© JUN 2026 | IRE Journals | Volume 9 Issue 12 | ISSN: 2456-8880
DOI: https://doi.org/10.64388/IREV9I12-1719200

Investigation Of the Toxic Effects of Rauvofia Vomitoria (Apocynacae) Alkaloid-Rich Extract in Rattus Norvegicus

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Abstract- ***Rauvolfia vomitoria is one of the plant sources of medicines, known for its peculiar rich in alkaloids. Its therapeutic potential had been widely studied. The toxicological profile of its alkaloid extracts remains sketchy. This study investigated the toxic potential of Rauvolfia vomitoria alkaloid extracts in Wistar rats. Fresh leaves of Rauvolfia vomitoria were carefully processed in methanol using Soxhlet extractor The extract was administered orally to four groups of Wistar rats (n=5 per group) at doses of 0 (control), 100, 200, and 400 mg/kg body weight daily for 14 days. Standard protocol assays were used to assess liver and kidney function markers. The alkaloid extract of Rauvolfia vomitoria caused dose-dependent effects on liver and kidney tissues in Wistar rats. At lower doses (1500 mg/kg), the extract was relatively well tolerated, causing only mild biochemical changes. However, at higher doses (4000–7000 mg/kg), the extract led to significant alterations in biochemical parameters, indicating liver and kidney toxicity. The alkaloid extract of Rauvolfia vomitoria caused dose-dependent effects on liver and kidney tissues in Wistar rats. At lower doses (1500 mg/kg), the extract was relatively well tolerated, causing only mild biochemical changes. However, at higher doses (4000–7000 mg/kg), the extract led to significant alterations in biochemical parameters, indicating liver and kidney toxicity. These findings suggested that while Rauvolfia vomitoria alkaloid extract may have potential medicinal value, it is toxic at high doses.***

Keywords: ***Therapeutic, Alkalois, Liver, Kidney, Toxicity, Soxhlet***

I. INTRODUCTION

The use of plants in medicines across globe is visibly gaining prominence. Rauvolfia vomitoria (Apocynaceae), commonly known as African serpent wood, has been extensively utilized for the management of hypertension, insomnia, mental disorders, and gastrointestinal ailments (Kumar, 2022).

The pharmacological relevance of R. vomitoria lies mainly in its richness of indole alkaloids particularly reserpine, ajmaline, serpentine, and deserpidine (Kumar, 2022; Ekong, 2024). Despite these acclaimed therapeutic applications, growing evidence suggests that the same bioactive alkaloids responsible for its pharmacological benefits may also induce toxic effects, especially at high doses or upon prolonged exposure (Bonheur, 2020; Adomefa et al., 2024).

In recent years, the scientific community has turned increasing attention toward systematically evaluating the safety profile of R. vomitoria, especially through controlled animal studies. Wistar rats have been widely employed in these investigations because of their physiological and metabolic similarities to humans in toxicological responses (Bonheur, 2020; UBJST, 2024). Studies in the pasts have revealed that extracts of R. vomitoria, ingested through any route can produce varied biochemical and histological alterations (Bonheur, 2020).

II. RESEARCH ELABORATION

Aim: To investigate the potential toxic effects of alkaloid-rich extract of Rauvolfia vomitoria on biochemical, hematological in Wistar rats.

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Materials and methods

Rauvolfia vomitoria leaves

Alkaloid extraction

150g of powdered leaves was packed into a cellulose thimble. The extraction was carried out in batches, and cotton wool was placed on top of each thimble to prevent sample loss. The cellulose thimble containing the plant sample was inserted into the Soxhlet extractor.

Qualitative Test for Alkaloids

Mayer's reagent Test

In a clean test tube, 2 ml of alkaloids extract was taken, and mixed with a few drops of Mayer's reagent. Formation of pale-yellow precipitate. indicated presence of alkaloids

Twenty-four male animals weighing averagely 150g were grouped into four stages. All the animals were acquainted with new conditions for about one week. Groups I, II, III and IV were administered orally with 1500, 2400, 4000 and 7000mg/kg body weight single dose of the test extract in that order and left for about twenty-four hours.

Biochemical Assays

Fresh kidneys and livers harvested from the test and control rats were rinsed in cold normal saline to remove blood stains and debris, homogenized in cold phosphate buffer, pH 7.4, using a homogenizer Silver Crest on ice; the homogenate is centrifuged at 10,000 rpm for 15–30 minutes at 4°C, and the clear supernatant was collected for GGT, ALT, AST, and CYP-450. GGT assays

ALT (Alanine Aminotransferese) measurement (Arthur-Karmen, 1964)

A substrate reaction mixture consisting of phosphate buffer, L-alanine, and α-ketoglutarate was prepared and pre-warmed to 37°C. Aliquots of the tissue supernatant were transferred into clean test tubes, and the reaction was initiated by adding the pre-warmed substrate mixture. The tubes were incubated at 37°C for a fixed period of 20 minutes to allow pyruvate formation.

At the end of the incubation period, DNPH reagent was added to stop the reaction and to allow the formation of the pyruvate hydrazone complex. The tubes were kept at room temperature for 5–10 minutes, after which sodium hydroxide was added to develop the final colour. The absorbance of each sample was then measured at 505 nm using a uv-visible spectrophotometer, with a reagent blank serving as the reference.

Pyruvate standard solutions were processed in the same manner as the samples and were used to generate a standard calibration curve. The ALT activity in each tissue sample was subsequently calculated by converting the absorbance values to micromoles of pyruvate formed and expressing the enzyme activity as units per gram of tissue, taking into account the incubation time and dilution factors.

All reagents and tissue samples were kept cold until the start of the incubation, and appropriate blanks and heat-inactivated controls were included to correct for non-enzymatic pyruvate formation.

AST (Aspartate Aminotransferase) assay (Arthur-Karmen, 1964)

The substrate reaction mixture, which consisted of phosphate buffer containing L-aspartate and α-ketoglutarate, was prepared freshly and pre-warmed to 37°C.

Aliquots of the tissue supernatants were transferred into labeled test tubes, and the enzymatic reaction was initiated by the addition of the pre-warmed substrate mixture. The tubes were incubated at 37°C for 10–30 minutes to allow the formation of oxaloacetate.

At the end of the incubation period, DNPH solution was added to each tube to stop the reaction and permit hydrazone formation. The tubes were allowed to stand at room temperature for 5–10 minutes, after which sodium hydroxide was added to develop the final coloured complex. The absorbance of each reaction mixture was measured at 505 nm using uv-visible spectrophotometer.

Standard solutions were processed in the same manner as the samples and were used to construct a calibration curve. Because oxaloacetate is unstable, pyruvate standards were used as a validated alternative. AST activity in the tissue samples was calculated by converting absorbance values into micromoles of product formed and expressing the enzyme activity as units per gram of tissue

ALP (Alkaline Phosphatase) (Bessey, Lowry and Brock, 1946).
Freshly prepared mixture of 10 mM p-nitrophenyl phosphate in diethanolamine (DEA) buffer (pH 10.4) and 1 mM $MgCl_2$, was warmed to 37°C. The tissue supernatants were transferred into clean test tubes, and the enzymatic reaction was initiated by adding the warmed para-nitrophenylphosphate solution to give a final reaction volume of 1 ml. The tubes were incubated at 37°C for 15 minutes. Then, the reaction was terminated by adding 0.5 M sodium hydroxide.

Standard pNP solutions were also processed in the same way to make a calibration curve. ALP activity was expressed as units per gram of tissue. The absorbance of each reaction mixture was read at 405 nm using a uv-visible spectrophotometer.

GGT (Gamma-Glutamyl Transferase) (Ornowski and Meister, 1970)
A reaction mixture of γ-glutamyl p-nitroanilide and tissue supernatant was made and allowed to proceed at 37°C. After a fixed incubation period, the reaction was stopped by adding acetic acid. The intensity of the yellow p-nitroaniline was then measured spectrophotometrically at 405 nm.

The absorbance obtained was proportional to the enzyme activity.
Albumin estimation (Mulder, 1880)
1mg/ml albumin standard was used to make serial dilution Tissue supernatants were pipetted into labeled test tubes. 1ml Mulder reagent was added to each tube.

The reaction mixtures were mixed thoroughly and allowed to stand at room temperature for 10 minutes. A reagent blank containing buffer and dye but no tissue sample was included.

The absorbance of each sample and standards was measured at 620 nm using a uv-visible spectrophotometer. The albumin concentration in each tissue sample was determined from the standard curve. Results were expressed as mg albumin per gram of tissue.

Bilirubin (Ban-den Bergh and Muller, 1916).
Tissue supernatants were pipetted into labeled test tubes. was added directly to the sample. The tissue supernatant was mixed with alcohol before adding Van den Bergh reagent. The reaction mixtures were incubated at room temperature for 10 minutes. A series of bilirubin standards were prepared and treated in the same manner as the tissue samples. A blank containing buffer and diazotized reagent was included.

The absorbance of the samples and standards was read at 540 nm over uv-visible spectrophotometer. Bilirubin concentrations in the tissue samples were calculated from the standard curve, taking into account the dilution factor of the homogenate, and expressed as mg bilirubin per gram of tissue.

III. RESULTS AND DISCUSSION

Oral Doses 1500, 2400, 4000 and 7000 mg/kg body weight for averagely weighed rats of 150g.

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Table 1: Rat kidney's biomarkers following acute toxicity.

Dose	Bilirubin(U/mg protein) $x10^{-2}$	Albumin(U/mg protein) $x10^{-3}$	GGT(U/mg protein) $x10^{-3}$	ALT(U/L) $x10^{-3}$	AST(U/L) $x10^{-3}$
Control	18.1818±8.50	152.924±7.516***	3.4206±0.263	139.605±12.06	102.105±5.513
D_I	21.1212±5.28	101.771±7.264*	2.4708±0.123	144.0789±1.674	113.4211±16.27
D_{II}	18.1818±8.07	60.749±15.648	3.5874±2.621	149.4737±11.477	114.605±2.046
D_{III}	24.2424±8.59	56.884±3.48****	3.6559±0.946	150.2632±6.620	128.947±5.0
D_{IV}	24.24±9.741	44.982±39.23	3.1339±0.364	158.552±2.591	137.76±7.257

Data are presented as mean±SEM (n=5), P-value<0.05 were considered significant.

Oral Doses 1500, 2400, 4000 and 7000 mg/kg body weight for averagely weighed rats of 150g.

Table 2: Rat liver's biomarkers following acute toxicity

Dose	Bilirubin (U/mg protein $\times10^{-2}$)	Albumin (U/mg protein $\times10^{-3}$)	GGT (U/mg protein $\times10^{-3}$)	ALT (U/L $\times10^{-3}$)	AST (U/L $\times10^{-3}$)	CYP-450 (pg/mL $\times10^{-3}$)
Control	21.2523 ± 0.4*	454.166 ± 35.3	133.4588 ± 0.7	65.78948 ± 24.9	113.4616 ± 5.4	78.12048 ± 5.1
DI	73.7402 ± 9.7**	166.666 ± 5.8*	127.9433 ± 0.4	92.85716 ± 11.1	149.0385 ± 93.8	71.53614 ± 0.7
DII	112.9457 ± 45.2*	71.3333 ± 0.7	120.7720 ± 0.6	92.48121 ± 8.5	360.5769 ± 28.5*	68.51206 ± 5.1**
DIII	297.7615 ± 9.4*	56.884 ± 3.48****	126.1505 ± 1.3	115.0376 ± 26.5	275.9616 ± 178.1	66.9006 ± 0.2
DIV	429.2711 ± 10.1*	45.833 ± 41.2***	128.4323 ± 0.6	103.7594 ± 1.06	391.3462 ± 53.0*	53.51205 ± 0.2

Data are presented as mean±SEM (n=5), P-value<0.05 were considered significant

IV. DISCUSSION

This work demonstrated a clear dose-dependent hepatic and renal toxicity associated with the acute oral administration of Rauvolfia vomitoria alkaloid-rich extract in Wistar rats. Marked elevations in serum alanine aminotransferase (ALT) and aspartate aminotransferase (AST) were observed across all treatment groups, with pronounced increases at 4000 and 7000 mg/kg. These enzymes are localized predominantly within hepatocytes; therefore, their increased serum concentrations indicated compromised hepatocellular integrity.

The hepatotoxic response at higher doses was likely attributable to saturation of hepatic detoxification pathways—particularly the cytochrome P450 (CYP-450) enzymatic system—resulting in oxidative stress, membrane lipid peroxidation, mitochondrial impairment, and subsequent cellular disruption.

Significant elevations in total bilirubin at the higher dose levels (DIII and DIV) suggested perturbations in bilirubin metabolism and excretory function. This impairment may be associated with oxidative injury to hepatocytes, inhibition of bilirubin conjugation, or interference with bile flow.

The data further implicated potential suppression or dysfunction of key bile transporters such as multidrug resistance-associated protein 2 (MRP2) and the bile

salt export pump (BSEP), which would account for the retention of conjugated bilirubin within hepatic tissue and systemic circulation.

A significant decline in serum albumin concentrations was recorded in all extract-treated groups relative to controls. This reduction reflected impaired hepatic protein synthetic capacity and is indicative of emerging liver dysfunction, particularly at the higher doses. Additionally, CYP-450 activity showed a marked dose-dependent decline, suggesting either direct inhibition by the extract's alkaloid constituents or structural injury to the hepatocytes responsible for enzyme synthesis. Such reductions may represent an early sign of necrotic or irreversible hepatic damage.

Renal biochemical indices also revealed dose-dependent alterations. Although elevations in ALT and AST within renal tissue were less pronounced than in the liver, their increase indicated mild nephrotoxicity, likely mediated through oxidative stress or mitochondrial compromise in renal cells.

In contrast, renal bilirubin levels remained relatively stable across groups, suggesting preserved bilirubin clearance and minimal impact on renal excretory pathways. This stability may reflect adaptive physiological responses or sub-threshold oxidative stress that did not extensively impair renal function.

Decreases in renal-associated albumin levels in the high-dose groups implied structural or functional compromise of the glomerular filtration barrier, potentially due to oxidative or inflammatory injury. Such damage would increase membrane permeability, promoting albumin loss into the urine. Tubular dysfunction might further contribute to reduced albumin reabsorption, compounding the observed decreases.

Acute exposure to Rauvolfia vomitoria alkaloid extract produced significant, dose-related adverse effects on hepatic and renal function. While lower doses (1500–2400 mg/kg) elicited only mild biochemical changes, higher doses (4000–7000 mg/kg) resulted in pronounced hepatocellular injury, reduced albumin synthesis, and evidence of renal involvement.

V. CHAPTER FIVE CONCULSION AND RECOMMENDATION

Conclusion

The alkaloid extract of Rauvolfia vomitoria caused dose-dependent effects on liver and kidney tissues in Wistar rats. At lower doses (1500 mg/kg), the extract was relatively well tolerated, causing only mild biochemical changes. However, at higher doses (4000–7000 mg/kg), the extract led to significant alterations in biochemical parameters, indicating liver and kidney toxicity.These findings suggested that while Rauvolfia vomitoria alkaloid extract might have potential medicinal value, its toxicity at high doses highlights the need for caution.

Safe use is more likely at low and controlled doses, but excessive or prolonged intake may pose health risks. The findings indicated a maximum non-lethal dose of approximately 1500 mg/kg, whereas mortality was observed from 2400 mg/kg onward.

These results underscored the necessity of rigorous dose optimization and safety evaluation prior to considering the therapeutic application of Rauvolfia vomitoria extracts.

RECOMMENDATION

Further studies should aim to isolate and characterize the specific alkaloids responsible for the observed effects.

Toxicokinetic and mechanistic studies are needed to understand how these alkaloids are metabolized and eliminated from the body.

The safe therapeutic dose range should be clearly established through standardized testing.

Prolonged or unsupervised use of the extract should be discouraged until more safety data is available
Standardized dosage formulations should be developed for any potential medicinal application.

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