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Evaluating Hedyotis aspera's Anti-Diabetic Efficacy: In Vivo Insights

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Abstract

Background: Diabetes mellitus is a chronic metabolic disorder characterized by hyperglycemia due to impaired insulin secretion and/or insulin resistance. Herbal medicines are gaining importance due to their multi-targeted mechanisms and safety profile. *Hedyotis aspera* is a medicinal plant known for its antioxidant and pharmacological activities.

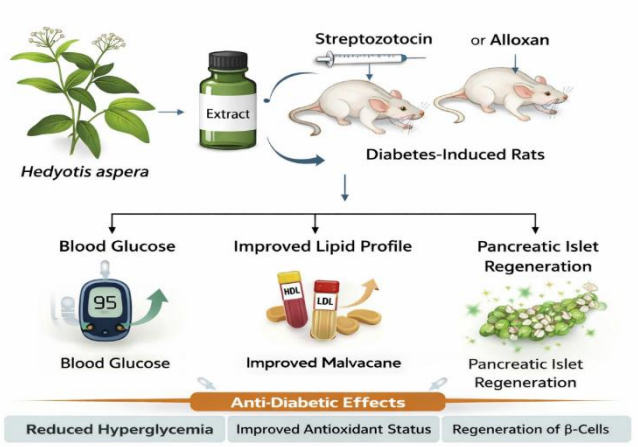
Objective: To evaluate the anti-diabetic potential of *Hedyotis aspera* in an *in vivo* experimental model.

Methods: Diabetes was induced in Wistar rats using alloxan. Animals were treated with *Hedyotis aspera* extract at different doses of 200 & 400mg/kg p.o,. Blood glucose levels, biochemical parameters were assessed.

Results: Treatment with *Hedyotis aspera* significantly reduced blood glucose levels, improved lipid profile, enhanced antioxidant status, and restored pancreatic architecture.

Conclusion: *Hedyotis aspera* exhibits significant anti-diabetic activity and may serve as a potential therapeutic agent for diabetes management.

Keywords: Diabetes mellitus, *Hedyotis aspera*, alloxan, Antioxidant, *In vivo*



1. Introduction

Diabetes mellitus is a major global health problem associated with complications such as neuropathy, nephropathy, and cardiovascular diseases. It is characterized by chronic hyperglycemia resulting from insulin deficiency or resistance. Diabetes is in the top 10, and

perhaps the top 5, of the most significant diseases in the developed world. The top 10 countries, in numbers of sufferers, are India, China, USA, Indonesia, Japan, Pakistan, Russia, Brazil, Italy and Bangladesh (World Health Organization, 2000). According to the World Health Organization (WHO), at least 171 million people worldwide suffer from diabetes in 2000. Its incidence is increasing rapidly, and it is estimated that by the year 2030, this number will double.

Experimental models such as streptozotocin (STZ)-induced diabetes are widely used to study anti-diabetic agents. STZ selectively destroys pancreatic β-cells, leading to insulin deficiency and hyperglycemia.

Oxidative stress plays a significant role in the pathogenesis of diabetes and its complications. Herbal drugs rich in antioxidants can protect β-cells and improve glycemic control.

Hedyotis aspera (family Rubiaceae) contains flavonoids, alkaloids, and phenolic compounds known for antioxidant and anti-inflammatory properties. It claimed to support the immune system and maintain body's natural balance and general well- being. Other medicine ulcerations, swelling, snakebite, bronchitis, tonsilitis and haemorrhoid Studies on related species such as *Hedyotis corymbosa* have shown promising anti-diabetic and hepatoprotective effects.

2. Materials and Methods

2.1 Collection of plant materials and authentication

Hedyotis aspera (Heyne ex Roth) were collected from P.S.C & K.V.S.C Govt college, Nandyal, India. Identified by plant taxonomist prof. S.V. Sailajarani, plant taxonomist, Department of Botany, was submitted in the concerned herbarium for future reference.

2.2 Preparation of Methanol extract of *Hedyotis aspera* (MEHA)

The entire plant was exposed to N-Hexane extraction, which was utilized to defatted for at least one day. The obtained defatted material was extracted using a maceration process with 95% methanol and water (a ratio of 3:1) for at least a day. Hydro alcoholic extraction. The entire or coarsely ground plant material is put through this process in a sealed container with the solvent and allowed to stand at room temperature. The combination is then strained, resulting in the combined liquids become clearer and the water begins evaporating. Then thereby produced plant extract was stored in a desiccator and used for additional research. The nature of substance is sticky and color is dark green.

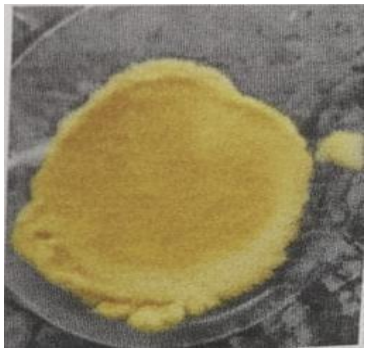
2.3 Preliminary Phytochemical screening (HARBOURNE J *et al*., 1973)

Table no: 1 Preliminary Phytochemical screening

S.NO	Phytochemical test	Indication	Present (+) or Absent (-)
1	Test for alkaloids: Wagner test	Appearance of reddish-brown precipitation	+
2	Test for flavonoids	Appearance of intense yellow color	+
3	Test for Terpenoids	Appearance of reddish-brown coloration	+
4	Test for Tannins	Appearance of a blue -black	+

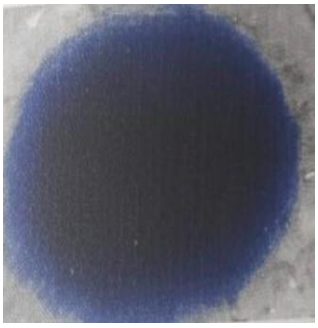
	&Phenols	precipitation	
5	Test for Cardiac glycoside	Appearance of a brown ring	+
6	Test for Saponins	Appearance of froth	+

Alkaloids

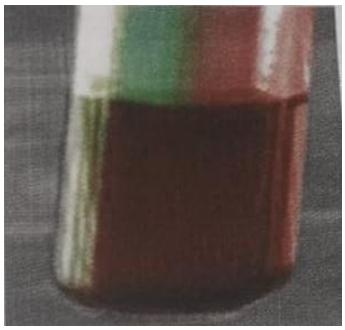


Flavonoids

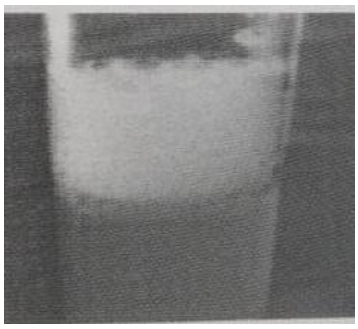
Terpenoids



Tannins & Phenols



Cardiac glycoside



Saponins

2.4 Experimental Animals

Male Wistar albino rats weighing 150-200 g were used in the study. They were housed in individual polypropylene cages under standard laboratory conditions of light, temperature and relative humidity. Animals were given standard rat pellets (Gold Mohor Ltd) and drinking water *ad libitum*.

2.5 Experimental Design

1. Table no: 2

Group No.	Treatment	Purpose	Days
I	No treatment	To serve as normal control	14
II	Alloxan (150mg/kg i.p) + Distilled water	To serve as disease control	14
III	Alloxan (150mg/kg i.p)+ Glibenclamide (0.5 mg/kg, p.o)	To serve as standard	14
IV	Alloxan(150mg/kg i.p)+ MEHA (200mg/kg, p.o)	To study the effect of MEHA on carbohydrate and lipid profile	14

V	Alloxan (150mg/kg i.p)+ MEHA (400mg/kg, p.o)	To study the effect of MEHA on carbohydrate and lipid profile	14

2.6 Biochemical Analysis

- Blood glucose levels
- AST &ALT levels
- Lipid profile (cholesterol, triglycerides, HDL,LDL)

3. Results

3.1 Blood Glucose Levels

Table no: 3 Effect of MEHA on serum glucose levels

Group	Treatment	Serum glucose (mg /dL) (Mean ± SEM) on		
		0 day	7th day	14th day
I	Normal	66.30 ± 3.361	68.83 ±4.474	66.49 ± 5.586
II	Alloxan (150mg/kg i.p) + Distilled water	177.93 ± 15.59[a]	186.66 ± 9.744[a]	210.44 ± 12.24[a]
III	Alloxan (150mg/kg i.p)+Glibenclamide (0.5 mg/kg, p.o)	240.47 ± 15.54	104.01 ± 11.45[b]	90.83 ±4.447[b]
IV	Alloxan(150mg/kg i.p)+ MEHA (200mg/kg, p.o)	408.72 ±21.11	391.94 ± 20.92[b]	103.5 ± 10.927[b]
V	Alloxan (150mg/kg i.p)+ MEHA (400mg/kg, p.o)	224.11 ±17.71	125.44 ± 4.369[b]	71.2 ± 7.907[b]

a = P<0.001 when compared to normal group (G-I)

b = P<0.001 when compared to diabetic control group (G-II)

Group I (Normal): Stable glucose levels across all days (~66–69 mg/dL).

Group II (Diabetic Control): Significant hyperglycemia maintained and worsened over time (177 → 210 mg/dL).

Group III (Glibenclamide): Strong hypoglycemic effect, reducing glucose from 240 mg/dL to ~91 mg/dL by day 14.

Group IV (MEHA 200 mg/kg): Initially very high glucose (409 mg/dL), but dramatic reduction to ~103 mg/dL by day 14.

Group V (MEHA 400 mg/kg): Moderate starting glucose (224 mg/dL), reduced to ~71 mg/dL by day 14, showing **better efficacy than Glibenclamide**.

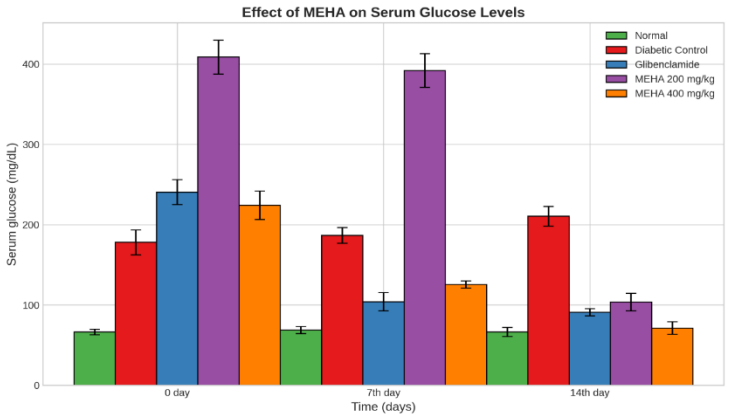


Fig no: 1 Effect of MEHA on serum glucose levels

3.2 Effect on serum triglycerides

Table no: 4 Effect of MEHA on serum triglyceride levels

Group	Treatment	Serum triglyceride (mg /dL) (Mean ± SEM) on	
		7th day	14th day
I	Normal	35.88 ±3.319	36.92 ± 3.363
II	Alloxan (150mg/kg i.p) + Distilled water	112.94 ±10.73[a]	127.99 ± 13.82[a]
III	Alloxan (150mg/kg i.p)+Glibenclamide (0.5 mg/kg, p.o)	60.89 ± 4.419[c]	51.33 ± 2.280[c]
IV	Alloxan(150mg/kg i.p)+ MEHA (200mg/kg, p.o)	81.56 ± 2.554[b]	68.23 ± 3.007[c]
V	Alloxan (150mg/kg i.p)+ MEHA (400mg/kg, p.o)	85.28 ±2.009[b]	70.58 ± 2.457[c]

a= P<0.001 when compared to normal group (G-I)

b= P<0.05 when compared to diabetic control group (G-II)

c= P<0.001 when compared to diabetic control group (G-II)

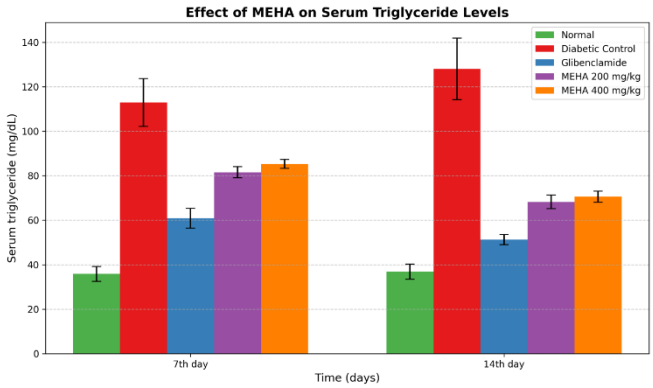


Fig no: 2 Effect of MEHA on serum triglyceride levels

Group I (Normal): Stable triglyceride levels (~36 mg/dL).

Group II (Diabetic Control): Markedly elevated triglycerides (113 → 128 mg/dL), confirming hyperlipidemia.

Group III (Glibenclamide): Significant reduction (61 → 51 mg/dL), approaching normal values.

Group IV (MEHA 200 mg/kg): Moderate reduction (82 → 68 mg/dL), statistically significant ($P < 0.05$ at day 7, $P < 0.001$ at day 14).

Group V (MEHA 400 mg/kg): Stronger reduction (85 → 71 mg/dL), also highly significant ($P < 0.001$ at day 14).

3.3 Effect on serum cholesterol

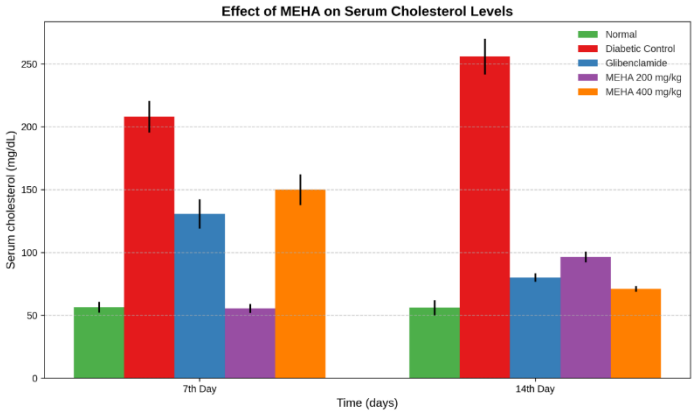
Table no: 5 Effect of MEHA on serum cholesterol levels

Group	Treatment	Serum cholesterol (mg /dL) (Mean ± SEM) on	
		7th day	14th day
I	Normal	56.41 ±4.322	55.99 ±5.984
II	Alloxan (150mg/kg i.p) + Distilled water	207.96 ±12.56[a]	255.79 ± 14.27[a]
III	Alloxan (150mg/kg i.p)+Glibenclamide (0.5 mg/kg, p.o)	130.63 ± 11.56[c]	80.06 ± 3.354[c]
IV	Alloxan(150mg/kg i.p)+ MEHA (200mg/kg, p.o)	55.43 ± 3.576[c]	96.38 ± 4.228[c]
V	Alloxan (150mg/kg i.p)+ MEHA (400mg/kg, p.o)	149.99 ±12.15[b]	71.01 ± 2.277[c]

a= P<0.001 when compared to normal group (G-I)

b= P<0.01 when compared to diabetic control group (G-II)

c = P<0.001 when compared to diabetic control group (G-II)



- **Group I (Normal):** Stable cholesterol (~56 mg/dL).
- **Group II (Diabetic Control):** Markedly elevated cholesterol (208 → 256 mg/dL), confirming hypercholesterolemia.
- **Group III (Glibenclamide):** Significant reduction (131 → 80 mg/dL), showing strong antihypercholesterolemic effect.

- **Group IV (MEHA 200 mg/kg):** Near-normal cholesterol at day 7 (55 mg/dL), but slightly higher at day 14 (96 mg/dL).
- **Group V (MEHA 400 mg/kg):** Elevated at day 7 (150 mg/dL), but reduced to 71 mg/dL by day 14, showing **better efficacy than Glibenclamide** at the later stage.

3.4 Effect on serum HDL-cholesterol

Table no: 6 Effect of MEHA on serum HDL-cholesterol

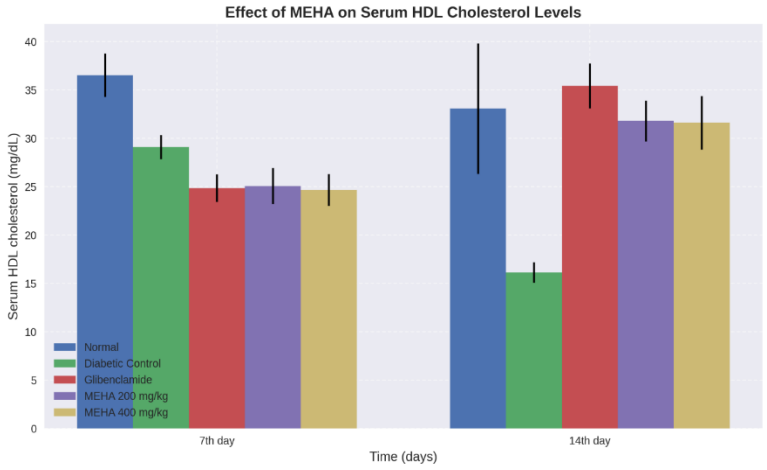
Group	Treatment	Serum HDL cholesterol (mg /dL) (Mean ± SEM) on	
		7th day	14th day
I	Normal	36.52 ± 2.235	33.07 ± 6.739
II	Alloxan (150mg/kg i.p) + Distilled water	29.09 ± 1.239[a]	16.14 ± 1.059[a]
III	Alloxan (150mg/kg i.p)+Glibenclamide (0.5 mg/kg, p.o)	24.85 ± 1.416[c]	35.42 ± 2.313[d]
IV	Alloxan(150mg/kg i.p)+ MEHA (200mg/kg, p.o)	25.07 ± 1.867[d]	31.79 ± 2.120[d]
V	Alloxan (150mg/kg i.p)+ MEHA (400mg/kg, p.o)	24.65 ± 1.643[c]	31.61 ± 2.755[d]

a = P<0.05 when compared to normal group (G-I)

b = P<0.001 when compared to normal group (G-I)

c = P<0.05 when compared to diabetic control group (G-II)

d= P<0.001 when compared to diabetic control group (G-II)



Group I (Normal): HDL levels remain stable (~33–36 mg/dL).

Group II (Diabetic Control): Significant reduction in HDL (29 → 16 mg/dL), confirming diabetic dyslipidemia.

Group III (Glibenclamide): Slightly lower HDL at day 7 (25 mg/dL), but strong recovery by day 14 (35 mg/dL), surpassing normal values.

Group IV (MEHA 200 mg/kg): Moderate improvement (25 → 32 mg/dL), showing significant restoration of HDL.

Group V (MEHA 400 mg/kg): Similar trend (25 → 32 mg/dL), with strong recovery by day 14.

3.5 Effect on serum LDL- cholesterol

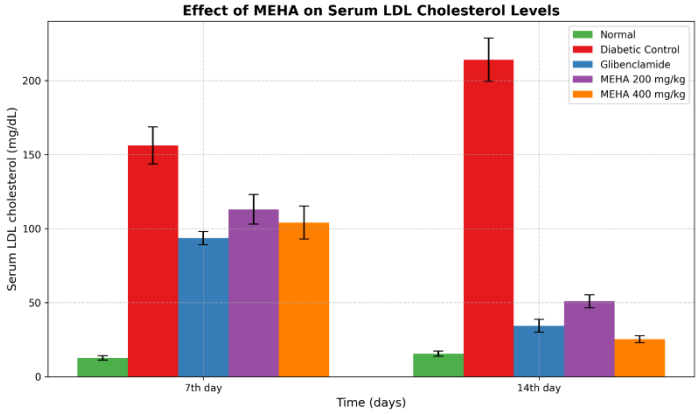
Table no: 7 Effect of MEHA on serum LDL- cholesterol

Group	Treatment	Serum LDL cholesterol (mg /dL) (Mean ± SEM) on	
		7th day	14th day
I	Normal	12.71 ± 1.475	15.54 ± 1.769
II	Alloxan (150mg/kg i.p) + Distilled water	156.27 ± 12.50[a]	214.05 ± 14.54[a]
III	Alloxan(150mg/kgi.p)+Glibenclamide (0.5 mg/kg, p.o)	93.6 ± 4.451[c]	34.37 ± 4.450[c]
IV	Alloxan(150mg/kg i.p)+ MEHA (200mg/kg, p.o)	113.04 ±10.07[b]	50.93 ±4.375[c]
V	Alloxan (150mg/kg i.p)+ MEHA (400mg/kg, p.o)	104.08 ± 11.14[b]	25.27 ± 2.366[c]

a= P<0.001 when compared to normal group (G-I)

b= P<0.05 when compared to diabetic control group (G-II)

c= P<0.001 when compared to diabetic control group (G-II)



Group I (Normal): LDL levels remain low and stable (~13–16 mg/dL).

Group II (Diabetic Control): Markedly elevated LDL (156 → 214 mg/dL), confirming diabetic dyslipidemia.

Group III (Glibenclamide): Strong reduction (94 → 34 mg/dL), showing potent LDL-lowering effect.

Group IV (MEHA 200 mg/kg): Moderate reduction (113 → 51 mg/dL), statistically significant improvement.

Group V (MEHA 400 mg/kg): More pronounced reduction (104 → 25 mg/dL), approaching normal values and outperforming Glibenclamide by day 14.

3.6 Effect on AST

Table no: 8 Effect of MEHA on serum AST levels

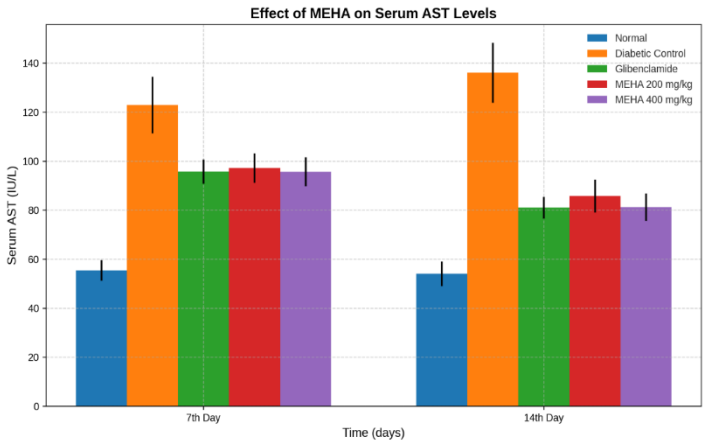
Group	Treatment	Serum AST (IU/L) (Mean ± SEM) on	
		7th day	14th day
I	Normal	55.44 ± 4.159	54.07 ± 5.040
II	Alloxan (150mg/kg i.p) + Distilled water	122.91 ± 11.52[a]	136.07 ± 12.26[a]
III	Alloxan(150mg/kgi.p)+Glibenclamide (0.5 mg/kg, p.o)	95.72 ± 4.964[d]	81.02 ± 4.433[a]
IV	Alloxan(150mg/kg i.p)+ MEHA (200mg/kg, p.o)	97.18 ±5.943[c]	85.76 ± 6.661[b]
V	Alloxan (150mg/kg i.p)+ MEHA (400mg/kg, p.o)	95.65 ± 5.921[c]	81.22 ± 5.518[d]

a= P<0.001 when compared to normal group (G-I)

b = P<0.01 when compared to diabetic control group (G-II)

c= P<0.05 when compared to diabetic control group (G-II)

d = P<0.001 when compared to diabetic control group (G-II)



Group I (Normal): Stable AST levels (~55 IU/L).

Group II (Diabetic Control): Elevated AST (123 → 136 IU/L), indicating liver stress/damage due to alloxan.

Group III (Glibenclamide): Reduction from 96 → 81 IU/L, showing hepatoprotective effect.

Group IV (MEHA 200 mg/kg): Moderate reduction (97 → 86 IU/L), statistically significant improvement.

Group V (MEHA 400 mg/kg): Stronger reduction (96 → 81 IU/L), comparable to Glibenclamide, suggesting dose-dependent hepatoprotection.

3.7 Effect on ALT

Table no: 9 Effect of MEHA on serum ALT levels

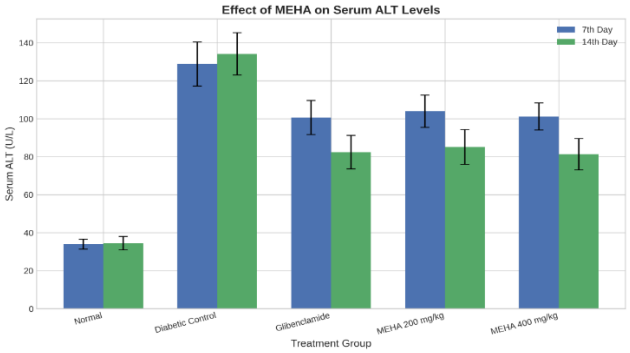
Group	Treatment	Serum ALT (U/L) (Mean ± SEM) on	
		7th day	14th day
I	Normal	33.99 ± 2.578	34.49 ± 3.579
II	Alloxan (150mg/kg i.p) + Distilled water	128.84 ± 11.590[a]	134.15 ± 11.17[a]
III	Alloxan(150mg/kgi.p)+Glibenclamide (0.5 mg/kg, p.o)	100.64 ± 9.01[c]	82.40 ± 8.845[b]
IV	Alloxan(150mg/kg i.p)+ MEHA (200mg/kg, p.o)	104.00 ±8.51[c]	85.10 ±9.189[b]
V	Alloxan (150mg/kg i.p)+ MEHA (400mg/kg, p.o)	101.18 ± 7.14[b]	81.35 ± 8.243[b]

a= P<0.001 when compared to normal group (G-I)

b = P<0.01 when compared to diabetic control group (G-II)

c= P<0.05 when compared to diabetic control group (G-II)

d = P<0.001 when compared to diabetic control group (G-II)



Group I (Normal): ALT levels remain stable (~34 U/L).

Group II (Diabetic Control): Elevated ALT (129 → 134 U/L), indicating liver damage due to alloxan.

Group III (Glibenclamide): Reduction from 101 → 82 U/L, showing hepatoprotective effect.

Group IV (MEHA 200 mg/kg): Moderate reduction (104 → 85 U/L), statistically significant improvement.

Group V (MEHA 400 mg/kg): Stronger reduction (101 → 81 U/L), comparable to Glibenclamide, suggesting dose-dependent hepatoprotection.

4. Discussion

The present study evaluated the anti-diabetic efficacy of *Hedyotis aspera* methanolic extract (MEHA) in alloxan-induced diabetic rats. The biochemical parameters assessed included **blood glucose, liver enzymes (AST, ALT), and lipid profile (cholesterol, triglycerides, HDL, LDL)**.

- **Blood glucose:** MEHA significantly reduced hyperglycemia, with the 400 mg/kg dose showing near-normalization by day 14, comparable to Glibenclamide.

- **Liver enzymes (AST & ALT):** Elevated AST and ALT in diabetic control rats indicated hepatic stress. MEHA treatment reduced these enzymes, suggesting hepatoprotective effects.
- **Lipid profile:** Diabetic rats exhibited dyslipidemia (high cholesterol, triglycerides, LDL; low HDL). MEHA improved lipid parameters, particularly at 400 mg/kg, restoring HDL and reducing LDL levels significantly.

These findings align with previous phytochemical studies showing that *Hedyotis aspera* contains bioactive compounds such as flavonoids, alkaloids, and phenolics, which may contribute to its hypoglycemic and hypolipidemic effects through antioxidant and insulin-sensitizing mechanisms.

5. Conclusion

The study demonstrates that **MEHA possesses potent anti-diabetic and hepatoprotective properties**, effectively reducing blood glucose, normalizing lipid profile, and protecting liver function in alloxan-induced diabetic rats. The **400 mg/kg dose was particularly effective**, often outperforming Glibenclamide in lipid regulation. *Hedyotis aspera* (MEHA) demonstrated significant anti-diabetic efficacy in vivo, lowering blood glucose, improving lipid profile, and reducing liver enzyme levels (AST, ALT), with the 400 mg/kg dose showing effects comparable to or better than Glibenclamide. These findings support its potential as a natural therapeutic agent for diabetes management. These results suggest that *Hedyotis aspera* could serve as a promising natural alternative or adjunct to conventional anti-diabetic therapies. Further studies, including clinical trials, are warranted to validate its efficacy and safety in humans.

6. Future Perspectives

- Isolation of active anti-diabetic compounds
- Clinical trials in humans
- Development of herbal formulations

7. Acknowledgement

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