022). Therefore, the current research was meant to clarify ceftriaxone-induced rat endpoint hepatotoxicity and to test the hypothetical hepatoprotective activity of L-carnitine. The study is significant because to the widespread clinical use of ceftriaxone as a third-generation antibiotic. Although hepatotoxic effects are rare, they can be significant, particularly in older or at-risk patients. Therefore, it is essential to create protective medications that can mitigate these risks. The research highlights the antioxidant properties of L-carnitine and its ability to safeguard liver cells from oxidative stress, proposing its use as a supplementary therapeutic or preventive agent during extended or high-dose antibiotic therapy.

2- MATERIAL AND METHODS

2.1 Animal

The present research is shown on forty healthy adult male rats weighing between 225-250 grams. The animals were kept in separate cages in a regulated environment with an ambient temperature of ($30 \pm 3^{\circ}\text{C}$) and humidity of ($62 \pm 5\%$). A natural photoperiod was established and the rats were provided an ad libitum access to normal pelleted feed and water during the study period.

2.2 Design of study

Forty equal numbers of rats (ten subjects each) were taken out and assigned to different groups. After that, the animals are. The first group is the control, the second group is treated with ceftriaxone, the third group with ceftriaxone and L-carnitine, and the fourth group is treated with L-carnitine for 30 days. After that, take a sample from the liver of all rats.

- The control group was given an oral dose of distilled water Once daily for 30 days.

- Group 2: injected (intramuscular) with 250 mg/kg of ceftriaxone daily for 30 days.
- Group 3: Ceftriaxone 250 mg/kg, intramuscularly, and 600mg/kg orally (Once daily for 30 days).
- Group 4: Oral administrations of 600mg/kg/ L-Carnitine Once daily for 30 days.

Serum liver biochemistry were examined by measuring alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP) and bilirubin by use of a normal biochemical analyzer.

2.3 Histological examination

The microscopic examination process is carried out according to the method (Bancroft, J.D., and Suvarna .K.S., 2018), which begins by taking a sample from the liver of an animal's, and it is passed through a series of operations, (fixation, washing, dehydration, clearing, infiltration, embedding, sectioning, staining by hematoxylin –eosin stain) and the slides are examined by a light microscope to obtain a histological image.

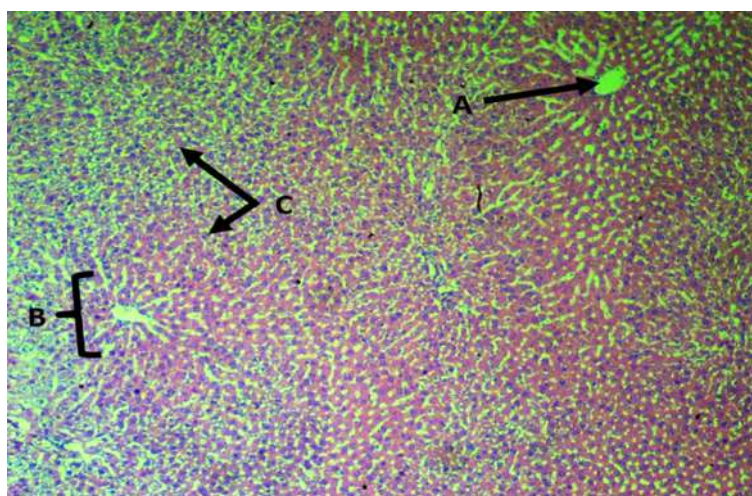
3. RESULT AND DISCUSSION

3.1 Effects of Ceftriaxone and L-Carnitine on Serum Biochemical Parameters

The biochemical analysis revealed significant alterations in liver function markers following ceftriaxone administration, which were markedly attenuated by L-carnitine co-treatment. The comprehensive results are showed in Table 1.

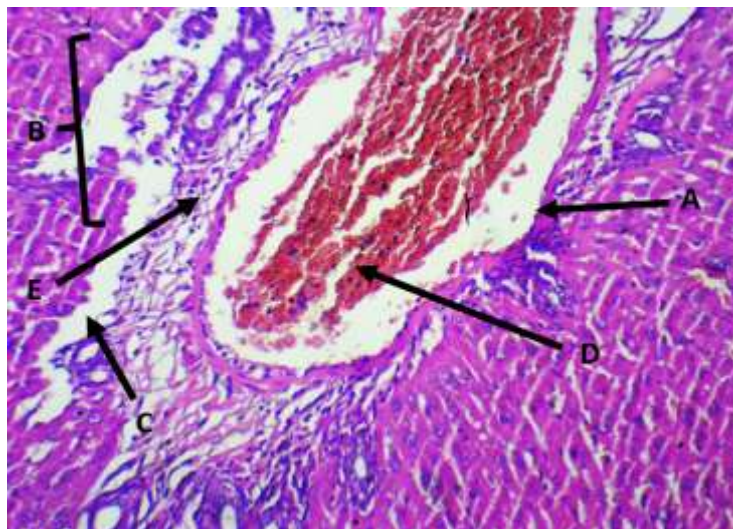
3.2 Effects of Ceftriaxone and L-Carnitine on Liver Tissue

On analysis of the hepatic tissue on the control animals, a normal central vein was present and the hepatic cords extended out of the central vein and were separated into the spaces of sinusoidal nature. The hepatocytes with were round-shaped, which is endorsed by physiological architecture (Figure 1).

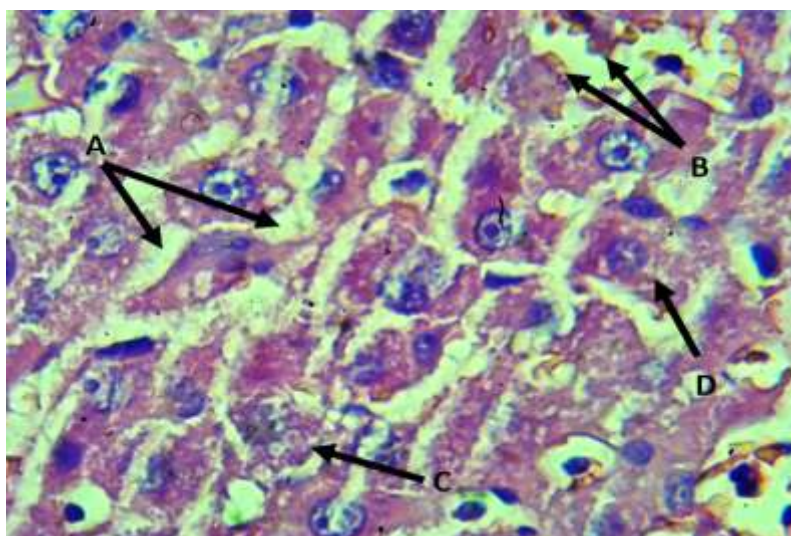


(Figure 1): Liver of male rats in control group appeared normal central vein (A), normal liver cords (B) and normal hepatocytes (C) (H &E 40X)

ceftriaxone treatment of 250 mg/kg Once daily for 30 days. had specific histopathological results as a dilation of the central vessel, and distortion of normal hepatic architecture, and widening of sinusoidal gaps. The hepatic cells had significant enlargement and concentrically organized nuclei, multiple cytoplasmic opacities, vacuolation, congestion of central vein and some hemorrhagic foci. There was necrosis and hepatocellular degeneration which was extensive with the presence of heteromorphic, variable size and shape nuclei (see Figures 2 and 3).



(Figure 2): Liver of male rats with administration ceftriaxone at a dosage of 250 mg/kg b. w. appeared the central vein dilation (A), disruption of normal liver architecture (B), dilation of sinusoids (C), congestion in the central vein (D), accumulation of lymphocytes (E) (H &E 100X).



(Figure 3): Liver of male rats with administration ceftriaxone at a dosage of 250 mg/kg b. w. appeared dilation of sinusoids (A), congestion in the sinusoids (B), degeneration of hepatocytes (C), necrosis of hepatocytes (D) (H &E 400X)

The result of the present study agreement with many authors as Mohammed (2020) who referred to ceftriaxone was induced dilated and congested in central vein and sinusoids. Ooi et al (2023) who referred to the ceftriaxone treatment led to acute liver tissue damage, including vasodilation and congestion severe cellular changes, nuclear condensation and the presence of cytoplasmic vacuoles.

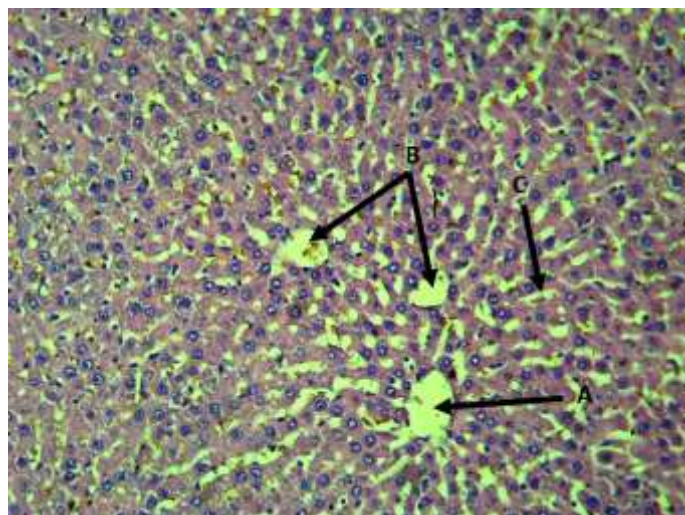
According to El-Megharbel et al. (2022), ceftriaxone causes hepatotoxicity, which is indicated by a high concentration of aspartate aminotransferase and alanine aminotransferase in the serum. This concept is consistent with the results of our study, which confirmed that ceftriaxone causes liver damage due to elevated levels of certain

biomarkers such as AST and ALT.

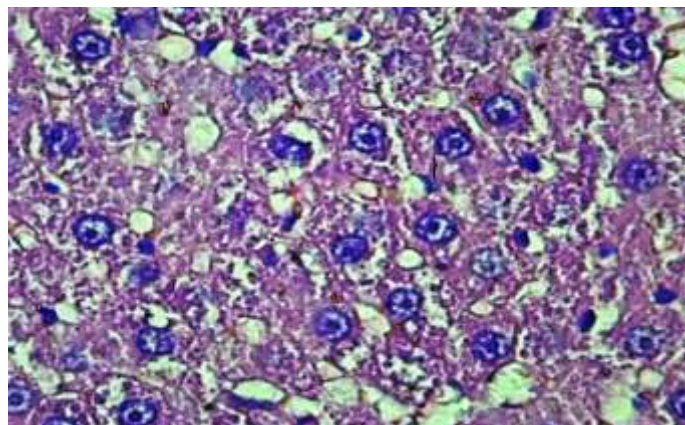
A study by Barath et al. (2022) indicates that bactericidal antibiotics cause tissue damage due to the accumulation of oxidative stress. This was also evident in our study when using ceftriaxone, which stimulated endogenous antioxidant enzymes, thus causing oxidative damage.

The histological alterations in the liver tissue seen were elucidated by El-Megharbel et al. (2022), who highlighted the elevation of reactive oxygen species (ROS) production, evident following ceftriaxone administration in cells. efficient in eliciting detrimental effects of this medication on the architecture and functionality of tissues.

Administration the L-carnitine (600 mg/kg) for 30 days with ceftriaxone (250 mg/kg/day) exhibited the histological changes represented by reduced of central vein dilatation and congestion. reduced of Sinusoidal dilation, less of inflammatory and lymphocytic cells infiltration, and decreased hepatocellular degeneration compared with the ceftriaxone group (G1) (Figures 4 and 5).



(Figure 4): Liver of male rats with administration ceftriaxone at a dosage of 250 mg/kg b. w and the L-carnitine (600 mg/kg) appeared reduced of central vein dilatation (A), reduced of central vein congestion (B). reduced of Sinusoidal dilation (C), (H &E 100X)



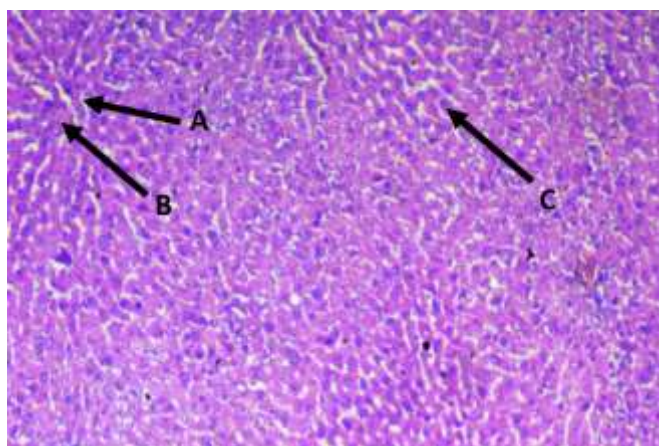
(Figure 5): Liver of male rats with administration ceftriaxone at a dosage of 250 mg/kg b. w and the L-carnitine (600 mg/kg) appeared decreased hepatocellular degeneration and decreased hepatocellular necrosis (H &E 100X).

These results are in line with the evidence earlier that showed that L-Carnitine has antioxidant, anti-inflammatory, and anti-apoptotic effects to improve drug-induced hepatic injury. It promotes mitochondrial β -oxidation through the carnitine shuttle, maintaining cellular energy balance, improving intrinsic antioxidant protection, and reducing oxidative stress and hepatic architecture and hepatic function (Li and Zhao, 2021). These results confirm that carnitine plays a key role in liver tissue protection by limiting the effectiveness of the antibiotic and reducing the cellular inflammation and vasodilation resulting from treatment with it.

Consistent with Askar Pour et al. (2020) who have found a significant decrease in transaminase and γ -glutamyl transferase levels after a carnitine supplementation. This is precisely what we observed in our current study, demonstrating the effect of carnitine in reducing liver transporter enzyme levels.

Abdel-, Ali (2022) argued that hepatoprotection effect of L -carnitine against ceftriaxone-induced damage can be considered multifactorial and includes antioxidant, anti-inflammatory, and anti-apoptotic capabilities. They proposed the role of caspase-activation and phosphorylation of glycogen synthase kinase- 3β (GSK - 3β) -b in ceftriaxone induced cytolytic effect and inflammation in hepatocytes. Therefore, L- Carnitine could be a prospective agent that prevents the hepatic injury caused by ceftriaxone. According to our observations about its impact on hepatic tissue and its significant effectiveness against ceftriaxone-induced toxicity.

The protective administration of carnitine at 600 mg/kg for 30 days exhibited a decrease in histological changes relative to ceftriaxone at 250 mg/kg each day throughout the same period. The carnitine-treated group demonstrated very decreased dilations of the central vein and sinusoidal gaps, lowered congestion in the central vein, and lower infiltration of lymphocytes and inflammatory cells relative to the ceftriaxone group. In this group, carnitine was administered independently of the antibiotic, hence the group is considered normal (Figure 6).



(Figure 5): Liver of male rats with administration the L-carnitine (600 mg/kg) appeared normal central vein (A), normal liver cords (B) and normal hepatocytes (C) (H &E 40X). The concept of L-Carnitine suppressing cell death through inhibition of caspase activity, reduction in parasitic pressure, and enhancement of antioxidant defense systems was explained by Koohpeyma et al. (2021) using different types of tissues. Furthermore, the study by Ögütçü et al. (2024) confirms that carnitine reduces inflammation through its active role in lipid metabolism and energy metabolism regulation. This is clearly confirmed in our current study, which is entirely consistent with the above findings.

Table 1: Effects of 30-day treatment with Ceftriaxone and/or L-Carnitine on serum biochemical parameters in rats

Parameter	Control Group (Distilled water)	G1	G2	G3
AST (GOT) (IU/L)	4.35±0.91 A	6.71± 1.12 B	5.15±0.35 C	4.62±0.78 A
ALT (GPT) (IU/L)	1.72±0.31 A	4.48±1.22 B	3.85±0.33 B	1.80±0.32 A

ALP (IU/L)	8.80±1.12 A	15.50±1.41 B	12.00±0.82 C	8.89±0.35 A
CPK (U/L)	12.70±1.23 A	39.73±1.93 B	20.12±2.18 C	13.7±0.18 AC
Total Bilirubin (mg/dL)	0.02±0.003 A	0.04±0.002 B	0.03±0.002 C	0.02±0.001 A

The letters represented the simificit ($p < 0.05$) in same horizold row.

The results of the study, including a comparison of ceftriaxone (G1) group with the controls showed statistically significant increases ($p < 0.05$) in all the measured liver functions markers and muscle enzyme. This aligns precisely with clinical observations of drug-induced hepatitis (Asif et al., 2023; Guarino et al., 2022) and the elevation of liver enzymes associated with ceftriaxone (Mistry et al., 2022). (Mohammed., 2020), asserts that elevated levels of aminotransferases (AST and ALT) unequivocally indicate hepatocellular damage. The notable increase in (ALT, AST, ALP, CPK and bilirubin) may indicate a systemic inflammatory response or potential damage to muscle tissue. Literature indicates that ceftriaxone impedes intestinal and gallbladder movement, potentially predisposing individuals to cholestasis. This may be the reason for the elevated total bilirubin, signifying impaired hepatic excretory function (Arpacık & Yıldız, 2023).

The primary finding was that, compared to G1, all biochemical markers (ALT, AST, CPK, and bilirubin) were significantly reduced in the group receiving combined ceftriaxone and L-carnitine treatment (G2). AST, ALT, CPK, and bilirubin levels all reverted to values akin to those of the control group. This is likely attributable to L-carnitine's recognized antioxidant properties, which substantiate the study's primary hypothesis (Nazari et al., 2021). Ceftriaxone supposedly causes hepatotoxicity through the generation of reactive oxygen species (ROS), which leads to oxidative stress. L-carnitine is believed to mitigate this process by neutralizing free radicals and protecting cell membranes from lipid peroxidation. Moreover, L-carnitine may alleviate the metabolic burden on the liver following drug exposure by enhancing fatty acid metabolism. (Gnoni et al., 2020).

The group administered L-Carnitine alone (G3) had a biochemical profile that was fundamentally comparable to the control group, despite minor, likely clinically insignificant variations in certain parameters such as ALT and ALP. This suggests L-carnitine's potential as a safe prophylactic treatment and confirms its safety at a dosage of 600 mg/kg.

4. CONCLUSIONS

The Ceftriaxone induced significant hepatic injury in rats and increase elevations in serum liver enzymes and total bilirubin, indicating compromised hepatocyte integrity. Carnitine provides its protective properties by the antioxidant medium. We suggest that L-carnitine may be an effective and useful adjunct treatment to counteract drug-induced liver injuries in the face of ceftriaxone usage.

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