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POTATO CHIPS INDUCED HISTOLOGICAL CHANGES IN THE LIVER OF WISTAR ALBINO RATS AND ITS MANAGEMENT WITH *BHRINGARAJA SWARASA*

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ABSTRACT

Background: The liver is the principal organ of metabolism, detoxification and biotransformation, and is highly vulnerable to dietary and oxidative insults. Rising consumption of processed, deep-fried potato chips, rich in oxidised fats, excess salt and acrylamide formed during frying, has been linked to hepatocellular injury. Many *Rasayana* drugs have hepatoprotective activity, one among them is *Bhringaraja* (*Eclipta alba*). **Objective**: To evaluate potato chips induced hepatotoxicity, with liver function test and liver histopathology and to assess the curative efficacy of *Bhringaraja Swarasa* in Wistar albino rats. **Methods**: Eighteen adult Wistar albino rats (weighing around 200–250 gm) were allotted to three groups of six each, i.e, Group I (control), Group II (disease group) and Group III (curative group). Liver function tests (LFTs) and liver histopathological test were performed at the end of each phase. **Results**: Group II showed marked elevation of Aspartate amino Transferase (AST), Alanine amino Transferase (ALT) and Alkaline Phosphatase (ALP), compared with control ($p < 0.001$), with histopathology showing hepatocyte ballooning, cytoplasmic vacuolation, sinusoidal congestion and inflammatory infiltration. Group III showed significant biochemical recovery and near-restoration of normal hepatic lobular architecture.

Conclusion: Chronic potato chips consumption produces measurable structural and biochemical hepatotoxicity in rats and *Bhringaraja Swarasa* demonstrates significant hepatoprotective and hepato-regenerative activity.

KEYWORDS: Potato chips, Wistar albino rats, acrylamide, hepatotoxicity, hepatoprotective, histopathology, *Bhringaraja* (*Eclipta alba).*

INTRODUCTION

The liver performs major role in carbohydrate and lipid metabolism, protein synthesis, bile formation and detoxification, and its structural integrity is a sensitive marker of systemic metabolic and toxic stress. Urbanisation and dietary transition have driven a sharp rise in consumption of processed potato chips, which is high in saturated and thermally oxidised fats, sodium and acrylamide, a reactive compound generated from asparagine and reducing sugars during high-temperature frying (the Maillard reaction). Acrylamide is bioactivated hepatically by cytochrome P450 2E1 (CYP2E1) to glycidamide, a genotoxic epoxide that forms DNA adducts and generates reactive oxygen species, driving lipid peroxidation, membrane injury and hepatocellular damage.

Bhringaraja (*Eclipta alba*) is one among the hepato-protective *Rasayana* drug. *Swarasa* is regarded as the most potent of the *Panchavidha Kashaya Kalpanas*. Modern phytochemical studies attribute hepatoprotective activity by coumestans (wedelolactone, demethylwedelolactone), flavonoids (luteolin, apigenin), triterpenoid saponins (eclalbasaponins) and phytosterols (β-sitosterol, stigmasterol), which act through antioxidant, anti-inflammatory, membrane-stabilising and hepato-regenerative mechanisms.

This study was designed to experimentally correlate the biochemical with histopathological consequences of potato chips induced hepatotoxicity and to evaluate whether *Bhringaraja Swarasa* can reverse these structural changes.

AIM: To evaluate the effect of potato chips on the liver and the therapeutic effect of *Bhringaraja Swarasa* in Wistar albino rats.

OBJECTIVES

- To identify the histopathological changes in the liver of Wistar albino rats following administration of potato chips.

- To assess the therapeutic role of *Bhringaraja Swarasa* on the liver of Wistar albino rats after potato chips administration.

MATERIALS AND METHODS

Experimental animals and housing

Eighteen healthy adult Wistar albino rats (200–250 g) were procured and housed in the institutional animal facility of Alva's ayurveda medical college, Moodubidire, under CPCSEA guidelines: polypropylene cages with stainless-steel grill tops, paddy-husk bedding changed daily, a 12-hour light/dark cycle, 50–70% relative humidity and 22 °C ambient temperature, with standard pelleted feed and water ad libitum.

Experimental design

Animals were randomly divided into three groups of six (n = 6 each):

- Group I (Control): normal diet and water only, 60 days.
- Group II (Disease): standard feed supplemented with 30% potato chips (~5 g/rat/day) for 30 days.
- Group III (Curative): the same potato-chip diet for 30 days, followed by *Bhringaraja Swarasa* for a further 30 days.

Bhringaraja Swarasa was prepared by the classical *Yantra-Nishpidana* method: fresh, authenticated *Eclipta alba* leaves were washed, pounded in a *khalva yantra* and expressed through cloth to obtain fresh juice.

Liver function tests

Blood was collected under diethyl ether anaesthesia from Group II on day 30 and from Groups I and III on day 60 and analysed for serum bilirubin (total, direct, indirect), AST, ALT, alkaline phosphatase, total protein, albumin, globulin and A/G ratio, using standard kinetic/end-point methods.

Histopathological processing

After sacrifice, the abdomen was opened by a mid-sagittal incision and the liver excised, fixed in 10% neutral-buffered formalin, and processed for paraffin embedding. Sections were stained with haematoxylin and eosin (H&E), later examined by light microscopy for architectural, cellular and vascular changes.

Statistical Analysis

Data are expressed as mean ± SD. Normality was assessed with the Shapiro–Wilk test; inter-group comparisons used one-way ANOVA with post-hoc pairwise tests, with $p < 0.05$ considered significant.

RESULTS

Liver function tests

Group II (Disease) animals showed a marked and statistically significant rise in serum AST, ALT and ALP compared with Group I (Control) ($p < 0.001$ for all pairwise comparisons), consistent with hepatocellular membrane injury and enzyme leakage. Total protein was mildly reduced and the A/G ratio elevated in Group II. Group III showed substantial, statistically significant recovery toward control values for AST, ALT and ALP following *Bhringaraja Swarasa* administration, while bilirubin fractions did not differ significantly between groups at any stage.

Table 1: Liver function parameters (mean ± SD, n = 6/group) across control, disease and curative groups.

Parameter	Control (Gr. I)	Disease (Gr. II)	Curative (Gr. III)
AST (IU/L)	38.7 ± 1.6	188.7 ± 8.3	110.8 ± 7.9
ALT (IU/L)	34.2 ± 3.1	76.7 ± 1.2	56.0 ± 2.3
ALP (IU/L)	119.5 ± 2.4	778.2 ± 60.8	432.7 ± 14.5
Total protein (g/dL)	6.9 ± 0.1	6.1 ± 0.1	6.6 ± 0.1
A/G ratio	0.8 ± 0.05	1.9 ± 0.25	0.8 ± 0.04

Histopathological findings

Histopathological examination is considered as standard method for correlating biochemical derangement with actual tissue injury. Light-microscopic (H&E) sections from all 18 animals were assessed for hepatocyte morphology, central-vein and sinusoidal architecture, cytoplasmic vacuolation, inflammatory infiltration and Kupffer-cell activity.

a) Control group (Group I)

Sections showed normal hepatic lobular architecture: Polygonal hepatocytes with centrally placed nuclei, radiating in cords around an intact, centrally located central vein lined by endothelium and containing red blood cells. Kupffer cells were visible, indicating normal reticuloendothelial activity. No necrosis, fatty change, cytoplasmic vacuolation or sinusoidal dilatation was seen, confirming preserved structural and functional integrity of hepatic tissue.

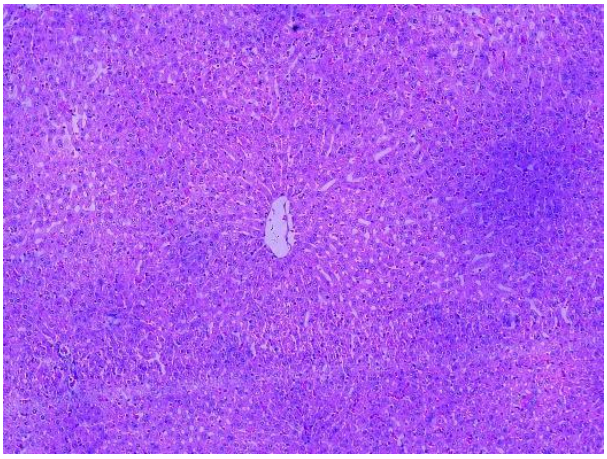


Fig. 1: Light micrograph of liver, Group I (control), showing normal hepatocyte architecture radiating around an intact central vein, with visible Kupffer cells and no evidence of necrosis, vacuolation or congestion (H&E, ×10).

b) Disease group (Group II)

Potato chips administration produced significant histopathological derangement. Numerous hepatocytes showed multiple small clear cytoplasmic vacuoles consistent with fatty change, alongside hepatocyte ballooning degeneration. Hepatic sinusoids were congested and dilated, with erythrocyte accumulation. There was mild to moderate inflammatory cell infiltration with prominent Kupffer-cell activation. These findings are attributable to acrylamide and oxidised lipid driven oxidative stress, impaired β-oxidation and excess triglyceride re-esterification within hepatocytes, and directly parallel the sharp rise in serum AST, ALT and ALP observed biochemically in this group.

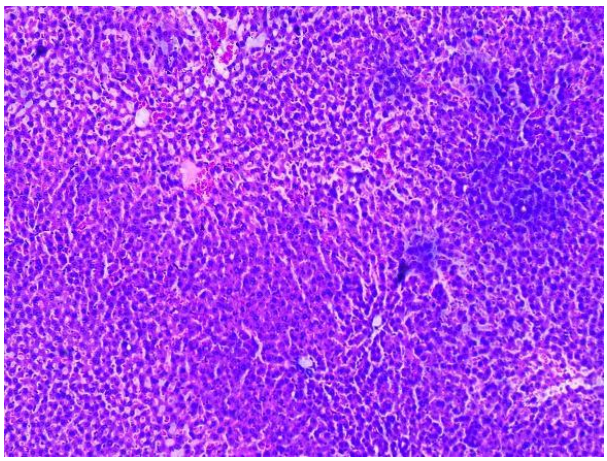


Fig. 2: Light micrograph of liver, Group II (disease), showing hepatocellular vacuolation/fatty change, sinusoidal congestion and inflammatory infiltration following 30 days of potato-chip administration (H&E, ×40).

c) Curative group (Group III)

Following 30 days of *Bhringaraja Swarasa* administration, hepatic sections showed marked structural recovery: mild residual cytoplasmic vacuolation, near-normal sinusoidal architecture and hepatocellular plates realigned in a near-normal radiating pattern around the central vein, with reduced inflammatory infiltration compared with Group II. These changes indicate active tissue remodelling and regeneration, corresponding with the biochemical normalisation of AST, ALT and ALP in this group.

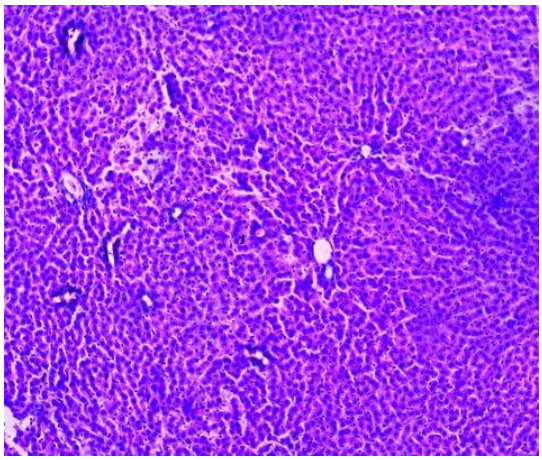


Fig. 3: Light micrograph of liver, Group III (curative), showing restoration of hepatic lobular architecture with mild residual vacuolation and reduced inflammation after *Bhringaraja Swarasa* therapy (H&E, ×10).

Overall, the histological findings showed, normal architecture in control group, marked fatty/inflammatory injury in disease group and substantial architectural restitution in curative group which closely mirrored the biochemical liver-enzyme profile, reinforcing histopathology as the most direct structural evidence of both hepatotoxicity and hepatoprotection in this model.

Body weight and liver weight

Group II & III showed a significantly greater increase in body weight during the potato chips administration period than Group I, consistent with the known obesogenic effect of energy dense, palatable processed snacks. Liver weight was correspondingly increased in Group II, reflecting lipid accumulation and fatty infiltration. Group III showed partial normalisation of liver weight following treatment.

DISCUSSION

Deep-frying of carbohydrate-rich foods such as potatoes generates acrylamide via the Maillard reaction. Hepatic CYP2E1 gene converts acrylamide to glycidamide, its principal genotoxic metabolite, which forms DNA adducts and generates reactive oxygen species that overwhelm endogenous antioxidant defences (superoxide dismutase, catalase, glutathione peroxidase). The resulting oxidative stress drives lipid peroxidation of hepatocyte membranes, increasing permeability and enzyme leakage, explaining the sharp rise in serum AST and ALT observed in Group II. Concurrently, the substantial fat load in potato chips (30–50% by weight, including thermally oxidised polyunsaturated fatty acids) exceeds hepatic β-oxidation and VLDL-export capacity, driving triglyceride re-esterification and the fatty-change/ballooning degeneration.

The histological gradient across groups are, intact architecture in Group I, fatty degeneration with sinusoidal congestion and inflammatory infiltration in Group II and substantial restitution in Group III, is consistent with reports of acrylamide-associated hepatocellular degeneration, sinusoidal congestion and cytoplasmic vacuolation in comparable rodent models and directly corroborates the biochemical enzyme profile in each group.

The hepatoprotective effect of *Bhringaraja Swarasa* in Group III is seen. Its flavonoid fraction (luteolin, apigenin) scavenges reactive oxygen species and restores intracellular glutathione, the principal substrate for detoxifying acrylamide and glycidamide. Wedelolactone inhibits NF-κB signalling in Kupffer cells and hepatocytes, reducing pro-inflammatory cytokine production (TNF-α, IL-1β, IL-6) and thereby the inflammatory infiltration seen on histology. Phytosterols such as β-sitosterol and stigmasterol intercalate into hepatocyte membranes, reducing enzyme leakage, while the polyphenolic fraction suppresses hepatic stellate-cell activation, limiting fibrotic progression and supporting hepatocyte regeneration. These convergent antioxidant, anti-inflammatory, membrane-stabilising and regenerative mechanisms together explain the near-normalisation of both liver enzymes and hepatic histoarchitecture observed in the curative group.

Excessive intake of *Ati Snigdha*, *Lavana* and *Vidahi Ahara* is understood to aggravate *Pitta-Kapha Dosha* and impair *Agni*, leading to *Ama* accumulation. That affects *Rakta Dhatu* and *Yakrit*, findings that parallel the structural and functional hepatic derangement observed here. *Bhringaraja Swarasa*, by virtue of its *Pitta shamaka* property, is understood to correct this vitiation, as observed here in biochemical and histopathological restorative effect.

CONCLUSION

Chronic administration of potato chips produced significant hepatotoxicity in Wistar albino rats, evidenced biochemically by elevated AST, ALT and ALP, and structurally by hepatocellular degeneration, fatty change, sinusoidal dilatation and inflammatory infiltration on histopathology. *Bhringaraja Swarasa* produced marked biochemical recovery and substantial restoration of normal hepatic architecture, supporting its classical reference as a hepatoprotective drug. These findings validate dietary hepatotoxicity and hepatoprotective activity of *Bhringaraja Swarasa* in the management of diet-induced liver disorders.

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