# Experimental Evaluation of *Buchananialanzan* in NDEA + $CCl_4$-Induced Hepatocellular Carcinoma in Rats

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***Abstract:***

Chemical hepatotoxins like diethylnitrosamine (NDEA) and carbon tetrachloride ($CCl_4$) frequently cause hepatocellular carcinoma (HCC), which is a primary cause of cancer-related mortality globally. Conventional therapy is limited because to toxicity and poor prognosis, emphasising the need for safer options. *Buchananialanzan (B. lanzan),* a plant widely used in Indian medicine, includes flavonoids, phenolics, saponins, and tannins that have antioxidant and hepatoprotective activities. In this investigation, rats were exposed to NDEA + $CCl_4$-induced hepatocarcinogenesis and treated with *B. lanzan* extract. Biochemical parameters such as serum liver enzymes, bilirubin, and total protein were measured alongside oxidative stress markers such as superoxide dismutase (SOD), catalase (CAT), and reduced glutathione. A histopathological analysis of liver samples was conducted to determine structural integrity and tumour development. Treatment with *B. lanzan* extract dramatically normalised liver function indices, reduced lipid peroxidation, increased antioxidant enzyme activity, and improved hepatic architecture with less necrosis and fibrosis. These data suggest that *B. lanzan* has substantial hepatoprotective and chemopreventive properties, which are likely mediated by antioxidant and anti-inflammatory mechanisms. The study verifies its historic use in liver problems.

***Keywords:****B. lanzan,* Hepatocellular Carcinoma, NDEA, $CCl_4$, Oxidative Stress, Hepatoprotection, Phytotherapy.

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## Introduction

Hepatocellular carcinoma (HCC) is the largest cause of cancer-related death worldwide, especially in areas with high rates of chronic liver illnesses such hepatitis B and C, alcoholic liver disease, and non-alcoholic fatty liver disease (1,2). Chemical induction of HCC in animal models, such as diethylnitrosamine (DEN) and carbon tetrachloride ($CCl_4$), is a useful tool for researching hepatocarcinogenesis and evaluating prospective therapies (3,4). These substances cause oxidative stress, DNA damage, and inflammatory responses, which all contribute to liver injury and cancer (5).

*B.lanzan,* often known as Chironji, is a deciduous tree native to India and southeast Asia. Its seeds are frequently consumed and used in traditional medicine to treat liver diseases and other conditions (6). Phytochemical investigations have revealed a variety of bioactive components in plants, including flavonoids, saponins, tannins, and phenolic compounds, all of which are renowned for their antioxidant, anti-inflammatory, and liver-protective effects (7).

plantshave previously been shown to have hepatoprotective properties in a variety of liver injury models. For example, ethanolic extracts of plant leaves protected mice from DEN-induced hepatocellular cancer, as demonstrated by improved blood liver enzymes and histological markers (8). Methanolic and aqueous bark extracts demonstrated hepatoprotective efficacy against paracetamol-induced liver damage in rats (9-11). These findings indicate that several plant sections of *B. lanzan*contain bioactive elements capable of preventing or mitigating hepatocellular damage.

Given this backdrop, the current study sought to assess the hepatoprotective and chemopreventive capabilities of *B. lanzan*extract in a rat model of chemically induced HCC. The study focused on serum biochemical markers, oxidative stress parameters, and histological changes to determine the therapeutic potential.

## Materials and methods:

### Plant material:

### Preparation of plant extract:

*B. lanzan*plant material was collected from the Botanical Garden of the National Botanical Research Institute in Lucknow, India (NBRI). Dr.SayeedaKhatoon, a taxonomist, confirmed the plant material, and the voucher specimens (NAB2B04293, 200495, and 200492) were archived in the departmental herbarium and the Institutional Museum for future reference.

*B. lanzan*fresh plant material was rinsed with distilled water to remove dirt and soil. *Buchananialanzan*powder (100g) was air-dried (30±20c) and extracted overnight with 10 ml of ethanaol. It was then centrifuged at 10000 rev/min on a Rota evaporator (Bouchi, USA) and dried in a lyophilizer (Labeonco, USA) under reduced pressure. The extract was then subjected to phytochemical and pharmacological testing.

**HPTLC analysis:**

Reflux 5 g of finely powdered medication with 25 ml of ethanol over a water bath for 25 minutes three times in a row, then filter and extract the solvent under low pressure. Dissolve 25 milligrammes of extractive in 20 millilitres of ethanol. Using an automator applicator (CAMAG Linomat IV), apply 10 l of extract on Merck percoated silica gel 60 F254 plates with a 0.2mm thickness. The plates were then run using a fresh solvent system (ethyl acetate). Formic acid: Acetic acid. Water in a 100:11:11:27 ratio for *B. lanzan*in a CAMAG twin through chamber up to a distance of approximately 9 cm, dry, and scan. UV 254 and visible light were used to view the plates. If necessary, spray the plate with anisaldehyde-sulphuric acid.If necessary, spray the plate with anisaldehyde-sulphuric acid and heat at 1100°C for 10 minutes. Record the Rf values and colour of the resolved bands, then use the Desaga video documentation unit to document the movie. The plates were densitometrically scanned with a CAMAG TLC scanner at the relevant wavelength. (12)

**In vivo study:**

**Animals:**

Studies are carried out on rats weighing between 140 and 160 g. They were obtained from the toxicity control animal house at the Central Drug Research Institute in Lochnow, as well as cattle raised in the departmental animal facility. The rats were housed in polyacrylic cages with no more than 6 animals per cage and kept under typical laboratory conditions (temperature 25±20C, 12h dark/light cycle). They have free access to a standard dry pellet meal (amrut, India) and unrestricted tap water. The institutional committee for the ethical use of animals reviewed and approved all of the disclosed procedures.

**Induction of Hepatocellular Carcinoma:**

The rats were randomly assigned to the experimental and control groups (n=6). Group 1 rats were given 0.9% normal saline. Group II rats with chemically induced HCC (NDEA+CCl4) were given a single intraperitoneal injection of N-nitroso di ethyl amine (200 +3 mg/kg b.w.s.c). Group III rats received 100 mg/kg b.w.i.p. of *B. lanzan*in 50% ethanol (EtoH). Groups IV and V were given i.p injections of 200 and 400 mg/kg/b.w, respectively, whereas Group VI rats received 6 mg/kg b.w of 50% EtoH*B. lanzan.*

**laboratory investigations:**

Body weights were measured on the day of receipt, before randomisation, on the day of dosing, and weekly thereafter for the treatment and recovery groups, while food and drink consumption were documented daily and reported weekly. All animals had their blood drawn for haematology and clinical biochemistry. Animals were placed in metabolic cages and fasted overnight before blood sample, but they had unlimited access to water. Blood was drawn from the retroorbital plexus using a micro-hematocrit heparinised glass capillary tube.
Potassium EDTA was employed as an anticoagulant during haematology tests. Blood samples were stored in serum tubes at room temperature for around 30 minutes before being aliquoted. After clotting, the blood tubes were centrifuged at 3000 rpm for 15 minutes. The supernatants were decanted and stored at 700°C for subsequent analysis.

**Biochemical marker estimation:**

The biochemical markers like SGOT(U/l), SGPT(U/l), SALP(U/l), Bilirubin level (U/l) and Gamma glutamyltranspeptidase, GGT (U/l) were determined for both control and treated groups by using standard biochemical method. (13)

**Estimation of free radical generation:**

The liver homogenate (5%) in ice-cold phosphate buffer was centrifuged at 800X g for 10 minutes, followed by centrifugation of the supernatant at 12,000X g for 15 minutes to obtain the mitochondrial fractions, which were used to measure lipid peroxidation (LPO), superoxide dismutase activity (SOD), catalase activity (CAT), reduced glutathione (GSH), and glutathione peroxidase (GPX). (14)

**Hematological estimation:**

Red blood cell counts, White blood cell counts and hemoglobin were estimated with the help of hematology analyzer (Medonic CA620, Boule, Sweden). (15)

**Histopathological analysis:**

The livers were removed immediately upon autopsy for histological examination, and the tissues were stored in 10% formalin for at least 24 hours. The paraffin sections were then treated (Automatic Tissue Processor, Lipshaw) and sliced into 5 um-thick sections using a rotary microtome. The sections were then stained with haematoxilin-eosin dye (Merck) and mounted on Canada balasam. The histology slides were examined and photographed under a picture zoom microscope (3.2X10 and 10X10). (16)

**Statistical analysis:**

Data are expressed as mean +SEM (standard error of mean). The difference among means has been analyzed by unpaired students t-test.

## Results:

**HPTLC analysis of 50% EtOH extract of*B. lanzan:***

*B. lanzan*TLC plates with a 50% EtOH extract were visible at UV 254 nm. The plates were coated with anisaldehyde-sulfuric acid and then heated to 1100 degrees Celsius for 10 minutes. A band (Rf=0.51), colour of the resolved bands, and video were documented. The plates were densitometrically scanned using a CAMAG TLC scanner at 254 nm (Figure 1). The plant was extracted with 50% EtOH, producing a percentage yield of 6.26 (Table 1).

**Figure. 1. HPTLC finger print of ethanolic extract of *B. lanzan*-EtOAcToluene: EtOAc : Formic acid(5:5:1)**

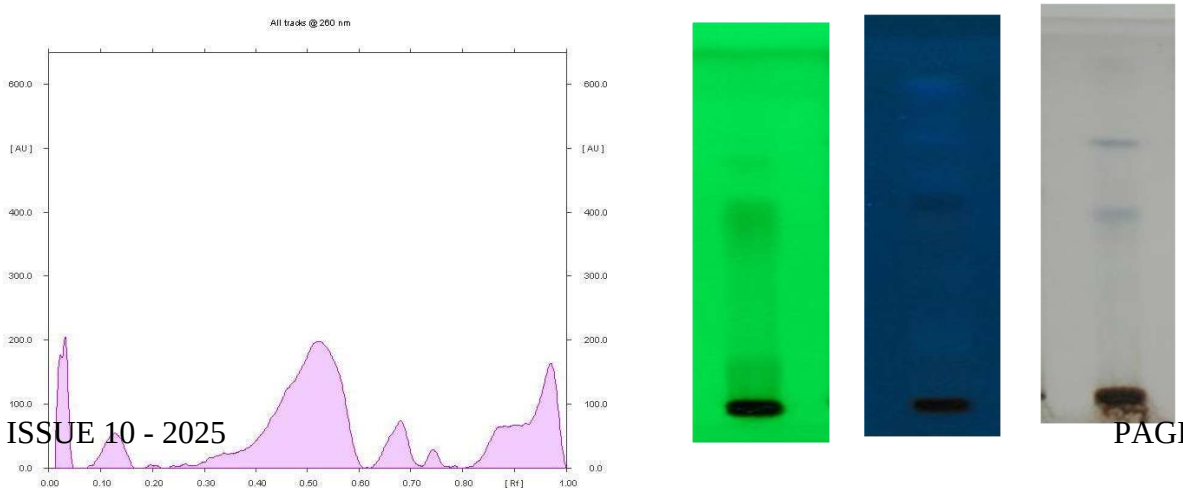


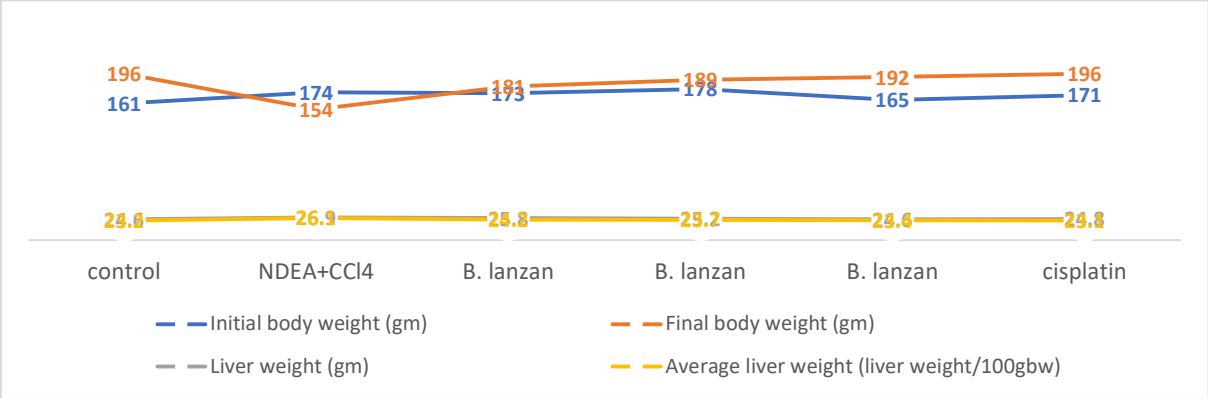
**Table 1: HPTLC values**

| Peak | Start Rf | Height % | End Rf | Area % |
|---|---|---|---|---|
| 1 | 0.12 | 17.03 | 0.14 | 12.93 |
| 2 | 0.15 | 23.99 | 0.16 | 14.48 |
| 3 | 0.16 | 13.41 | 0.20 | 11.47 |
| 4 | 0.23 | 11.67 | 0.30 | 11.52 |
| 5 | 0.33 | 11.26 | 0.36 | 10.62 |
| 6 | 0.36 | 11.39 | 0.40 | 11.05 |
| 7 | 0.50 | 12.98 | 0.57 | 13.30 |
| 8 | 0.57 | 17.84 | 0.68 | 13.44 |
| 9 | 0.68 | 24.19 | 0.81 | 35.74 |
| 10 | 0.82 | 18.05 | 0.87 | 15.49 |
| 11 | 0.87 | 18.21 | 0.95 | 19.04 |
| 12 | 0.96 | 18.73 | 0.103 | 19.40 |
| 13 | 0.98 | 32.26 | 1.10 | 31.53 |

**Effect of 50% ethanolic extract of*B. lanzan*on body weight, liver weight and average liver weight in (NDEA+CCl4) induced HCC rat**

50% ethanolic extracts of*B. lanzan,* at a dose of 100, 200 mg and 400 mg once daily for 28 days and standard Cisplatin at a dose of 6 mg/kg were subjected for studying the body weight, liver weight and average liver weight in HCC rats. The study showed that the liver weights were significantly increased from 19.3,15.33 to 22.8,15.78 in NDEA+CCl4 group to 21.1,15.36 to 19.8, 10.66 in *B. lanzan*treated animal. whereas, standard drug Cisplatin 6 mg/kg showed significant reduction in liver weight compared to NDEA+CCl4 group figure 2.

**Figure 2: Effect of 50% ethanolic extract of *B. lanzan* on body weight, liver weight and average liver weight in (NDEA+CCl4) induced HCC rat**




Values are mean of 6 rats in each group

**Effect of 50% ethanolic extract of *B. lanzan*on SGOT, SGPT, SALP, BL and GGT against NDEA+CCl4 induced HCC**

In the NDEA+CCl4 group, the level of SGOT (211.22-382.21, P<0.001), SGPT (101.25-398.51, P<0.001), SALP (251.11-457.38, P<0.01), BL (0.96-1.52, P<0.001) and GGT (50.8-178.9, P<0.001). in contrast, the groups treated with *B. lanzan*extract at dose of (100-400 mg/kg) once daily for 28 days prevented the incidence of cancer of in a dose related manner. The ranges of protection in the serum marker were found to be SGOT (314.14-238.84, P<0.05 to p<0.001), SGPT (345.33-144.03, P<0.05 to p<0.001), SALP (368.22-263.91, P<0.01 to p<0.001), BL (1.38-0.101, p<0.001), and GGT (162.1-94.9, P<0.001) respectively. The protection of cisplatin ranged for SGOT (382.21-216.38, p<0.001), SGPT (398.51-114.38, p<0.001), SALP (457.38-258.34, p<0.001), BL (1.52-0.99, p<0.01) and GGT (178.9-58.9, p<0.001) respectively as shown in Table 2.

**Table 2: Effect of the 50% ethanolic extract of *B. alanzan*on SGOT(U/l), SGPT(U/l), SALP(U/l), Bilirubin level (U/l) and Gamma glutamyltranspeptidase, GGT (U/l) in serum of rat:**

| Groups | Treatment | Dose | SGOT | SGPT | SALP | BL | GGT |
|---|---|---|---|---|---|---|---|
| I | Control | --- | 211.22±3.19 | 101.25±1.58 | 251.11±11.31 | 0.96±0.03 | 50.8±5.1 |
| II | NDEA+$CCl_4$ | 200 mg (NDEA)+3ml/ kgbw($CCl_4$) | 382.21±22.32 z | 398.51±29.78 z | 457.38±28.33$^z$ | 1.52±0.06$^z$ | 178.9±8.2 z |
| III | *B. alanzan* | 100mg/kg | 314.14±19.51 a | 345.33±27.24 | 368.22±24.38$^a$ | 1.38±0.05 | 162.1±6.8 |
| IV | *B. alanzan* | 200mg/kg | 273.48±18.36 b | 238.11±26.21 b | 304.21±23.33$^b$ | 0.109±0.13$^a$ | 144.8±5.6$^b$ |
| V | *B. alanzan* | 400mg/kg | 238.84±18.90$^c$ | 144.03±22.31 c | 263.91±21.83$^c$ | 0.101±0.12$^b$ | 94.9±4.2 c |
| VI | Cisplatin | 6mg/kg | 216.38±19.02$^c$ | 114.38±19.44$^c$ | 258.34±26.62$^c$ | 0.99±0.14 c | 58.9±6.1 c |

Values are mean ± S.E.M. of 6 rats in each group

P values: $^z$<0.001 compared with respective control group

P values: $^a$<0.05, $^b$<0.01, $^c$<0.001 compared with group II (NDEA+$CCl_4$)

**Effect of 50% ethanolic extract of *B. lanzan*on LPO, SOD, CAT, GPX, GST and GSH against NDEA+CCl4 induced HCC**

Administration of NDEA+CCl4 led to increase in the levels of LPO (20.56-26.04, p<0.001), and decrease in SOD (136.12-57.87, p<0.001), CAT (46.02-27.38, p<0.001), GPX (24.58-21.40, p<0.001), GST (21.14-20.49, p<0.001) and GSH (23.48-23.06, P<0.001) levels in the 5% w/v liver homogenate. Treatment of rats with 50% ethanolic extract of *B. lanzan*at dose of (100-400 mg/kg b.w) markedly prevented the NDEA+CCl4 induced alterations of various

parameters LPO (24.89-21.19, p<0.05 to p<0.01), SOD (82.18-112.50, p<0.05 to p<0.001), CAT (31.67-38.89, p<0.05 to p<0.001), GPX (22.67-23.12, p<0.001), GST (20.76-20.98, p<0.001) and GSH (23.19-23.31, p<0.001) respectively. The protection of Cisplatin ranged for LPO (26.04-20.98, p<0.05 to p<0.01), SOD (57.87-126.68, p<0.05 to p<0.001), CAT (27.38-39.02, p<0.05 to p<0.001), GPX (21.40-23.29, p<0.001), GST (20.49-21.04, p<0.001) and GSH (23.06-23.34, p<0.001) respectively as shown in Table 3.

**Table 3: Effect of 50% ethanolic extract of*B. alanzan* on LPO, SOD, CAT, GPX, GST and GSH against NDEA+CCl4 induced HCC**

| Groups | Treatment | Dose | SOD | CAT | LPO | GPx | GST | GSH |
|---|---|---|---|---|---|---|---|---|
| I | Control | --- | 136.12±8.4 | 46.02±1.60 | 20.56±0.01 | 24.58±0.02 | 21.14±0.16 | 23.48±0.34 |
| II | $NDEA+CCl_4$ | 200mg/kg (NDEA)+3ml/ kgbw($CCl_4$) | 57.87±8.12$^z$ | 27.38±0.61$^z$ | 26.04±1.24$^z$ | 21.40±0.01$^z$ | 20.49±0.02$^z$ | 23.06 ± 0.32$^z$ |
| III | *B. alanzan* | 100mg/kg | 82.18±9.56$^a$ | 31.67±1.40$^a$ | 24.89±0.43 | 22.67±0.02$^a$ | 20.76±0.04$^c$ | 23.19 ±0.34 |
| IV | *B. alanzan* | 200mg/kg | 101.29±12.49$^b$ | 34.38±1.32$^b$ | 22.24±0.54$^a$ | 22.92±0.03$^c$ | 20.84±0.02$^c$ | 23.21 ±0.35 |
| V | *B. alanzan* | 400mg/kg | 112.50±9.21$^c$ | 38.89±0.89$^c$ | 21.19±0.82$^b$ | 23.12±0.02$^c$ | 20.98±0.05$^c$ | 23.31 ±0.32 |
| VI | Cisplatin | 6mg/kg | 126.68±6.28$^c$ | 39.02±1.10$^c$ | 20.98±0.62$^b$ | 23.29±0.02$^c$ | 21.04±0.09$^c$ | 23.34 ±0.33 |

Values are mean ± S.E.M. of 6 rats in each group

P values: $z$<0.001compared with respective control group

P values: $a$<0.05, $b$<0.01, $c$<0.001compared with group II (NDEA+CCl4)

**Effect of 50% ethanolic extract of *B. lanzan*on haematological parameters (RBC, WBC and Hb) of control and NDEA+CCl4 induced HCC**

The Table 4 shows the level of Hb, RBC counts, all of which were significantly decreased (30.81-28.34, p<0.001) and (28.32-25.98, p<0.05) and with simultaneous increase in WBC (26.56-29.27, p<0.01) with respect to control. In contrast, the groups treated with *B. lanzan*extract at dose of (100-400 mg/kg b.w) once daily for 28 days prevented the cancer in a dose related manner. The range of protection in the Hb, RBC and WBC shows (28.67-29.78, p<0.05), (26.34-27.79) and (28.45-27.12, p<0.01). the protection of Cisplatin ranged for Hb (28.34-30.12, p<0.01), RBC (25.98-28.12, p<0.05) and WBC (29.27.79-26.32, P<0.01) respectively.

**Table 4: Effects of *B. alanzan*on haematological parameter (RBC, WBC and Hb) of control and (NDEA+$CCl_4$) induced HCC in rat**

| Group | Parameter | Dose | RBC (million/ $mm^3$) | WBC (million/ $mm^3$) | Hb(g/dl) |
|---|---|---|---|---|---|
| I | Control | --- | 28.32±0.68 | 26.56±0.04 | 30.81±0.39 |
| II | NDEA+$CCl_4$ | 200mg/kg(NDEA)+3 ml/kgbw($CCl_4$) | 25.98±0.49$^{X}$ | 29.27±0.80$^{x}$ | 28.34±0.30$^{z}$ |
| III | *B. alanzan* | 100mg/kg | 26.34±0.51 | 28.45±0.69 | 28.67±0.68 |
| IV | *B. alanzan* | 200mg/kg | 26.94±0.29 | 27.96±0.51 | 29.24±0.15$^{a}$ |
| V | *B. alanzan* | 400mg/kg | 27.79±0.81 | 27.12±0.39$^{a}$ | 29.78±0.22$^{a}$ |
| VI | Cisplatin | 6mg/kg | 28.12±0.54$^{a}$ | 26.32±0.34$^{b}$ | 30.12±0.28$^{b}$ |

Values are mean ±S.E.M. of 6 rats in each group

P values: x<0.05, y<0.01, *z*<0.001 compared with respective control group

P values: *a*<0.05, *b*<0.01 compared with group II (NDEA +CCl4)

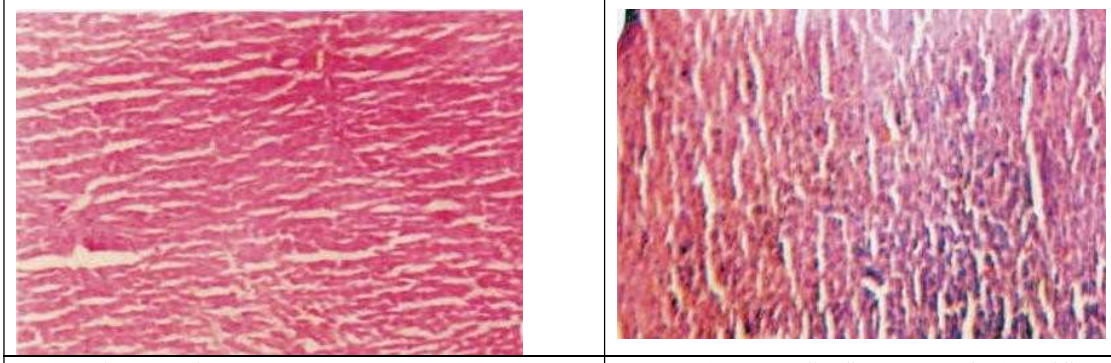
**Histopathological analysis:**

A histopathological analysis of liver tissues was conducted to assess the structural changes caused by NDEA + $CCl_4$ and the preventive effects of *B. lanzan*extract. Liver sections from the control group showed normal hepatic architecture, including well-preserved hepatocytes, prominent central veins, and radiating hepatic cords. In contrast, the HCC control group exhibited significant histological abnormalities such as hepatocyte degradation, widespread necrosis, fatty changes, inflammatory cell infiltration, fibrosis, and nodular development, all of which indicated tumour progression.

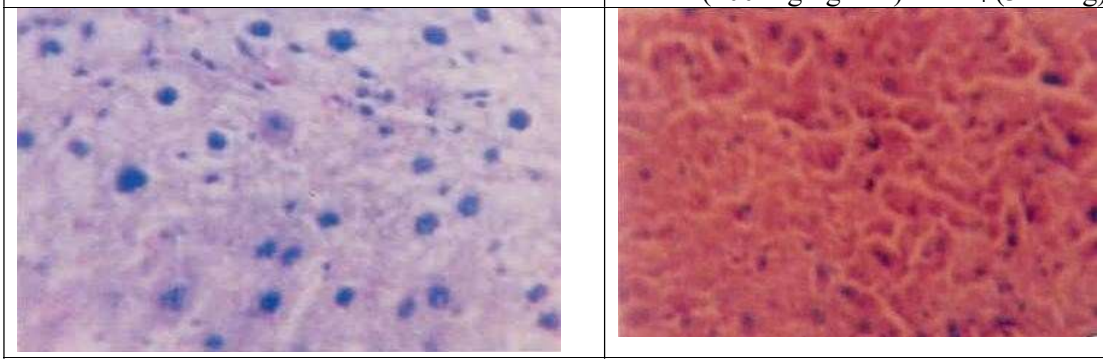
Rats treated with *B. lanzan*extract showed significant improvement in hepatic architecture. Hepatocyte necrosis was reduced, as was inflammatory infiltration, and normal lobular shape was restored. Fibrotic alterations and tumour nodules were significantly reduced in the extract-treated animals, especially at higher doses as shown in figure 3. These data imply that *B. lanzan*has a hepatoprotective effect, which is most likely mediated by its antioxidant, anti-inflammatory, and anti-fibrotic phytochemicals.

The histological observations are consistent with the metabolic and oxidative stress measures, indicating that *B. lanzan* reduces liver damage and stops the progression of chemically caused HCC. The ability to preserve hepatocyte integrity while suppressing fibrotic and neoplastic alterations highlights its promise as a chemo preventive drug.

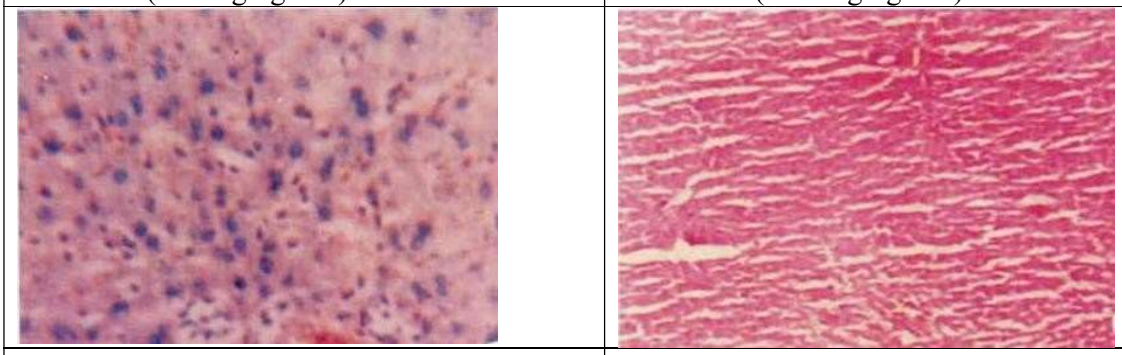
**Figure 3: Histopathological analysis of*B. lanzan* against NDEA+CCl4 induced HCC**



Normal control | NDEA(200 mg/kg/b.w)+ $CCl_4$ (3 ml/kg)



*B. alanzan*(100 mg/kg/b.w) | *B. alanzan* ( 200 mg/kg/b.w)



*B. alanzan*(400 mg/kg/b.w) | cisplatin (6mg/kg/b.w)

Magnification: Haematoxylin and Eosin x 100.

## Discussion

The study found that *B. lanzan*extract effectively reduces biochemical and histological changes caused by NDEA + $CCl_4$, demonstrating strong hepatoprotective and chemopreventive properties. Rats treated with NDEA + $CCl_4$ had higher serum transaminases, alkaline phosphatase, and bilirubin levels, as well as enhanced lipid peroxidation and decreased antioxidant enzyme activity. These findings are consistent with prior research, which identified oxidative stress and inflammation as important drivers of chemically induced hepatocarcinogenesis.

Treatment with plant extract restored hepatic enzyme levels to normal, reduced malondialdehyde (MDA), and increased antioxidant enzymes like superoxide dismutase (SOD) and catalase (CAT). These findings are consistent with previous research showing the antioxidant and hepatoprotective capabilities of *B. lanzan*and other phytochemical-rich plants (17).

Histopathological examination demonstrated that *B. lanzan*therapy reduced hepatic necrosis, fibrosis, and nodular lesions while retaining lobular architecture compared to HCC controls. Similar protective benefits have been observed with other medicinal plants high in flavonoids and phenolics, which lower oxidative stress, inflammation, and cellular damage in hepatocarcinogenesis (18).

*B. lanzan*chemopreventive efficacy is due to its varied phytochemical composition. Flavonoids and phenolics scavenge free radicals and prevent lipid peroxidation, whereas saponins stabilise hepatocyte membranes and tannins regulate inflammatory pathways (19,20). These bioactive substances may promote p53-mediated apoptosis and reduce NF-κB-induced inflammation, avoiding malignant transformation (21,22).

*B. lanzan* extract was found to be as effective in restoring liver function parameters and histological architecture as typical hepatoprotective drugs such as silymarin (23). Toxicological tests show a favourable safety profile, which supports its clinical applicability. Overall, the study supports the traditional usage of *B. lanzan* in liver problems and emphasises its potential as a functional food or phytotherapeutic agent for liver cancer prevention (24).

## Conclusion:

The current investigation shows that *B. lanzan* extract has strong hepatoprotective and chemopreventive effects against NDEA + $CCl_4$-induced hepatocellular cancer in rats. The extract improved blood liver function indices, decreased lipid peroxidation, increased antioxidant enzyme activities, and preserved hepatic histoarchitecture by reducing necrosis, fibrosis, and tumour formation. These therapeutic effects are attributable to *B. lanzan*abundant phytochemical composition, which includes flavonoids, phenolics, saponins, and tannins that work together to combat oxidative stress and inflammation. The findings support *B. lanzan's* traditional use in liver problems while also highlighting its potential as a functional food or phytotherapeutic agent for liver cancer prevention.Future research should focus on bioactive compound isolation, molecular mechanism elucidation, and clinical evaluation in order to advance its translational applicability.

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## Conflict of interest:

There are no conflicts of interest declare by authors in this manuscript.

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