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Research Article

Comparative Study of the Protective Effects of Omega-3 Polyunsaturated Fatty Acids and Alanine on Cyclophosphamide-Induced Hepatotoxicity in Rats

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ABSTRACT

Objective: To evaluate and compare the hepatoprotective effects of Omega-3 Polyunsaturated Fatty Acids (n-3 PUFAs) and Alanine on Cyclophosphamide (CP)-induced hepatotoxicity in rats.

Study Design: Laboratory-based randomized control trial.

Place and Duration of Study: Department of Pharmacology and Therapeutics, Rawal Institute of Health Sciences Islamabad , in collaboration with the National Institute of Health (NIH), Islamabad, from 2nd August 2024 to 31st January 2025.

Methodology: A total of 120 mature male rats with weight between 350 to 500 grams were split into four equal groups at random (n=30). Group A was used as the control and was given regular saline. A single intraperitoneal dosage of CP (150 mg/kg) was given to Group B in order to cause hepatotoxicity. For a total of 10 days, Group D received 400 mg/kg/day of alanine and Group C received 400 mg/kg/day of n-3 PUFAs as pre- and co-treatments, both in addition to CP. On days 0 and 10, blood samples were collected in order to perform liver function testing (ALT, AST, ALP, bilirubin). Tissues from the liver were taken for histological analysis.

Data were analyzed using SPSS v28. Paired t-test, ANOVA and post hoc Tukey's test were applied.

Results: CP administration significantly elevated liver enzyme and bilirubin levels. Both n-3 PUFAs and Alanine reduced these elevations, but Alanine showed greater protective effects, evidenced by lower ALT and bilirubin levels and better histological architecture.

Conclusion: Alanine offers superior hepatoprotection compared to n-3 PUFAs in CP-induced liver injury. Further research is recommended to explore therapeutic applications in clinical settings.

Keywords: Cyclophosphamide, Hepatotoxicity, Omega-3 Polyunsaturated Fatty Acids, Alanine, Antioxidants.

INTRODUCTION

Cyclophosphamide (CP), an alkylating medication, is used to treat a number of cancers and to suppress the immune system in patients with multiple sclerosis, systemic lupus erythematosus, organ transplantation, and other benign illnesses[1]. Cyclophosphamide (CP), a member of the nitrogen mustard family, is categorized as a traditional alkylating agent. It is mentioned in the 22nd edition of the World Health Organization (WHO) Model List of Essential Medicines[2]. According to the Ann Arbor staging criteria, it is mainly advised for use in the treatment of malignant lymphoma stages III and IV, according to the Food and Drug Administration (FDA).These might include Burkitt lymphoma, multiple myeloma, lymphocytic lymphoma, small lymphocytic lymphoma, and Hodgkin and non-Hodgkin lymphoma[3]. Several research have emphasized that acrolein is liable for

weakening cellular antioxidant defense mechanisms and for boosting reactive oxygen species (ROS). CP enhances lipid peroxidation and eventually creates malondialdehyde (MDA), which is a significant oxidative stress molecule and answerable for CP-associated toxicities[4].Omega-3 Polyunsaturated Fatty Acids (n-3 PUFAs) are referred to as necessary fatty acids because the human body is unable to produce them from scratch[5]. The n–3 PUFAs are involved in many physiological functions, such as anti-inflammatory response, maintenance of the cardiovascular system, and cognitive functions[6]. In liver illness, n-3 PUFAs may lower hepatic lipogenesis, inflammation and hepatic fibrosis. A growing body of research indicates that n-3 PUFAs, especially EPA and DHA, reduce inflammation and have a positive impact on a number of inflammation-related conditions, including cancer, rheumatoid arthritis (RA), asthma, inflammatory bowel disease (IBD), and cardiovascular diseases (CVDs). The n-3 PUFAs decrease immunological responses and are utilized as adjuvant immunosuppressive medicines to treat inflammatory illnesses (RA and IBD) or after organ transplantation[7]. A recent meta-analysis done by Yan et al. reveals that n-3 PUFAs supplementation may lower liver fat and hepatic enzyme parameters in individuals with non-alcoholic fatty liver disease (NAFLD)[8]. Supplementing with n-3 PUFAs improved liver fat, some liver enzyme activity, serum TGs, fasting glucose, and insulin resistance, according to a 2018 meta-analysis of randomized controlled trials in individuals with NAFLD[9] . Alanine (a physiologically active version amino acid is an important micronutrient and fat-soluble antioxidant[10]. Alanine (a physiologically active version of amino acid) has caught researchers' interest as a potential adjuvant treatment for many illnesses due to its superior antioxidant and anti-inflammatory capabilities. Free radicals and ROS can be scavenged by alanine. In addition, various studies have indicated that Alanine might block the release of inflammation-mediating chemicals such as eicosanoids and cyclooxygenase-2 enzyme (COX-2)[11]. Alanine demonstrates promising anti-cancer qualities such as lowering cancer cell proliferation, blocking angiogenesis, modulating growth factors, stimulating cell cycle arrest, and causing death[12]. The development of NAFLD has been found to be reduced by high-dose supplementation of alanine [13].The Purpose of this study is to assess the hepatotoxicity caused by Cyclophosphamide (CP) in rats and to look into the preventive effects of Alanine and Omega-3 Polyunsaturated fatty acids (n-3 PUFAs).Specifically, it seeks to determine the extent of CP-induced liver damage, assess the hepatoprotective roles of n-3 PUFAs and Alanine, compare their relative efficacy. Based on prior evidence, the study hypothesizes that Alanine will provide greater protection than n-3 PUFAs. Since CP is a commonly used anticancer and immunosuppressive medication with hepatotoxic side effects, developing safe protective agents is vital. Both n-3 PUFAs and alanine, as natural dietary antioxidants with anti-inflammatory properties, are potential candidates for mitigating CP-induced hepatotoxicity.

METHODOLOGY

In cooperation with the National Institute of Health (NIH), Islamabad, and Rawal Institute of Health Sciences Islamabad, this randomized controlled experimental investigation was carried out at the Department of Pharmacology, Rawal Institute of Health Sciences, Islamabad. The research was completed over a duration of six months. A total of 120 healthy adult male rats were obtained from NIH, Islamabad, using convenient, non-probability sampling. The animals were kept on a regular diet of vegetables and tap water, and they were acclimated for a week in controlled laboratory circumstances (temperature 22 ± 3°C; 12-hour light/dark cycle). Rats with abnormal baseline liver function tests were excluded.

Cyclophosphamide (500 mg; Pharmedic Laboratories, Pakistan) was administered intraperitoneally (I/P) at a hepatotoxic dose of 150 mg/kg on day 6. Omega-3 Polyunsaturated Fatty acids (400 mg; The Protein Works™, UK) and Alanine (400 mg; Merck Pharmaceuticals) were given orally via gastric lavage, beginning six days before CP injection and continuing for four days afterward. A total of 120 rats were randomly assigned to four groups (n = 30 each): Group A (normal control; normal saline), Group B (CP only), Group C (CP + n-3 PUFAs), and Group D (CP + alanine).

On day 0, blood was drawn from the tail vein and on day 10, an intracardiac puncture was performed. Before analysis, Serum was isolated and kept at -20 °C. Commercial kits were used to test biochemical indicators, such as Bilirubin, Alkaline Phosphatase (ALP),

Aspartate Aminotransferase (AST), and Alanine Aminotransferase (ALT), in accordance with manufacturer instructions. On day 10, rats were sacrificed under anesthesia, and liver tissues were excised, rinsed, and preserved at –20 °C for histopathological evaluation. Formalin fixation, paraffin embedding, sectioning, and hematoxylin–eosin staining were performed. Histopathological alterations were graded on a five-point scale ranging from 0 (normal) to 5 (severe necrosis involving the entire lobule).

To evaluate the data, SPSS version 28 was employed. Results were given as mean ± SD. Paired t-test was employed to evaluate biochemical markers between baseline and day 10 within groups. ANOVA and Tukey's post hoc test were used to compare the groups.

The Chi-square test was used to analyze the histopathological data. A p-value < 0.05 was judged statistically significant.

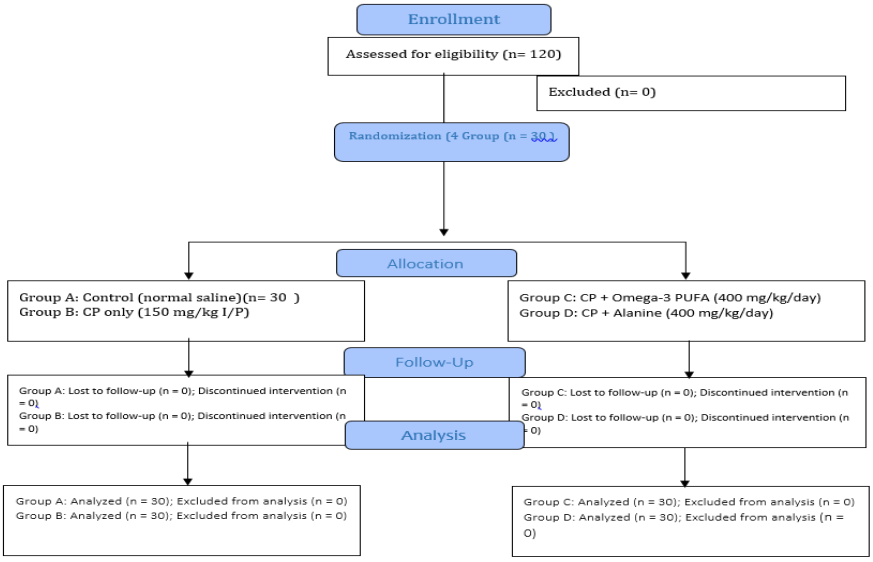
Inclusive Criteria

Only healthy adult male rats weighing 350-500gm were included.

Exclusion Criteria

Animals with abnormal baseline liver function tests were excluded.

Sample Patient Flow Diagram



RESULTS

Table 1. Effect Of Cyclophosphamide, N-3 Pufas, and Alanine on Serum Bilirubin, ALT, AST, and ALP Levels in Rats (N=120)

	Group A Day 0 Mean ± SD	Group A Day 10 Mean ± SD	Group A p (paired)	Group B Day 0 Mean ± SD	Group B Day 10 Mean ± SD	Group B p (paired)	Group C Day 0 Mean ± SD	Group C Day 10 Mean ± SD	Group C p (paired)	Group D Day 0 Mean ± SD	Group D Day 10 Mean ± SD	Group D p (paired)	ANOVA p (Day 0)	ANOVA p (Day 10)
Bilirubin	0.2087 ± 0.06927	0.1843 ± 0.07094	0.079#	0.2253 ± 0.06967	0.3513 ± 0.16458	<0.001*	0.2827 ± 0.28188	0.6637 ± 0.84882	0.001*	0.3467 ± 0.41723	0.5000 ± 0.63305	0.001*	0.154	0.006*
ALT	64.6	68.3	0.28	78.7	405.	<0.	66.5	174.	<0.	73.2	144.	0.03	0.0	<

	7 ± 16.36	6 ± 15.09	5#	3 ± 26.46	13 ± 127.87	001*	7 ± 25.13	27 ± 82.28	001*	7 ± 17.92	32 ± 174.13	6*	56#	0.0001*
AST	72.07 ± 34.21	70.25 ± 19.54	0.690#	85.87 ± 53.15	386.30 ± 169.66	<0.001*	94.56 ± 189.00	273.97 ± 573.74	0.017*	49.97 ± 26.49	167.93 ± 360.83	0.083#	0.343#	0.0005*
ALP	66.07 ± 26.49	69.57 ± 25.14	0.188#	82.30 ± 33.87	425.53 ± 146.53	<0.001*	76.87 ± 34.75	180.40 ± 93.04	<0.001*	85.47 ± 37.08	121.95 ± 55.09	<0.001*	0.123#	<0.0001*

Note: *Statistically Significant (p value<0.05), # statistically non- significant (p value>0.05)

The Serum Bilirubin, ALT, AST, and ALP levels in all groups are summarized in Table 1. Paired t-test analysis showed no significant changes in Bilirubin and ALT levels in Group A from day 0 to day 10, whereas Groups B, C, and D exhibited significant increases. AST levels remained insignificant in Groups A and D but increased significantly in Groups B and C. ALP levels were unchanged in Group A, while Groups B, C, and D demonstrated significant elevations.

One-way ANOVA revealed no significant differences in Bilirubin, ALT, AST, and ALP levels among groups on day 0 ($p = 0.154$, 0.056, 0.343, and 0.123, respectively). However, by day 10, significant intergroup differences were observed for Bilirubin ($p = 0.006$), ALT ($p < 0.001$), AST ($p = 0.005$), and ALP ($p < 0.001$).

Table 2: Tukey's Post Hoc Multiple Comparison Test for Biochemical Parameters among Different Groups (N=120).

Marker	Groups Compared	Mean Difference	Std. Error	p-value	Significance
Bilirubin	A vs B	-0.0167	0.13865	0.625	#
Bilirubin	A vs C	-0.4793	0.13865	0.004	*
Bilirubin	A vs D	-0.3157	0.13865	0.109	#
Bilirubin	B vs C	-0.3123	0.13865	0.115	#
Bilirubin	B vs D	0.1487	0.13865	0.707	#
Bilirubin	C vs D	0.1637	0.13865	0.64	#
ALT	A vs B	-336.77	29.91	<0.001	*
ALT	A vs C	-105.91	29.91	0.003	*
ALT	A vs D	-75.97	29.91	0.059	#
ALT	B vs C	230.87	29.91	<0.001	*
ALT	B vs D	260.82	29.91	<0.001	*
ALT	C vs D	29.95	29.91	0.749	#
AST	A vs B	-316.05	90.23	0.004	*
AST	A vs C	-203.72	90.23	0.114	#
AST	A vs D	-97.69	90.23	0.701	#
AST	B vs C	112.33	90.23	0.6	#
AST	B vs D	218.37	90.23	0.079	#
AST	C vs D	106.03	90.23	0.644	#
ALP	A vs B	-355.97	23.73	<0.001	*

ALP	A vs C	-110.83	23.73	<0.001	*
ALP	A vs D	-52.38	23.73	0.127	#
ALP	B vs C	245.13	23.73	<0.001	*
ALP	B vs D	303.58	23.73	<0.001	*
ALP	C vs D	58.45	23.73	0.071	#

Note: *Statistically Significant (p value<0.05), # Statistically non- significant (p value>0.05)
Post Hoc Tukey's test showed no significant differences in Serum Bilirubin levels between the groups on day 10. ALT levels were also comparable between Groups C and D. For AST, only Group B showed a significant increase compared to Group A, while differences among Groups B, C, and D were not significant. In contrast, ALP levels were significantly elevated in Group B compared to Groups A, C, and D, whereas the difference between Groups C and D was insignificant (Table 2).

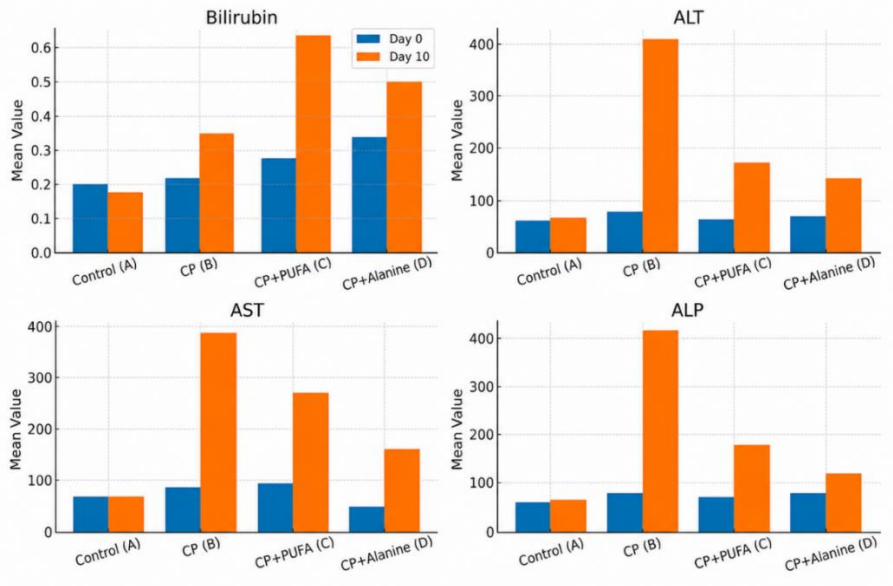


Figure 1. Multi-Panel Biochemical Analysis Showing Serum Bilirubin (Panel A), ALT (Panel B), AST (Panel C), and ALP (Panel D) Levels at Day 0 and Day 10 across the Experimental Groups: Control (Group A), CP (Group B), CP+PUFA (Group C), and CP+Alanine (Group D)

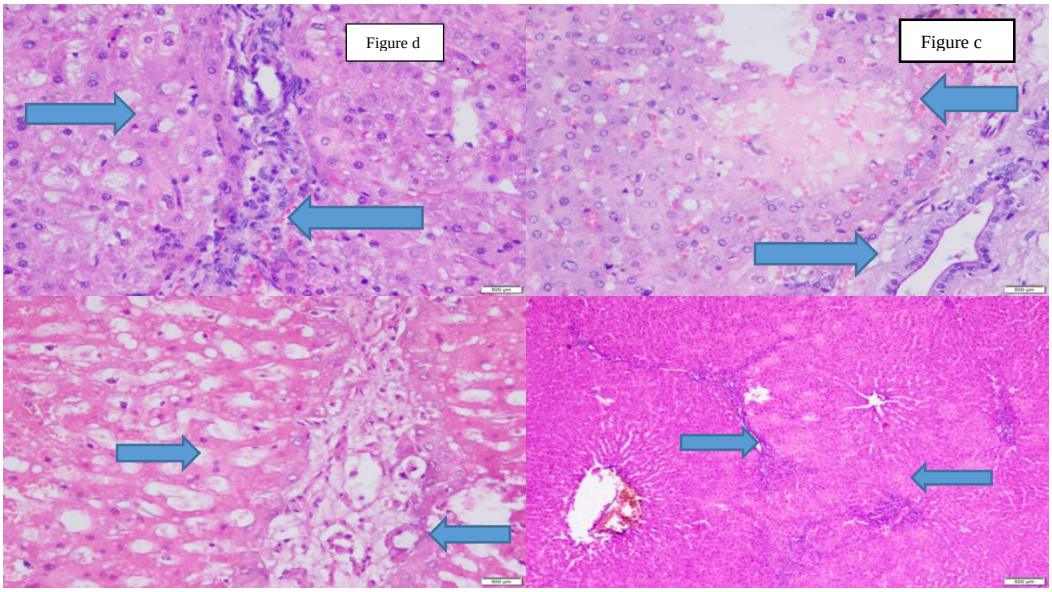
The data is presented as Mean ± SD. For statistical analysis, the paired t-test, one-way ANOVA, and post hoc Tukey's test were employed.

Table 3. Histopathological Findings across Groups Assessed By Chi-Square Test (N=120)

Histological Grading	Group A	Group B	Group C	Group D
Normal	30	0	0	0
Minimal-Mild	0	0	0	30
Mild-Moderate	0	0	30	0
Moderate-Severe	0	15	0	0
Severe-Multifocal	0	15	0	0

Control livers showed normal architecture. CP-treated livers exhibited severe necrosis and inflammation. N-3 PUFAs provided partial protection, while Alanine markedly preserved hepatic structure.

Figure 2



Histopathological examination of hepatic tissues under light microscopy.

Figure a: Essentially normal hepatic architecture in Group A (Negative control, 10x 40x).

Figure b: Severe centrilobular necrosis and steatosis in Group B (Cyclophosphamide administered)

Figure c: Moderate Pseudo-cell / Lytic necrosis and Steatosis (arrows) in Group C (CP + n-3 PUFAs group) (40x)

Figure d: Mild Pseudo-cell / Lytic necrosis and Steatosis (arrows) in Group D (CP + Alanine group)

40x Arrows indicate the sites of pathological changes.

DISCUSSION

The current investigation indicated that Cyclophosphamide (CP) induced hepatotoxicity in rats, proven by substantial increases in Serum ALT, AST, ALP, and Bilirubin levels, coupled with histological alterations including steatosis and necrosis. On day 10, group B Serum Bilirubin levels were significantly higher than those of group A. On day 10, the Bilirubin levels in groups C and D were lower than those in group B. Group C Bilirubin levels on day 10 were statistically significant when compared to day 0, suggesting that n-3 PUFAs did not sufficiently reduce Serum Bilirubin levels in CP-induced hepatitis [14,15]. Administration of Alanine (group D) likewise did not give any help in avoiding the hepatic damage generated by CP[16].

On day 10, group B Serum ALT levels were significantly higher than those of group A. Levels of ALT in groups C and D were less than that of group B on day 10. In comparison to day 0, Group C Serum ALT levels on day 10 were statistically significant, showing that n-3 PUFAs could not significantly ameliorate CP-induced hepatitis[14,15]. Alanine administration has been shown to be helpful in reducing CP-induced liver damage[16].

On day 10, group B Serum AST levels were considerably greater than group A. Levels of AST in groups C and D were smaller than that of group B on day 10. Group C Serum AST levels on day 10 were statistically significant as compared to day 0, indicating insufficient n-3 PUFA ameliorating activity[14,15]. Alanine administration has been shown to be helpful in reducing CP-induced liver damage[16].

Serum ALP levels were also elevated considerably in group B compared to group A on day 10. Levels of ALP in groups C and D were smaller than that of group B on day 10. ALP levels in group C on day 10 were statistically significant compared to day 0, showing inadequate ameliorating action of n-3 PUFAs[14,15]. Administration of Alanine (group D) also did not provide much benefit in preventing the hepatic injury induced by CP [16].

Steatosis and necrosis seen under high and low power microscopy were used to assess CP-induced liver damage. Significant histological alterations, such as severe steatosis and necrosis, were linked to CP therapy in group B. In groups C and D, the administration of n-3 PUFAs and alanine preserved the liver histology, as evidenced by a decrease in the frequency of specimens exhibiting necrosis and steatosis.[14,16]. Fibrotic nodules were detected in several of the liver specimens. Some slides also revealed evidence

of regrowth. When the groups were compared with each other, it was observed that Alanine conferred better hepatoprotection than n-3 PUFAs. Alanine's ability to reduce CP-induced oxidative stressmay be the likely explanation for this additional protection.

These findings partially align with previous studies conducted in rats. Administration of n-3 PUFAs in Thioacetamide-induced and Alcohol-induced hepatitis decreased Serum Bilirubin, ALT, AST, and ALP levels[14,15]. In contrast, in the CP-induced rat model, n-3 PUFAs demonstrated an insufficient ameliorating effect. Administration of Alanine in Diclofenac-induced hepatitis models in rats demonstrated protective effects, which are similar to the protective effects found in ALT and AST levels in the current research[16]. However, its effect on Bilirubin and ALP levels was limited, indicating a partial hepatoprotective effect. The differences in findings may be attributed to species-specific responses and differences in the hepatotoxic mechanism of CP compared to Thioacetamide, Alcohol, or Diclofenac.

The findings imply that Alanine may have a hepatoprotective impact in CP patients, thereby lowering the risk of liver damage. n-3 PUFAs, while effective in other models, may not give significant protection in CP-induced hepatic impairment, underscoring the necessity for careful assessment of hepatoprotective measures in chemotherapy.

Hence, this study validates the research hypothesis as Alanine appears to be more hepatoprotective than n-3 PUFAs against CP-induced hepatitis based on the biochemical and histopathological findings.

CONCLUSION

Alanine demonstrates significantly greater hepatoprotective potential than Omega-3 PUFAs in CP-induced hepatotoxicity in rats. These results provide credence to its possible use as an adjunctive therapy to lessen liver damage brought on by CP. Further clinical studies are warranted. Further studies using larger sample sizes, longer duration, and combined supplementation may provide additional insights. Moreover, clinical studies are needed to translate these findings into human therapeutic settings.

Future Direction

Future studies should include a larger and more diverse sample from various academic institutions. Longitudinal studies can be conducted to explore causality and psychological progression over time.

Author's contributions

M.S. Mehmood conceived and designed the study, supervised the overall project, and finalized the manuscript. N.S.Bajwa participated in the manuscript's preparation, data collecting, and research design.M Ilyas was responsible for conducting the experimental work, data acquisition, and data management. W.M. Malik performed statistical analysis, interpreted the results, and supported manuscript preparation. Q.A. Haider prepared the initial draft of the manuscript, contributed to revisions, and managed the refences. F.Q. Malik provided pharmacological expertise, critically reviewed the manuscript, and approved the final version for submission.

Conflict of Interest

No conflict of interest has been declared by the authors

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