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Protective effects of *Adansonia digitata* extract on acetaminophen-induced hepato-renal injury in rats

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Acetaminophen (paracetamol) overdose is a well-established cause of hepatic and renal toxicity due to oxidative stress and cellular damage. *Adansonia digitata* (baobab) pulp is rich in antioxidants and has been traditionally used for various therapeutic purposes. Aims of this article – to estimate the hepatoprotective and nephroprotective effects of *A. digitata* pulp extract against acetaminophen-induced liver and kidney injury in female albino rats. Forty adult female rats were divided into 4 groups. Group I (control), Group II (acetaminophen 500 mg/kg i.p. every day for 7 days), Group III (*A. digitata* extract 500 mg/kg/day orally for 15 days), and Group IV (co-treatment with acetaminophen and *A. digitata* extract). Liver and kidney functions were assessed through (ALT, AST, ALP, creatinine, and urea) markers, oxidative stress parameters (MDA, GSH, SOD, TAOS), and histopathological examination of liver and kidney tissues. Acetaminophen caused a significant increase in liver enzymes, creatinine, urea, and MDA levels, along with marked diminution of GSH, SOD, and TAOS, indicating hepatic and renal oxidative damage. Histopathological analysis corroborated these findings, revealing hepatocellular necrosis and renal tubular degeneration. Rats treated with *A. digitata* extract alone maintained near-normal biochemical and histological profiles. Co-treatment with the extract significantly ameliorated acetaminophen-induced alterations, though not to control levels, suggesting partial protective effects. The findings show that the pulp extract of *A. digitata* has protective effects on the liver and kidneys due, to its antioxidant properties. This highlights its promise as a natural therapeutic candidate for mitigating acetaminophen-induced organ toxicity.

Keywords: *Adansonia digitata*; hepatoprotection; nephroprotection; acetaminophen toxicity; oxidative stress; antioxidant therapy; female albino rats.

Introduction

Acetaminophen (paracetamol) is used as a pain reliever and fever reducer that can be bought without a prescription. Although its generally seen as safe when taken in recommended amounts for treatment purposes an excessive intake of acetaminophen can result in liver damage and failure in people and animals by causing a buildup of harmful byproducts that trigger oxidative stress and cell death, in the liver (Ramachandran & Jaeschke 2021). The main cause of liver damage due to acetaminophen is mostly linked to the creation of N-acetyl-p-benzoquinone imine (known as NAPR) a substance that reduces glutathione levels and harms cell proteins and membranes according to Luo et al. (2023). In light of the constraints associated with existing treatment options like N-acetylcysteine (known as NAC) which require timing for administration to be effective there is a rising interest in exploring substances with antioxidant and liver protective qualities as potential alternative or complementary treatments (Galicia Moreno et al. 2024). Throughout history, natural herbs and plants have been essential in medicine practice and there is a growing interest in exploring their ability to lessen the harm caused by medications to our organs.

The African baobab tree (*Adansonia digitata* L.) is a plant native to sub- Saharan Africa known for its nutrient rich fruit pulp containing vitamin C and various beneficial compounds, like flavonoids and polyphenols that exhibit strong antioxidant and anti-inflammatory properties as noted by Dogara & Al Zahrani (2024). Studies conducted before have indicated that extracts from the *A. digitata* plant may provide protection against conditions associated with oxidative stress as mentioned by Monroy García et al. (2025). However, there is scarce information available regarding its effectiveness, in alleviating drug induced liver damage.

The current research seeks to assess how effective the extract from *A. digitata* fruit is in protecting the liver against damage caused by acetaminophen in albino rats, focusing on liver function markers and examining tissue samples from the liver and kidneys for any protective effects.

Materials and methods

This study took place at the College of Veterinary Medicine at the University of Mosul. It was carried out with approval from the Postgraduate Studies Committee while following institutional guidelines, for the treatment and use of laboratory animals.

Forty healthy adult female albino rats aged between 6–12 weeks weighing of 250 to 300 grams were obtained from the College of Veterinary Medicine at the University of Tikrit. The rats left for two weeks to adapt to the laboratory environment with ambient temperatures from 20–25 °C and humidity between 40% to 60% following a 12 hr light/12 hr dark regime. During this period of acclimation, the rats were provided with access to a regular pellet diet and clean drinking water.

The animals were divided into 10 animals per group:

Group I, the control, was given an oral dose of normal saline (equivalent to one milliliter, per day) for a duration of 15 days.

Group II participants were given acetaminophen (paracetamol) with a dosage of 500 mg per kilogram administered every day over a span of 7 days.

Group three (*A. digitata*) rats were administered a dose of 500 mg/kg of *A. digitata* pulp extract orally for a duration of 15 days.

Group IV participants were administered a combination of acetaminophen at a dosage of 500 mg/kg every day for a span of 7 days along with *A. digitata* extract at 500 mg/kg daily delivered orally, over the course of 15 days.

Adansonia digitata extract preparation: the dried pericarps of *A. digitata* were sourced from Sudan. The pulp was carefully separated, extracted, and purified. The dosage administered (500 mg/kg/day) was calculated based on body surface area and delivered via oral gavage for 15 days.

Paracetamol (acetaminophen) was obtained from Samara Pharmaceutical Company (Iraq). Its molecular weight is 151.16 g/mol with a melting point of 169 °C. The compound was gently heated in deionized water with a small amount of propylene glycol. Then administered i.p. at a dose of 500 mg/kg every day for 7 days.

At the end of the experiment, 2–3 mL of blood was collected via retro-orbital puncture into capillary tubes. Blood samples were left to clot for 20 minutes, then centrifuged at 1500 rpm for 15 minutes. Then the serum was stored at –20 °C.

The International Federation of Clinical Chemistry (IFCC) guidelines were followed in determining ALT activity. L-alanine and 2-oxoglutarate were converted to pyruvate and L-glutamate by ALT. NADH then lactate dehydrogenase (LDH) was used to decrease the pyruvate, resulting in the production of L-lactate and NAD^+.The rate of NADH oxidation, measured spectrophotometrically as a decrease in absorbance, correlates with ALT activity.

AST was measured using a similar IFCC-compliant method. It catalyzed the conversion of L-aspartate and 2-oxoglutarate into oxaloacetate and L-glutamate. Oxaloacetate was then reduced by NADH in the presence of malate dehydrogenase (MDH), forming malate and NAD^+. The decline in NADH absorbance at 340 nm indicates AST activity.

Alkaline phosphatase (ALP) levels were measured using a sandwich ELISA technique. A microtiter plate was coated with specific capture antibodies. After sample addition, a biotin-conjugated secondary antibody and avidin-HRP were applied. The reaction was developed using TMB substrate and the absorption at 450 nm.

To evaluate oxidative / antioxidant status defense mechanisms, liver homogenates were analyzed for (MDA), (GSH), (SOD), and (TAOS). Liver tissues were collected post-mortem and homogenized in ice-cold phosphate-buffered saline (PBS, pH 7.4), followed by centrifugation at 3000 rpm for 10 minutes at 4 °C to obtain clear supernatants for biochemical assays.

MDA levels, an index of lipid peroxidation, were fixed using the thiobarbituric acid reactive substances (TBARS) method as described by Ohkawa et al. (1979). Absorbance at 532 nm.

GSH concentrations were assessed by method of Ellman (1959), which involves the reduction of 5,5'-dithiobis-(2-nitrobenzoic acid) (DTNB) by GSH to produce a yellow-colored complex measurable at 412 nm.

SOD activity was determined based on its ability to inhibit the autoxidation of pyrogallol, according to the method of Marklund & Marklund (1974). One unit of SOD activity was defined as the amount of enzyme required to inhibit the rate of pyrogallol autoxidation by 50%, with absorbance read at 420 nm.

TAOS was evaluated by a commercial colorimetric assay kit (e.g., Cayman Chemical, USA), following the manufacturer's instructions. The assay quantifies the cumulative antioxidant capacity in the tissue homogenates by comparing the ability of the sample to inhibit oxidation against a standard, and results were reported in mmol Trolox equivalents per liter.

All assays were performed in triplicate to ensure reproducibility, and protein concentrations were determined using the Bradford method (Bradford, 1976) for standardization of results.

Liver organ and kidney organ tissues were harvested post-euthanasia, fixed in 10% buffered formalin, and processed for histopathological evaluation. They were examined under light microscopy for signs of renal and hepatocellular damage after being stained with hematoxylin and eosin.

Duncan's multiple range test was used for post-hoc comparisons after one-way analysis of variance, with significance set at $P < 0.05$. The analysis was carried out using the methodology outlined by Suvarna et al. (2018).

Results

The data in Table 1 show that acetaminophen administration (Group 2) caused a substantial increase in liver enzymes (AST, ALT, ALP), creatinine, and urea levels compared to the control group (Group 1), indicating marked hepatic and renal injury. Conversely, rats treated with *A. digitata* alone (Group 3) maintained near-normal biochemical values, similar to the control. In the co-treatment group (Group 4), enzyme levels were significantly reduced compared to Group 2, although still elevated relative to the control, suggesting a partial hepatoprotective and reno protective effect.

Table 1
Effects of *Adansonia digitata* extract on liver and renal function parameters in rats treated with acetaminophen

Group	AST, U/L	ALP, U/L	ALT, U/L	Creatinine, mg/dL	Urea, mg/dL
G1 – Control	35.00 ± 1.58^a	122.20 ± 1.92^a	28.00 ± 1.58^a	0.51 ± 0.02^a	30.05 ± 0.85^a
G2 – Paracetamol	350.00 ± 7.90^d	426.20 ± 24.23^c	300.80 ± 7.46^d	0.91 ± 0.04^c	59.98 ± 3.29^b
G3 – *A. digitata*	42.00 ± 1.58^b	131.20 ± 6.53^a	38.00 ± 1.58^b	0.48 ± 0.02^a	31.64 ± 1.39^a
G4 – Combination	148.80 ± 5.71^c	243.60 ± 9.86^b	117.60 ± 5.59^c	0.65 ± 0.14^b	39.74 ± 14.39^a

Note: mean ± SD (n = 10); different superscripts ($^{a, b, c, d}$) mean substantial differences ($P \leq 0.05$, Duncan's test).

In Table 2 the paracetamol-treated group (G2) recorded a substantial rise in MDA levels and a significant decrease in GSH, SOD, and TAOS levels compared to the control group (G1), indicating marked oxidative stress ($P \leq 0.05$). Treatment with *A. digitata* extract alone (G3) maintained oxidative parameters close to normal, exhibition no substantial difference from the control. The combination group (G4) also demonstrated significantly improved oxidative stress markers compared to G2, with reduced MDA and increased GSH, SOD, and TAOS levels. However, G4 values remained slightly lower than the control and G3, indicating a partial but significant protective effect of *A. digitata* against acetaminophen-induced oxidative damage.

Table 2
Effects of *Adansonia digitata* extract on oxidative stress biomarkers in liver tissue of rats

Group	MDA, nmol/mg	GSH, µmol/mg	SOD, U/mg	TAOS, mmol Trolox equiv./L
G1 – control	0.50 ± 0.02^a	9.50 ± 0.15^b	12.70 ± 0.15^c	32.79 ± 1.24^b
G2 – Paracetamol	2.88 ± 1.39^c	3.20 ± 0.41^a	3.70 ± 0.41^a	15.45 ± 9.37^a
G3 – *A. digitata*	0.43 ± 0.07^a	7.66 ± 2.44^b	12.40 ± 0.40^c	30.35 ± 3.37^b
G4 – combination	1.84 ± 0.38^b	8.10 ± 0.99^b	10.20 ± 0.71^b	29.76 ± 1.79^b

Note: data as mean ± SD (n = 10); superscripts ($^{a, b, c}$) in the same column indicate substantial at $P \leq 0.05$.

Liver sections from the control group showed normal architecture, including intact central veins, polygonal hepatocytes, continuous blood sinusoids, and Kupffer cells. Acetaminophen-treated livers exhibited significant pathology, including lymphocytic infiltration around bile ducts, hepatocellular vacuolar degeneration, coagulative necrosis, and increased Kupffer cells. In the baobab extract group, liver tissue appeared preserved, with well-organized hepatocytes and normal sinusoidal networks. Co-treatment with acetaminophen and baobab extract showed mild hepatocellular vacuolation and narrowed sinusoids, indicating partial protective effects.

Renal sections from the control group revealed normal glomeruli, capsular space, and intact proximal and distal convoluted tubules. Acetaminophen exposure caused glomerular atrophy, expanded capsular space, tubular epithelial vacuolation, and necrosis. Kidneys from the baobab extract group maintained normal histology. The combination treatment group displayed segmented glomeruli, preserved capsular space, and mild vacuolar changes in tubular epithelium, suggesting a mitigating effect of baobab extract on acetaminophen-induced nephrotoxicity.

Discussion

The study utilized a crude extract of *A. digitata* pulp without performing phytochemical profiling to identify or quantify active constituents such as vitamin C, flavonoids, or phenolic compounds. Pprevious literature supports the antioxidant content of baobab fruit (Dogara & Al Zahrani, 2024). Future investigations should incorporate analytical techniques such as HPLC, GC-MS, or LC-MS to confirm and correlate specific phytochemicals with observed biological effects.

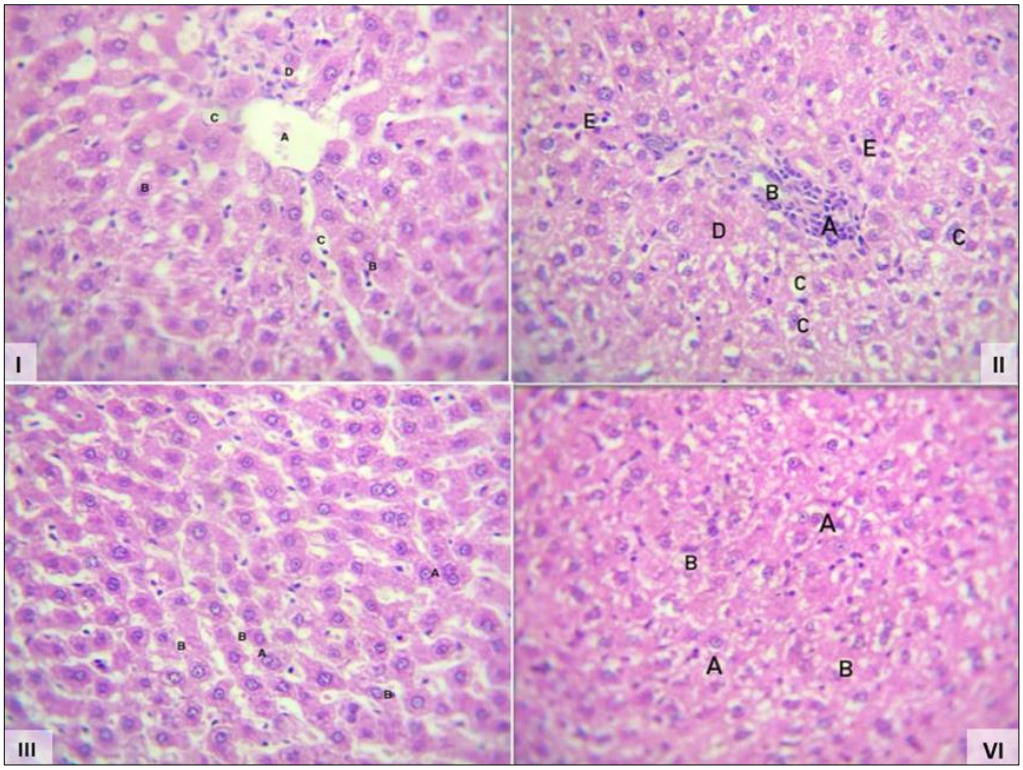

Fig. 1. Sections of the liver: *I* – control group reveals intact liver tissue of central vein (*A*), polygonal hepatocytes (*B*), blood sinusoid continuous (*C*), and Kupffer cells (*D*); *II* – acetaminophen group reveals lymphocytic aggregation (*A*) around the bile duct (*B*) vacuolar cytoplasmic degeneration of hepatocytes (*C*), coagulative necrosis (*D*) multiple Kupffer cells (*E*); *III* – baobab extract group reveals intact liver tissue as polyhedral hepatocytes (*A*) network of blood sinusoids with Kupffer cells (*B*); *VI* – acetaminophen and baobab extract group reveals crowded liver cells with vacuolar cytoplasmic degeneration (*A*) and narrow blood sinusoid (*B*); hematoxylin and eosin stain, X400

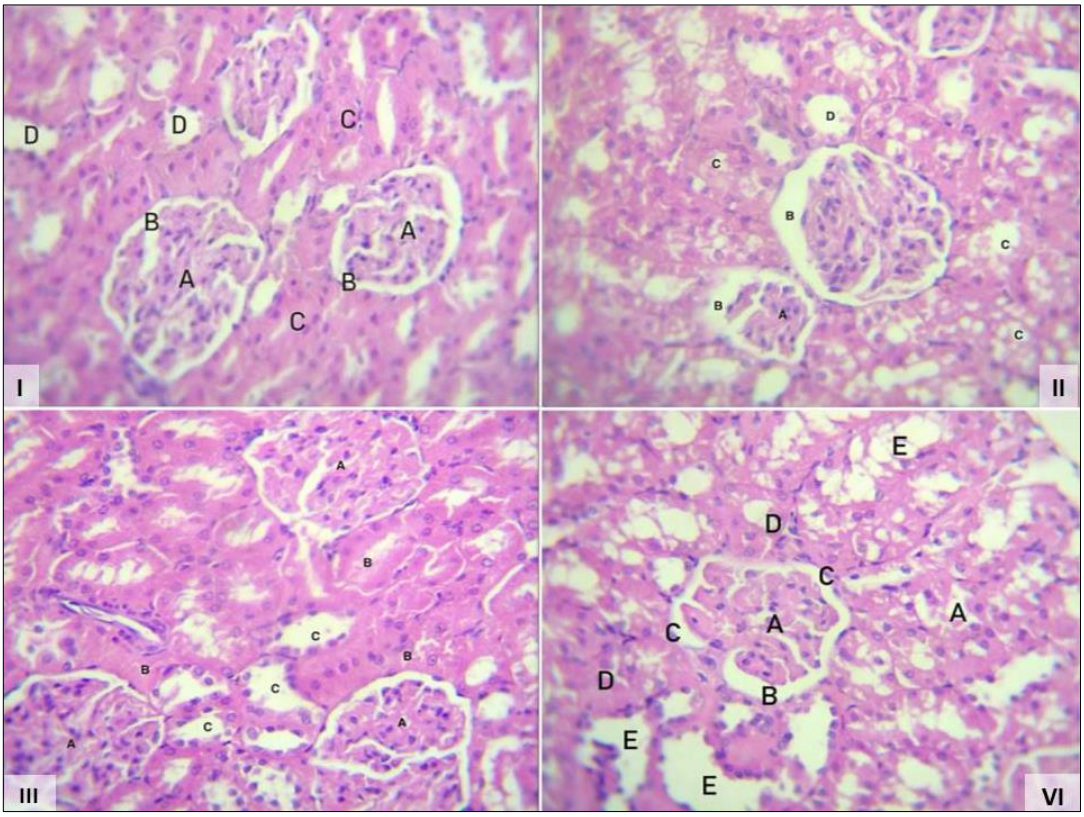

Fig. 2. Histological sections of the kidney: *I* – control group reveals intact renal tissue of glomeruli of normal size (*A*); capsular space (*B*) proximal convoluted tubules (*C*) distal convoluted tubules (*D*); *II* – acetaminophen group reveals cortex of kidney with atrophy of glomeruli (*A*), widening of capsular space (*B*), vacuolar cytoplasmic degeneration of the epithelial cells for convoluted tubules (*C*) and necrosis (*D*); *III* – baobab extract group reveals intact renal tissue of glomeruli of normal size (*A*), capsular space (*B*), proximal convoluted tubules (*C*), distal convoluted tubules (*D*); *VI* – acetaminophen and baobab extract group reveals segmented glomeruli (*A*), intact capsular space (*B*), mild vacuolar degeneration of the epithelium lining proximal convoluted (*C*) and distal tubules (*D*), distal CT (*E*); hematoxylin and eosin stain, X400

The outcome of the current study indicates that *A. digitata* pulp extract significantly ameliorates acetaminophen caused hepatotoxicity and nephrotoxicity in rats. Administration of acetaminophen at a high dose (500 mg/kg) led to substantial elevations in ALT, AST, and ALP – serum biomarkers that reflect hepatic cellular damage, as well as increased serum creatinine and urea levels indicative of renal impairment. These findings are consistent with previously reported models of acetaminophen-induced hepatotoxicity (Begriche et al., 2023; Livoti et al, 2025).

The liver samples from the acetaminophen group displayed signs of drug induced liver damage such as infiltration of immune cells and changes in cell structure and areas of necrosis caused by oxidative stress and inflammation mediated injury to liver cells. Studies, by Li et al. (2023) and Jaeschke et al. (2024) support these findings. On the other hand, rats given *A. digitata* extract exhibited maintained liver structure with minor degenerative alterations present suggesting a somewhat protective impact. These enhancements correspond with research that has shown the conservation provided by plant extracts high in antioxidants (Kumar, 2017; Kazmi et al., 2024).

In the acetaminophen groups, renal histology examination revealed signs of kidney damage such as atrophy and vacoulation of tubular epithelium along with an increase, in capsular space – indicating potential nephrotoxicity (Mohamed, 2021; Adeva-Andany et al., 2023).

The group receiving both treatments showed that their kidney structures were mostly undamaged and there was harm to the tubules compared to the other groups' results, which indicates that *A. digitata* may help prevent kidney injury by possibly maintaining oxidative processes and reducing problems with mitochondrial function (Lee et al., 2024).

The results back the idea that antioxidant treatment does not only influence blood test indicators but also safeguards tissue structure on a tiny scale.

Using only *A. digitata* extract did not cause any harm to the liver or kidneys, which indicates that its safety record is good. However, when *A. digitata* was given alongside acetaminophen it notably decreased the levels of liver and kidney indicators compared to acetaminophen alone; although the values did not completely return to normal levels. This partial improvement shows that the extract plays a role probably due to its antioxidant and anti-inflammatory components such as vitamin C and flavonoids as well as phenolic acids (Magiera et al., 2022; Truong & Jeong, 2022).

The group that received both treatments showed glomeruli and less damage to the tubules as compared to the others, with *A. digitata* potentially lessening kidney damage by stabilizing oxidative processes and decreasing mitochondrial issues (Lee et al., 2024; Villegas Vazquez et al., 2025). The results back the idea that antioxidant treatment does not only influence blood test results but also maintains the structure of tissues on a scale.

Treatment using *A. digitata* extract by itself did not cause any harm to the liver or kidney functions, which highlights its safety features significantly. Notably, when *A. digitata* was given together with acetaminophen it considerably reduced the levels of liver and kidney indicators in comparison to acetaminophen alone; although the values did not return entirely to normal levels. This partial improvement suggests that the extract provides a beneficial function possibly due to its anti-inflammatory and antioxidant components such as vitamin C and various types of flavonoids and phenolic acids.

The notable increase in liver MDA levels in the group treated with acetaminophen (Group 2) confirms the presence of damage and lipid peroxidation – key indicators of liver damage caused by N-acetyl-p-benzoquinoneimine (NAPQI), a harmful byproduct of acetaminophen. This is consistent with studies suggesting that overdosing on acetaminophen leads to production of reactive oxygen species resulting in depletion of glutathione reserves and compromised antioxidant enzyme activity. The significant decrease in GSH (glutathione) SOD (superoxide dismutase) and TAOS (antioxidant status) levels observed in G1 indicates a weakened antioxidant defense mechanism that struggles in combating harmful free radicals effectively. This decline intensifies the impairment of mitochondrial function and cellular damage as stated by the research of Sadiq in 2023. Rats that received the *A. digitata* extract (referred as G3) exhibited levels of MDA and improved GSH and SOD activities., suggesting the strong antioxidant properties of the extract. The beneficial effects are probably due to the concentration of flavonoids,vitamin C and phenolic compounds present in the *A. digitata* fruit pulp.These components are recognized for their capability in neutralizing radicals and boosting the production of natural antioxidant enzymes, within the body (Vinha et al., 2024).

Furthermore, the balancing of levels in the *A. digitata* group indicates an overall enhancement, in the body's ability to combat oxidative stress effectively These results align with earlier research demonstrating the liver protective properties of baobab extract in different models of oxidative stress (Hamad et al., 2022; Kumar et al., 2024).

Conclusion

The evidence gathered suggests that the *Adansonia digitata* tree showcases liver protection abilities by utilizing its antioxidant features to help alleviate acetaminophen- induced liver damage.

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