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THE PHYTOCHEMICAL INVESTIGATION, ISOLATION AND EVALUATION OF ANTI-PARKINSONIAN ACTIVITY OF RHIZOME OF *DELPHINIUM DENUDATUM WALL* IN RATS

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ABSTRACT
The study was aimed to explore the anti-Parkinsonian (catatonian) potential of Delphinium denudatum wall in animal model. *Delphinium* species, annual or perennial, erect and hardy ornamental herbs are grown for their beautiful flowers. *Delphinium denudatum wall* is recommended for disorders like epilepsy, tremors, paralysis, facial paralysis, convulsions, chronic coryza, sinusitis, numbness, scorpion bite, etc. Qualitative phytochemical analysis of the ethanolic extract of Delphinium denudatum wall rhizome showed the presence of alkaloids, phenol, tannins, steroids, proteins, amino acids, carbohydrates, fats and oils in the ethanolic extract of Delphinium denudatum wall rhizome. The anti-parkinsonian activity of *Delphinium denudatum wall* rhizome was evaluated by drug induced catatonia method in Wister albino rats. Haloperidol was used to induce catatonia in rats and Bromocripitine was used as standard. It was observed that the % inhibition of catatonia after 30 minutes of haloperidol administration in group II, III, IV and V were 87, 74, 78 and 83 respectively. The observation was recorded for 2 hours and it was observed that there is decrease in duration of catatonia in group II, III, IV and V which was statistically more significant. The results suggested that the ethanolic extract of *Delphinium denudatum wall* rhizome showing anti-parkinsonianian activity. Hence, it can be safely administered in anti-parkinsonian patients with minimal effects.

KEYWORDS: Anti-Parkinsonian, Delphinium denudatum wall, catatonian, Haloperidol, Bromocriptine, Wister Albino rats.

INTRODUCTION
The aim of this study is the phytochemical investigation, isolation and evaluation of anti-parkinsonian activity of rhizome of *Delphinium denudatum wall* in rats. To explore the anti-parkinsonian potential of *Delphinium denudatum* in animal model and thereby extend its use to mankind safely with minimal adverse effects when compared to the conventional anti-parkinsonian drugs. A fundamental lesion in Parkinson's disease is a marked deficiency in the dopaminergic innervations of the basal ganglia owing to degeneration of neurons in the substantia nigra. Enhancement of dopaminergic transmission restores motor function at least partially. The decrease in dopaminergic activity in the basal ganglia results in a relative excess of cholinergic influence. Therefore, dopaminergic agonists, such as levodopa a precursor of dopamine, and cholinergic (muscarinic) antagonists can be combined in the treatment of Parkinson's disease. Parkinson-like syndromes also occur after depletion of central stores by reserpine and after treatment with phenothiazines and other antipsychotic drugs blocking dopamine receptors. (Vernier 1964; Marsden et al. 1975; Duvoisin 1976; Hornykiewicz 1975; Miller and Hiley 1975).[1,5]

Purpose and Rationale
Human embryonic stem cells may potentially serve as a renewable source of cells for transplantation. In Parkinson's disease, embryonic stem-cell-derived dopaminergic neurons may replace the degenerate neurons in the brain. To substantiate this goal, numerous animal experiments were performed. Most authors used rats rendered hemi-Parkinsonian by injection of 6-OHDA into the substantia nigra as recipients (Zawada et

al. 1998; Mendez et al. 2000; Sawamoto et al. 2001; Björklund et al. 2002; Hao et al. 2002; Kim et al. 2002; Ben-Hur et al. 2004; Burnstein et al. 2004; Jollivet et al. 2004; Levy et al. 2004; Yoshizaki et al. 2004; Rafuse et al. 2005; Richardson et al. 2005). Takagi et al. (2005) reported that dopaminergic neurons generated from cynomolgus monkey embryonicstem cells functioned in the MPTP primate model of Parkinson's disease. However, complications were reported by Blanchet et al. (2003) using a similar approach. In spite of almost 20 years of experimental experience, clinical trials with cell transplantation for Parkinson's disease have had disappointing results.[5,9] Parkinsonism is a neurodegenerative extra pyramidal motor disorder characterized by rigidity, tremor and hyperkinesias with secondary manifestations like detective posture and gait, mask-like face and sialorrhoea; dementia may accompany. If untreated the symptoms progress over several years to end-stage disease in which the patient is rigid, unable to move, unable to breathe properly; succumbs mostly to chest infections.[10,13] Parkinson's disease (PD) is a chronic neurological disorder, characterized by a selective loss of dopaminergic neurons in the substantia nigra (SN) of the ventral midbrain area, causing a subsequent reduction of dopamine (DA) levels in the stratium.[14,17] Parkinson's disease (PD) is a progressive degenerative disorder mostly affecting older people and is a common movement disorder. It is characterized by progressive loss of muscle control, which leads to trembling of the limbs and head while at rest, stiffness, slowness and impaired balance. As the symptoms worsen, it becomes difficult to walk, talk and complete simple tasks. The most consistent lesion in PD is degeneration of neurons in the substantia nigra pars compacta (SN-PC) and the nigrostriatal (dopaminergic) tract. This results in deficiency of dopamine in the stratium which controls muscle tone and coordinates movements. When dopamine producing cells are lost, normal movement is impossible. Premature death is usually due to complications such as falling-related injuries or pneumonia. In people with late-stage Parkinson's disease, 80% or more of these important cells are dead or impaired.[18,20]

Drug Extraction

A. Size Reduction of Drug

Previously dried crude drug was suitably size reduced to coarse particles. Size reduction was done by using ordinary motar and pestle.

B. Preparation of Extract

1) About 250g of delphinium denudatum wall (rhizome) to be extracted is packed in a paper cylinder made from a filter paper & is placed in the body of extractor.

α. The solvent ethanol is placed in flask.

β. The apparatus is fitted as shown in above figure.

χ. When solvent is boiled it is converted into vapors. They enter into the condenser through side tube & gets condensed into hot liquid which falls on the column of the drug.

δ. When extractor gets filled with solvent, the level of siphon tube also raises to its top.

2) Solvent containing active ingredients of drug siphon over & run into flask, thus emptying body of extractor.

3) The soluble active constituents of the drug remain in the flask while the solvent is repeatedly volatilized.

4) The process of filling and emptying of the extractor is repeated until the drug is exhausted.

After complete extraction of the drug, the extract was subjected to distillation for evaporation of solvent. Finally, the drug extract was collected as brownish sticky mass after distillation.

Column Chromatography

The ethanolic extract was concentrated to about 100ml and 100ml water was added to the concentrated extract. Then the concentrated extract was taken in a separating funnel where the extract was separated with three different solvents (Petroleum ether, Chloroform and ethyl acetate).

Steps involved in column chromatography are

1) Silica gel was used as adsorbent (stationary phase) and n-hexane : ethyl acetate at different ratios such as 9:1,8:2,7:3 and 6:4 were used as mobile phase which acts as solvent, developer and eluent.
2) The bottom of column was first packed with cotton wool, above which the column of silica gel was packed.
3) Required quantity of silica gel was mixed with mobile phase in a beaker and poured into the column.
4) The silica gel settles in the column uniformly.
5) A filter paper disc was placed above the stationary phase to avoid any disturbances during the addition of sample and mobile phase
6) Weighed quantity of extract was triturated with silica gel into fine powder and the sample was introduced into the column.
7) After packing the sample in the column, the mobile phase was introduced from the top into the column.
8) The components of the samples get separated depending upon the affinity towards stationary phase.
9) The recovery of the components was done by elution technique.
10) Totally 8 no of fractions were collected.

Isolation of individual components from column chromatography was confirmed by Thin Layer.

Chromatography (TLC)

TLC- Procedure

1) Precoated silica gel aluminum plates were used as stationary phase and n-hexane: ethyl acetate

(8.5:1.5) was used as mobile phase which acts as solvent and developer.
2) UV Chamber and iodine vapor were used as detecting agents.
3) A origin line was marked at the bottom of the silica gel aluminum plates with pencil.
4) The sample was applied on the origin line as a small spot with the help of a capillary tube.
5) The solvent or mobile phase was poured into the developing chamber.
6) The TLC plate with sample was placed into the developing chamber containing solvent with the help of forceps.
7) The solvent was allowed to travel up to $3/4^{th}$ of the plate.
8) The plate was allowed to dry and then the iodine vapor was used to detect the spots on TLC.
9) Fluorescent spots against dark background were observed under the UV light in UV chamber at 254 and 366µm.
10) This confirmed the isolation of individual components.

Spectral Data Analysis of Isolated Compounds
The isolated components from column chromatography were then subjected to spectral data analysis by FT-IR, ^{1}H-NMR and ^{13}C-NMR spectroscopy and Mass spectroscopy.

FT-IR Analysis
The powdered sample was mixed with dry potassium bromide (KBr) and subjected to a pressure of about $5x10^{6}$ pa in a evacuated die to produce a clear transparent disc of diameter 13mm and thickness 1mm. IR spectra in frequency 4000-400cm^{-1} was recorded at room temperature on a Perkin-Elmer transform spectrometer equipped with air cooled deuterated triglycine sulfate (DTGs) detector. For the spectrum 45 scans were co-added at a spectral resolution of 2cm^{-1}.

Nmr Spectroscopy
NMR spectroscopy of isolated fractions 6 and 7 was conducted at Hyderabad University where deuterated chloroform was used as solvent.

Mass Spectroscopy
Fraction 6 and 7 were analysed by mass spectroscopy.

Catatonia In Rats
Objective: To study antiparkinsonian (anti-catatonic) activity of Delphium denudatum wall using drug induced catatonia method in rats.

Requirements
Animals
Prior approval from IAEC was obtained ref no: IAEC/SCUP/03/2013 to carry out the work in animals.

Wister albino rats of either sex were used in the present study weighing about 150-200g. The animals were housed in propylene cages with dust free rich husk as bedding materials. 12 hours of light and 12 hours of dark cycle was maintained throughout the experimental period. All animals were provided with water and feed continuously.

Drugs
Haloperidol- 1mg/kg body weight of the animal
Bromocriptine- 1mg/kg body weight of the animal
Delphinium denudatum -100mg/kg, 200 mg/kg and 300 mg/kg body weight of the animal

Procedure
The animals were divided into five groups, each consisting of six (n=6).

Group 1: Control
To the control group, Haloperidol (1mg/kg) was administered intra peritoneally.

Group 2: Standard
To the standard group, Bromocriptine (1mg/kg) was administered intra peritoneally.

Group 3: Delphinium denudatum 100mg/kg
To the group 3 animals, *Delphinium denudatum* 100mg/kg (100 mg in 0.5% CMC) was administered orally and 30 minutes later, Haloperidol was administered intra peritoneally.

Group 4: Delphinium denudatum 200mg/kg
To the group 4 animals, *Delphinium denudatum* 200 mg/kg (200 mg in 0.5% CMC) was administered orally and 30 minutes later, Haloperidol was administered intra peritoneally.

Group 5: Delphinium denudatum 300mg/kg
To the group 5 animals, *Delphinium denudatum* 300mg/kg (300 mg in 0.5% CMC) was administered orally and 30 minutes later, Haloperidol was administered intra peritoneally. The severity of catatonia was observed at 15, 30, 45, 60, 90 and 120 minutes after Haloperidol administration. The severity of catatonic response in rats was scored as follows:

STAGE 1: Rat moves normally when placed on the table; score=0.
STAGE 2: Rat moves only when touched or pushed; score=0.5.
STAGE 3: Rat placed on the table with front paws set alternately on a 3cm high block fails to correct the posture in 10 seconds; score=0.5 for each paw with a total of 1 for this stage.
STAGE 4: Rat placed on the table with front paws set alternately on a 9 cm high block fails to correct the posture in 10 seconds; score=1 for each paw with a total of 2 for this stage.

Thus, for a single rat, the maximum possible score would be 3.5 revealing total catatonia. Plot a graph, time along

X-axis and % of inhibition on Y-axis. Note the difference in the onset and severity of catatonic response among the groups. The percent inhibition of catatonia in test drug and standard drug was calculated in comparison with control group by the formula,

100 x (1-a/b)

Where 'a' is the mean anti-catatonic score of the test / standard animals and 'b' is the mean anti-catatonic score of the control animals.

Statistical Significance

The mean score of various group of animals were statistically compared to determine significance of difference by using students unpaired t-test ($p<0.05$).[4]

RESULTS

Phytochemical Investigation

The phytochemical investigation identified and confirmed the presence of alkaloids, phenol, tannins, steroids, proteins, amino acids, carbohydrates, fats and oils in the ethanolic extract of *Delphinium denudatum wall* rhizome.

Spectral Analysis

Totally 8 fractions were isolated from the extract using column chromatography. These isolated individual components were then subjected to spectral data analysis FT-IR, ^{1}H-NMR and ^{13}C-NMR Spectroscopy and MASS Spectroscopy. The spectral data analysis is shown in fig 1, 2, 3, 4, 5, 6 (a), 6 (b), 7 (a) and 7 (b). From the FT-IR spectral data analysis of *Delphinium denudatum wall* it was predicted that it may contain the following functional groups as shown in Table 1:

IR peak cm-1 (KBr)	Group/vibration
2855	O-H Stretching
1594	N-H Bending
1339	C-N Stretching
1105	C-O Stretching
1008	C=C Stretching
859.7	C-H Bending

From the ^{1}H-NMR spectral data analysis of fraction 6 it was predicted that it may contain the following functional groups as shown in Table 1.1:

Chemical Shift δ in ppm	Type Of Proton
7.2	Ar-H
5.34	Ar-OH
2.3	CH_3-Ar
2.0	R-OH
1.6	CH_3-C=C
1.3	R_2CH_2

From the ^{13}C-NMR spectral data analysis of fraction 6 it was predicted that it may contain the following functional groups as shown in Table 1.2:

Chemical Shift $^{\delta}$C in ppm	Type Of Proton
178.9	C=O
130	C in aromatic
128	C=C
77.3	C-O
33.8	R_3CH
29.2	C-C
27.2	CH_3CO

From the ^{1}H-NMR spectral data analysis of fraction 7 it was predicted that it may contain the following functional groups as shown in Table 1.3:

Chemical Shift δ in ppm	Type Of Proton
5.3	Ar-H
4.3	RNH_2
2.3	CH_3-Ar
2.0	R-OH
1.6	CH_3-C=C
1.3	R_2CH_2

From the ^{13}C-NMR spectral data analysis of fraction 7 it was predicted that it may contain the following functional groups as shown in Table 1.4:

Chemical Shift $^{\delta}$C in ppm	Type Of Proton
130	C in aromatic
77.3	C-O
61.67	RCH_2OH
27.2	R_3CH
24.3	CH_3OH

Anti-Parkinsonian Activity

In the present study Haloperidol produced a catatonia which was significant as compared to vehicle treated animals. Standard drug Bromocriptine produced significant anti catatonic effect at all time intervals but maximum effect was seen at 30 minutes after haloperidol challenge. *Delphinium denudatum wall* has shown significant anti catatonic effect in dose dependant manner as compared to haloperidol alone treated animals at 15, 30, 45, 60, 90 and 120 minutes.

It was observed that the % inhibition of catatonia after 30 minutes of haloperidol administration in group II, III, IV and V were 87, 74, 78 and 83 respectively. There was a significant decrease in duration of catatonia in group II, III, IV and V as compared to control group ($p<0.05$) as shown in table no 4 and fig no 8.

The observation was recorded for 2 hours and it was observed that there is decrease in duration of catatonia in group II, III, IV and V which was statistically more significant. The peak protective anti-parkinsonian activity of *Delphinium denudatum wall* against haloperidol induced catatonia was observed at 30 minutes.

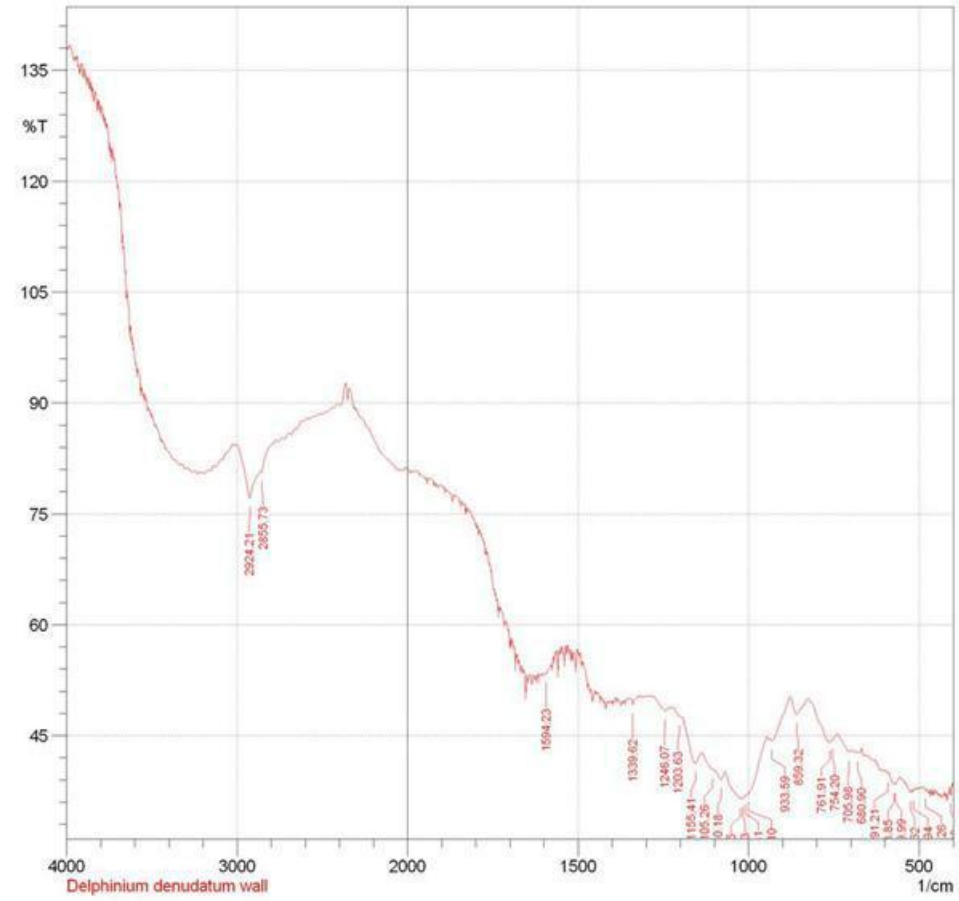

Fig. no. 1: IR Spectra of Delphinium Denudatum Wall Extract.

Table 2: IR Analysis of *Delphinium denudatum wall* Extract.

No.	Peak	Intensity	Corr. Inte	Base (H)	Base (L)	Area	Corr. Are
1	407.96	36.97	1.24	409.89	402.18	3.27	0.06
2	481.26	37.65	0.21	487.05	479.33	3.26	0.01
3	516.94	37.38	0.42	521.77	514.05	3.28	0.02
4	525.62	37.33	0.48	537.2	522.73	6.12	0.05
5	569.99	38.45	0.12	570.95	561.31	3.96	0.01
6	573.85	38.36	0.31	588.31	571.92	6.72	0.05
7	591.21	39.84	0.23	596.99	589.28	3.07	0.01
8	680.9	42.67	0.25	685.72	677.04	3.2	0.01
9	705.98	42.8	0.52	740.7	700.19	14.5	0.1
10	754.2	44.37	0.06	755.16	741.66	4.7	0
11	761.91	44.08	0.69	823.64	757.09	22.04	0.27
12	859.32	47.83	2.32	878.61	829.43	15.29	0.53
13	933.59	44.28	0.63	937.44	879.58	19.09	0.31
14	1001.1	37.19	0.14	1002.06	946.12	21.87	0.19
15	1008.81	36.86	0.05	1009.78	1002.06	3.33	0
16	1016.53	36.55	0.06	1017.49	1010.74	2.94	0
17	1024.25	36.42	0.5	1068.61	1019.42	20.85	0.34
18	1080.18	39.02	1.24	1101.4	1069.57	12.78	0.22
19	1105.26	40.4	0.24	1136.12	1102.37	12.97	0.09
20	1155.41	41.29	2.83	1199.78	1137.09	22.55	0.86
21	1203.63	47.6	0.19	1223.89	1200.74	7.34	0.01
22	1246.07	48.34	0.61	1258.61	1233.53	7.86	0.07
23	1339.62	49.19	0.96	1346.37	1322.26	7.28	0.06
24	1594.23	53.37	0.3	1598.09	1578.8	5.18	0.03
25	2855.73	80.67	0.52	2862.49	2801.73	5.02	0.03
26	2924.21	77.13	4.78	3000.4	2875.99	11.82	1.32

The IR of this compound may be: IR Cm^{-1} (KBr): 2855 (O-H *str*); 1594 (N-H *ben*); 1339 (C-N *str);* 1105 (C-O *str*); 1008 (C=C *str*); 859.7 (C-H *ben*).

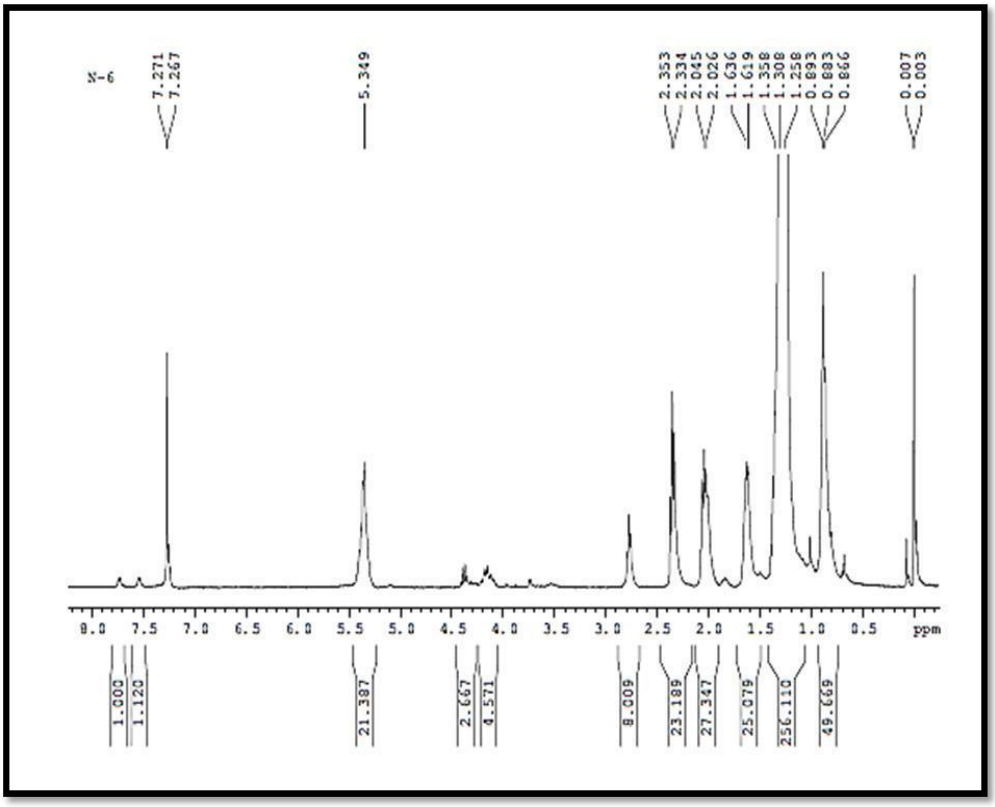

Fig. no. 2: ^{1}H -NMR Specrta of Fraction No 6.

The ^{1}H-NMR Spectral data of this fraction may be: ^{1}H-NMR ($CHCl_3$ δ ppm): 7.2 (Ar-H); 5.34 (Ar-OH); 2.3 (CH_3-Ar); 2.0 (R-OH); 1.6 (CH_3-C=C); 1.3 (R_2CH_2).

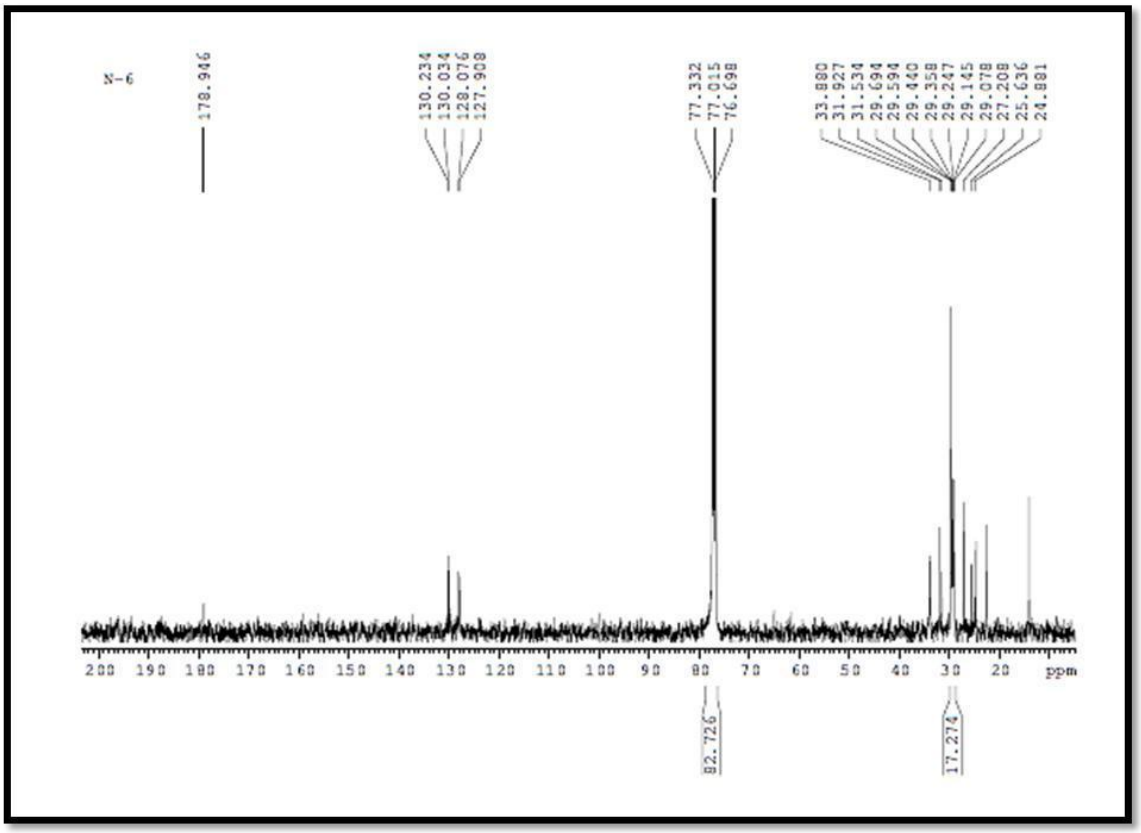

Fig. no. 3: ^{13}C- NMR Specrta of Fraction No 6.

The ^{13}C-NMR Spectral data of this fraction may be: ^{13}C-NMR ($CHCl_3$ $^{\delta}$C ppm): 178.9 (C=O); 130 (C in aromatic); 128 (C=C); 77.3 (C-O);33.8 (R_3CH); 29.2 (C-C); 27.2 (CH_3CO).

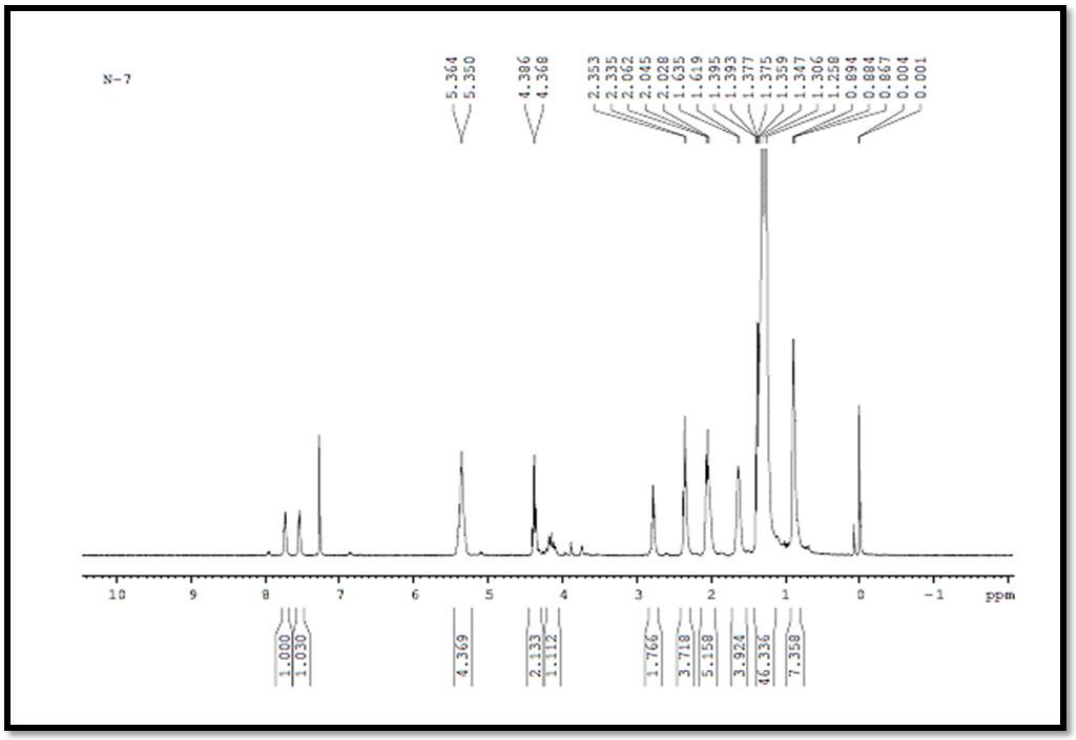

Fig. no. 4: ^{1}H -NMR Specrta of Fraction No 7.

The ^{1}H-NMR Spectral data of this fraction may be:
^{1}H-NMR ($CHCl_3$ δ ppm): 5.3 (Ar-OH); 4.3 (RNH_2); 2.3 (CH_3-Ar); 2.0 (R-OH); 1.6 (CH_3-C=C); 1.3 (R_2CH_2)

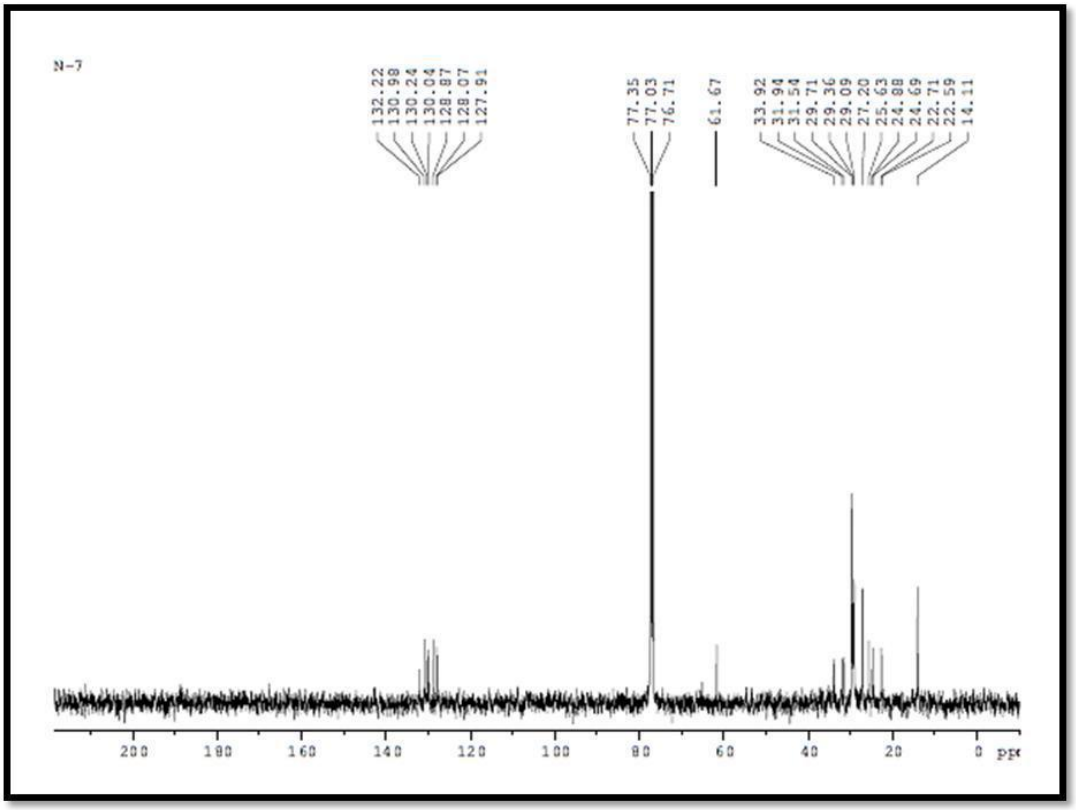

Fig. no. 5: ^{13}C- NMR Specrta of Fraction No 7.

The ^{13}C-NMR Spectral data of this fraction may be: ^{13}C-NMR ($CHCl_3$ $^{\delta}$C ppm): 130 (C in aromatic); 77.3 (C-O); 61.67 (RCH_2OH); 27.2 (R_3CH); 24.3 (CH_3CO).

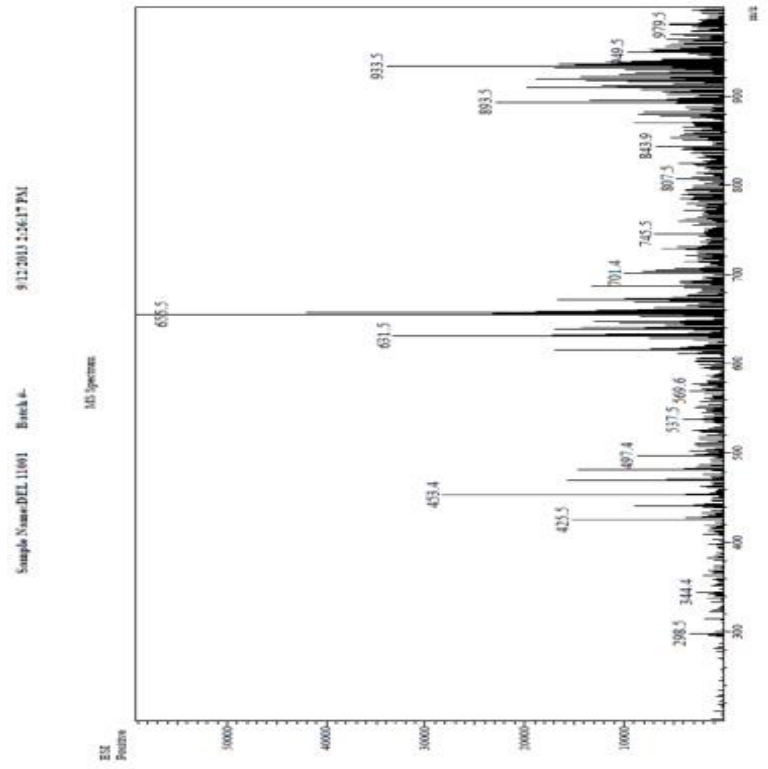

Fig. no. 6 (a): MASS Spectrum of Fraction 6.

From the mass spectrum, it can be assumed that 979.5 is the molecular ion.

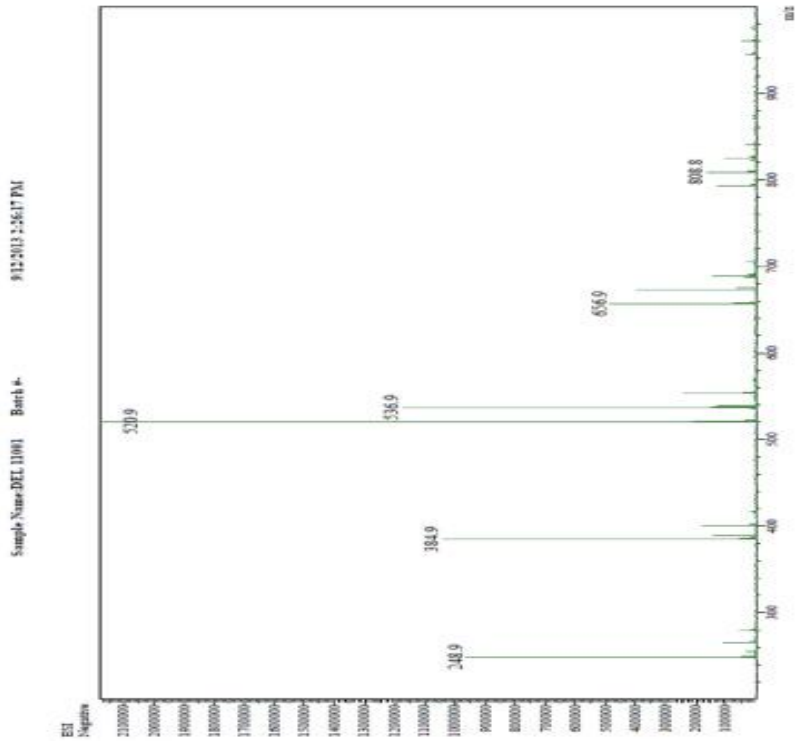

Fig. no. 6 (b): MASS Spectrum of Fraction 6.

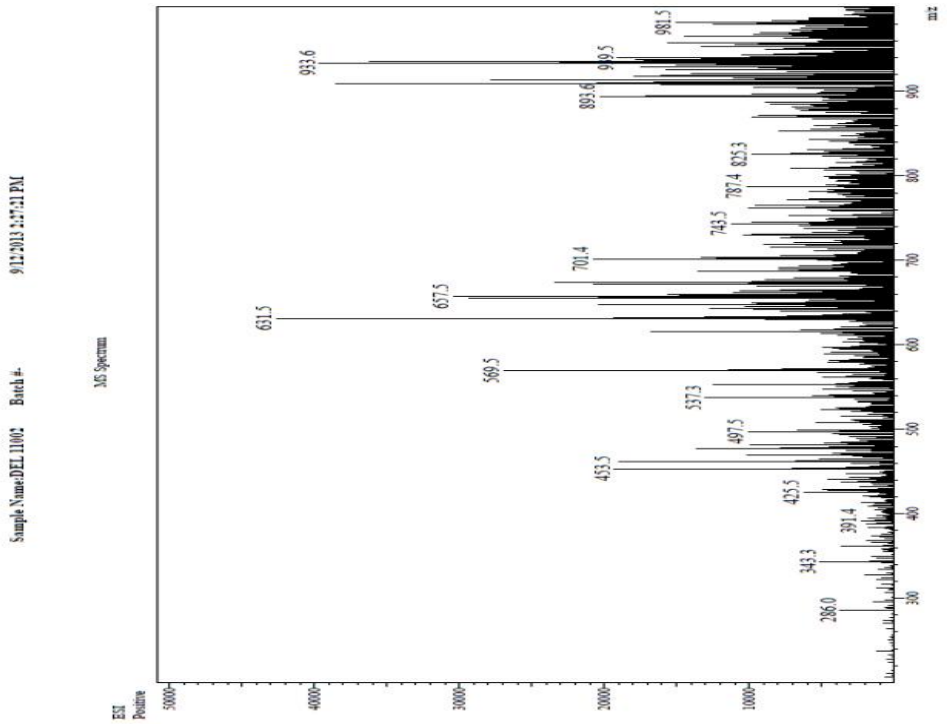

Fig. no. 7 (a): MASS Spectrum of Fraction 7.

From the mass spectrum, it can be assumed that 981.5 is the molecular ion.

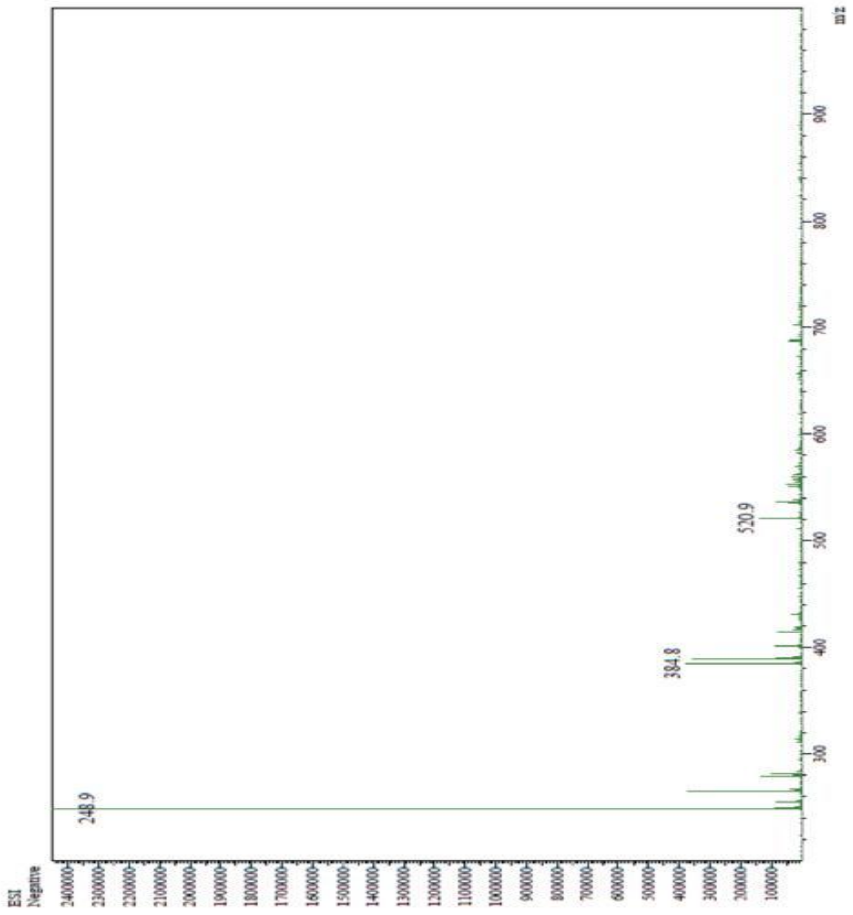

Fig. no. 7 (b): MASS Spectrum of Fraction 7.

Table No. 3: Mean Catatonic Score of Animals.

GROUP	15 MIN	30 MIN	45 MIN	60 MIN	90 MIN	120 MIN
	MEAN SCORE ± SE	MEAN SCORE ± SE	MEAN SCORE ± SE	MEAN SCORE ± SE	MEAN SCORE ± SE	MEAN SCORE ± SE
	2.45	2.77	2.85	2.68	2.60	2.48
GROUP I	±0.32	±0.46	±0.21	±0.19	±0.43	±0.52
	0.61	0.35	0.46	0.60	0.65	0.65
GROUP II	±0.31	±0.46	±0.27	±0.31	±0.36	±0.29
	0.94	0.71	0.78	0.84	0.95	0.96
GROUP III	±0.21	±0.26	±0.28	±0.23	±0.31	±0.19
	0.81	0.62	0.71	0.71	0.83	0.89
GROUP IV	±0.48	±0.39	±0.31	±0.41	±0.36	±0.35
	0.87	0.48	0.60	0.65	0.85	0.78
GROUP V	±0.43	±0.27	±0.31	±0.36	±0.46	±0.24

Table No. 4: % Inhibition Score of Animals.

GROUP	15 MIN	30 MIN	45 MIN	60 MIN	90 MIN	120 MIN
GROUP I	74	86	83	76	71	72
	75	87	84	78	75	74
GROUP II						
	61	74	72	69	63	61
GROUP III						
	67	78	75	74	68	64
GROUP IV						
	70	83	78	75	69	65
GROUP V						

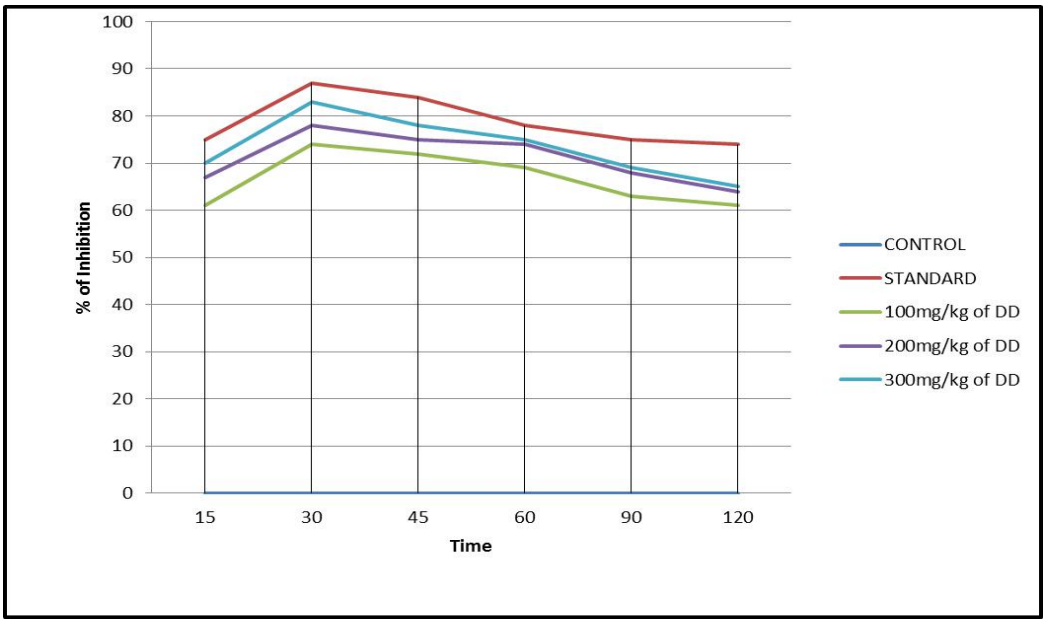

Fig. no. 8: Study of Anti-Parkinsonian Activity.

DISCUSSION

Phytochemical investigations for different chemical constituents in extract were done with respective chemical identification tests. These investigation identified and confirmed the presence of alkaloids, phenol, tannins, steroids, proteins, amino acids, carbohydrates, fats and oils. From the support from previous research work we can conclude that 'delphinine' may be the phytochemical constituent responsible for the anti-parkinsonian activity of the drug. The IR spectra of *Delphinium denudatum wall* shows Ar-OH stretching at 2855 cm^{-1} which can also be observed at 5.34 δ ppm in ^{1}H-NMR spectra of fraction 6. So, it can be concluded that aromatic OH stretching may be present in fraction 6.

The IR spectra show C-O stretching at 1105 cm^{-1} which can also be observed in ^{13}C-NMR spectra of fraction 6 at

77.3 δ ppm. C=C stretching observed at 1008 cm-1 in the ir spectra can also be observed at 128 δ ppm in ^{13}C-NMR spectra of fraction 6. Hence we can conclude that fraction 6 contain O-H, C-O and C=C stretching. The IR spectra of *Delphinium denudatum wall* shows N-H bending at 1594 cm^{-1} which can also be observed at 4.3 δ ppm in ^{1}H-NMR spectra of fraction 7. The IR spectra of *Delphinium denudatum wall* shows Ar-OH stretching at 2855 cm^{-1} which can also be observed at 5.34 δ ppm in ^{1}H-NMR spectra of fraction 7. So, it can be concluded that aromatic OH stretching and N-H bending may be present in fraction 7. The IR spectra shows C-O stretching at 1105 cm^{-1} which can also be observed in ^{13}C-NMR spectra of fraction 7 at 77.3 δ ppm. Hence we can conclude that fraction 7 may contain N-H, O-H and C-O stretching. Haloperidol induced catalepsy is one of the animal model to test for the side effects of anti-parkinsonian drugs. The Haloperidol, (a non selective D_2 dopamine antagonist) induced catalepsy is primarily due to blockade of dopamine receptor in stratrium. Serotonin is also involved in neuroleptic catalepsy.

Protective effect of *Delphinium denudatum wall* (100, 200 and 300mg/kg) against haloperidol induced catalepsy suggests that this unani medicine not only influence dopamine receptor-mediated neurotransmission but also serotonergic receptor –mediated neurotransmission. In the present study, it has been observed that *Delphinium denudatum wall* reduce the severity of haloperidol induced catalepsy score in rats. The anti-parkinsonian activity could be possibly due to the presence of alkaloid “delphinine. From the present study, it can be concluded that the Unani drug *Delphinium denudatum wall* can be added as beneficial adjuvant in treatment of extra pyramidal side effects and related disorders. Clinical trials in humans suffering from Parkinson disease may provide us with greater evidence for neuroprotective effect of this Unani drug.

CONCLUSION

Delphinium species, annual or perennial, erect and hardy ornamental herbs are grown for their beautiful flowers. *Delphinium denudatum* commonly known as Jadwar is found on the outer ranges of western Himalayas from Kashmir to Kumoan at a height between 8000 to 12000 feet above sea level, especially on grass slopes. The root of *Delphinium denudatum wall* is used as a sedative, analgesic, brain and nervine tonic. *Delphinium denudatum wall* is recommended for disorders like epilepsy, tremors, paralysis, facial paralysis, convulsions, chronic coryza, sinusitis, numbness, scorpion bite, acute poisoning, cholera, jaundice, palpitation, locally on enlargement/inflamed glands, hysteria, atony and morphine dependence. Qualitative phytochemical analysis showed the presence of alkaloids, phenol, tannins, steroids, proteins, amino acids, carbohydrates, fats and oils in the ethanolic extract of *Delphinium denudatum wall* rhizome. Spectral data analysis FT-IR, NMR Spectroscopy, MASS Spectroscopy of isolated individual components from column chromatography was conducted to reveal the presence of functional group present in the fractions. From the support from previous research work we can conclude that ‘delphinine’ may be the phytochemical constituent responsible for the anti-parkinsonian activity of the drug. The antiparkinsonian activity of *Delphinium denudatum wall* rhizome was evaluated by drug induced catatonia method in Wister albino rats. Haloperidol was used to induce catatonia in rats and Bromocripitine was used as standard.

With the available results through the experiments, we can conclude *Delphinium denudatum wall* has antagonizing activity against catatonia induced by Haloperidol. the anti-parkinsonian potential of *Delphinium denudatum wall* was found to be similar to that produced by the standard Bromocriptine drug. The results suggested that the ethanolic extract of *Delphinium denudatum wall* rhizome showing anti-parkinsonian activity. Hence, we can conclude that the unani drug, *Delphinium denudatum wall* can be safely administered in anti-parkinsonian patients with minimal effects.

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