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Clinical Trial on *Satavari* (*Asparagus racemosus* Willd.) in Duodenal Ulcer Disease

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Clinical efficacy of Satavari (Asparagus racemosus) was evaluated in 32 patients with proved duodenal ulcer disease. Drop out rate was 18.75 per cent. Root powder of Satavari (12.0 g/day in four divided doses) was given for an average duration of six weeks. Criteria of assessment were symptomatic relief, reduction in gastric acidity response (both AHT and FTM) and radiologic as well as endoscopic improvement. Satavari relieved most of the symptoms in majority of the patient promptly and persistently. Satavari did not exhibit antacid activity. foreover, Satavari does not appear to possess strong antisecretory effect since it inhibited basal output by only 48 percent, histamine induced maximum output by 38 percent and alcohol induced secretion by only 32 percent. Approximately three fourth patients had radiologic and endoscopic improvement, Probably, the ulcer healing effect of drug Satavari appears due to its direct healing effect probably via strengthening the mucosal resistance or cytoprotection.

Introduction

The management of *Parinama Sula*, a clinical entity quite akin to duodenal ulcer disease, has been elaborately delineated in Ayurvedic texts. Various beneficial diets and drugs have been described to treat this chronic and recurrent ailment. *Satavari* (*Asparagus racemosus*, Liliaceae) is a reputed herb for the patients with *Parinama Sula* or *Paittika Sula*. Few clinical trials on *Satavari* in duodenal ulcer patients have been reported earlier but the size of the sample being small (Maheshwari and Chaturvedi, 1977; Gupta et al, 1982; Mishra et al, 1985), these studies remained inconclusive. Therefore, the present clinical trail was launched to evaluate the effectiveness of *Satavari* in duodenal ulcer disease on a longer series adopting standard parameters.

Materials and Methods

The present series consisted of 32 patients with proved active duodenal ulcer disease. The powder of roots of trial drug *Satavari* was given in the dose of 120 g per day in four divided doses with water preferably after meals for an average duration of six weeks. The patients were asked to continue their routine dietary regimen throughout the period of trial. Results were assessed in terms of symptomatic improvement inferred from favourable shift of grades of each symptom from '3' to '0' reduction in gastric acidity response adopting both the methods e.g. augmented histamine test (AHT-Kay, 1953) and fractional test meal (FTM-Kolmer et al, 1969) and radiological and/or endoscopical ulcer healing after treatment. Patients were advised to take Gelusil MPS liquid or Baralgan Tablets whenever severe pain occurs and total quantity of antacid and/or anti-spasmodic

drug was recorded at the time of final assessment. Patients were also asked about adverse effect, if any, experienced during the drug intake period.

Results and Observations

The mean age (years) of trial group was 40.58±2.52 and mean body weight (kg) was 55.92±1.21. The dropout rate in the present study was 18.75 per cent indicating the follow up study of 26 patients. Symptomatic relief in terms of mean grade scores has been depicted in Table-I. Abdominal pain was present in all the patients initially, out of them 92.31 per cent got improvement which was usually felt within a week after treatment.

Table-I

Symptomatic relief in 26 patietns with peptic ulcer after treatment with *Satavari* (*Asparagus racemosus* Willd.)

Symptoms	Incidence%	Shift in mean grade score (mean ± SEM) (n=26)			
		Initial	After Tt.	t	P
pain in abdomen	100.00	2.54±0.11	1.00±0.13	11.000	<0.001
Nausea	61.54	0.88±0.17	0.15±0.07	05.615	<0.001
Vomiting	42.31	0.62±0.17	0.27±0.13	03.680	<0.001
Heartburn	92.31	2.15±0.16	0.58±0.11	10.000	<0.001
Regurgitation	84.62	1.65±0.19	0.50±0.011	08.846	<0.001
Belching	73.08	1.42±0.20	0.62±0.12	07.363	<0.001
Flatulence	73.08	1.54±0.21	0.69±0.13	07.083	<0.001
Constipation	50.00	0.85±0.14	0.58±0.12	03.000	<0.001

Patients occasionally required antacids or antispasmodic drugs. The initial mean grade score of pain was 2.55±0.11 that reduced to 1.00±0.13 showing highly significant improvement ($p<0.001$). Maximum relief was observed in the pain of burning nature. Likewise, heartburn and sour eructations, the features of hypersecretion which were observed initially in over 85 per cent patients reduced significantly. Similarly, certain other symptoms such as flatulence belching, nausea, constipation were also relieved.

Gastric acidity response could be evaluated in 76.92 per cent patients of this series, 46.15 per cent with augmented histamine test (AHT) and 30.77 per cent cases with fractional test meal (FTM). As much as 65 percent patients were found hypersecretors; 66.67 per cent cases in AHT series whereas 62.50 per cent patients in FTM series. Observations made in AHT series have been depicted in Table II. Obviously, a wide range of variation was noted in acidity response. Mean initial values of basal acid output (BAO) maximum acid output (MAO) and peak acid output (PAO) were 9.38±2.08 (1.2 to 24.5), 21.62±2.40 (8.5 to 41.0) and 25.43±2.23 (11.5 to 42.8) milliequivalents per hour (meq/h) respectively. After treatment, a highly significant reduction ($p<0.001$) was observed in each value. Similarly, a wide range of variation was also observed in FTM series.

Table-II

Effect of *Satavari* (*A. racemosus*) on gastric acidity response to histamine (augmented histamine test) after treatment in patients with peptic ulcer (mean ± SEM) (n)

Acidity Response	Gastric acidity (meq/h)		Mean per cent reduction	t	p
	Initial	After treatment			
(1) BAO	9.38±2.08 (12)	4.27±0.85 (12)	48.67 ±5.21	3.871	<0.001
(2) MAO	21.62±2.40 (12)	12.45±1.22 (12)	37.90 ±7.15	4.245	<0.001
(3) PAO	25.43±2.23 (12)	14.57±1.28 (12)	39.66 ±9.16	5.225	<0.001
Ratio (%)	39.42±6.06 (12)	32.07±4.07 (12)		1.783	NS
BAO: MAO					

Initial values of free acidity (Table III); e.g. 36.37±5.17 (16 to 60), 56.63±9.29 (35 to 92), 62.88±6.76 (50 to 96), 64±38±4.10 (60 to 80), 57.50±5.22 (32 to 80) and 53.86±6.50 (38 to 82) respectively for fasting and then for five consecutive 30 minutes samples, reduced to 22.88±2.72 (15 to 40), 35.38±5.71 (25 to 60), 45.75±4.30 (25 to 68), 38.00±6.26 (30 to 52), 36.75±3.21 (26 to 50) and 35.00±4.63 (20 to 50) respectively. The reduction was highly significant. Likewise the reduction in total acidity was also found significant (Table-IV).

Table-III

Effect of *Satavari* (*A. racemosus*) on free gastric acidity response to fractional test meal (FTM) after treatment in patients with peptic ulcer mean ± SEM

Sample	Free gastric acidity		Mean per cent reduction	t	p
	Initial	After treatment			
(1) 0.0 h	36.37±5.17 (8)	22.88±2.72 (8)	32.18±6.96	3.924	<0.01
(2) 0.5 h	56.63±9.29 (8)	35.38±5.71 (8)	36.04±3.24	3.571	<0.01
(3) 1.0 h	62.88±6.76 (8)	45.75±4.30 (8)	28.45±6.95	3.517	<0.01
(4) 1.5 h	64.38±4.10 (8)	38.00±6.26 (8)	34.48±4.10	6.715	<0.001
(5) 2.0 h	57.50±5.22	36.75±3.21	34.39±5.08	5.796	<0.001
(6) 2.5 h	53.86±6.50 (7)	35.00±4.63 (7)	35.36±3.43	6.908	<0.001

Ulcer healing effect of *Satavari* was evaluated in terms of radiological as well as endoscopic improvement. Barium X-ray study of stomach and duodenum could be done

in 69.23 per cent patients of this series. Duodenal bulb deformity with spasm was observed in 50 per cent cases of this study, of which equal number of patients (16.07 percent) got complete radiological recovery and partial improvement. Ulcer crater was seen in 38.89 percent cases and 33.33 percent patients got recovery form that. Pre-and post-treatment gastroduodenoscopy could be done in 46.15 per cent patients. Complete ulcer healing was observed in 50 per cent cases and partial healing in 25 per cent patients.

Table-IV
Effect of *Satavari* (*A. racemosus*) on free gastric acidity response to fractional test meal (FTM) after treatment in patients with peptic ulcer mean ± SEM (n)

Sample	Free gastric acidity		Mean per cent reduction	t	p
	Initial	After treatment			
(1) 0.0 h	52.50±11.28 (8)	28.87±4.09 (8)	35.08±8.32	2.607	<0.05
(2) 0.5 h	57.25±9.93 (8)	34.00±5.38 (8)	38.60±2.96	4.281	<0.001
(3) 1.0 h	70.12±6.62 (8)	47.37±3.74 (8)	31.18±6.28	3.428	<0.01
(4) 1.5 h	70.25±5.67 (8)	47.87±3.26 (8)	30.64±5.02	5.004	<0.001
(5) 2.0 h	64.87±5.41	42.87±4.75	30.84±10.65	3.728	<0.01
(6) 2.5 h	58.71±5.64	41.14±4.18	26.27±5.00	4.458	<0.001

Discussion

Although the abdominal pain related with meal has been discussed quite often in both the Vedic literature and classical Ayurvedic texts like Caraka Samhita, Susruta Samhita but it was a veteran Ayurvedic scholar *Madhavakara* who first identified this clinical condition as a separate disease entity. He named it '*Parinama Sula*' indicating its strong food relationship. *Parinama Sula* owing to its similar clinical features is fairly correlated with duodenal ulcer disease (Chaturvedi et al, 1979). Moreover, clinical efficacy of many Ayurvedic drugs prescribed for *Parinama Sula* has been proved in duodenal ulcer disease by various workers (Varma et al, 1977-Amalaki (*Emblica officinale*); Chaturvedi *et al*, 1979- *Madhuyasthi* (*Glycyrhiza glabra*). Predominant vitiation of Pitta *Dosa* appears to be a causal phenomenon in the genesis of *Parinama Sula*. Juice of *Satavari* is a well reputed remedy for *Paittika* diseases particularly *Paittika Sula* (Cakradutta-26:28, *Sarangadhara Samhita*-Ma. 1:15), Moreover, *Satavari* is well indicated in *Parinama Sula* (*Cakradutta* 27: 35-37, 49-33). The present clinical study evaluates its efficacy in patients with *Parinama Sula* vis-à-vis duodenal ulcer disease.

Satavari caused a significant improvement in various symptoms particularly pain, heartburn and sour eructations, in most of the patients usually within the first week of initiation of therapy. Maximum relief was observed in pain of 'burning' nature. There was found a considerable reduction in the consumption of antacids or antispasmodic drugs. Certain other non-specific dyspeptic symptoms like nausea, belching, flatulence and constipation were relieved significantly. Gupta et al, (1982) in a comparative study

reported symptomatic improvement with *Satavari* in 75 per cent patients with duodenal ulcer; that was some what more than that observed with *Amalaki* (in only 47 per cent cases) or antacids (in only 54 per cent cases).

Satavari induced significant reduction in basal, maximum and peak acid outputs. Mean per cent reduction was found maximum in BAO (48.67±5.21) and minimum in MAO (37.90±7.15). Almost similar reduction has been reported by Gupta et al, (1982) in BAO (49 per cent) and by Mishra et al, (1985) in MAO (36 per cent). However, Mishra et al, (1985) observed much more reduction in BAO (59 per cent) and Gupta et al, (1982) in MAO (47 per cent). Average mean per cent reduction in this AHT series of the present study was 42.07±3.34. Ratio of basal to maximum acid output reduced to 32 per cent from 39 per cent, the difference, however, being insignificant. This ratio remains below 20 per cent in normals and from 20 to 40 per cent in patients with duodenal ulcer. Statistically significant reduction in acid values was also observed in series of fractional test meal; however, the mean reduction was only 32 per cent. Conventional anticholinergics inhibit acid secretion markedly. Pirenzipine with cimetidine can inhibit acid secretion completely. H_2 blockers such as cimetidine or ranitidine inhibit basal and nocturnal acid secretion completely, while other acid outputs by 70 per cent. But *Satavari* was found to inhibit basal output by only 48 per cent, histamine induced maximum output by 38 per cent and alcohol meal induced secretion by only 32 per cent in patients with duodenal ulcer. Moreover, the ratio of BAO to MAO was neither reduced significantly nor upto the normal range. It appears, therefore, that *Satavari* may not have antisecretory property. The observed mild reduction in acid secretion may be due to some change in gastric mucosa, might be a decrease in parietal cell mass. Moreover, in vitro experimental study, *Satavari* did not exhibit even any trace of antacid activity (Singh et al, 1985).

Radiologic evaluation exhibited complete recovery in 44 per cent patients and partial in 28 per cent; rest 28 per cent had no change. Maximum improvement was observed in patients having ulcer grater, and least change in cases having duodenal bulb deformity. Gupta et al, (1982) observed complete recovery in 50 per cent, and partial in 25 per cent patients. However, Mishra et al, (1985) have reported radiological improvement in all *Satavari* treated duodenal ulcer patients and 83% of them got complete recovery, however, the number of studied cases was only six, and moreover, endoscopic healing was not observed in all cases, complete healing was reported in 50 per cent and partial in 33 per cent patients of that series. Similarly, endoscopic evaluation exhibited complete healing in 50 per cent and partial healing in 25 per cent patients, observations being identical to the study of Gupta et al, (1982). Failure rate of radiologic and endoscopic improvement in our study were observed in 28 per cent and 25 per cent patients respectively.

Observations, thus, made in this study suggest that *Satavari* has neither potent antisecretory activity nor antacid property. Nevertheless, *Satavari* relieves symptoms particularly pain significantly within few days of initiation of therapy, and heals the duodenal ulcer in an average period of six weeks as evidenced by radiologic as well as endoscopic improvement in approximately 75 per cent patients. *Satavari* appears to heal the ulcers by potentiating defensive factors. Hitherto, more emphasis had been given on aggressive factors but investigative follow-up study has changed the view, and now,

defensive mechanisms are being studied enthusiastically. Moreover, this phenomenon of changing view is being reflected in the management of duodenal ulcer. Quiescence of offensive acid either by chemical or physical neutralization in the stomach (antacids) or by antisecretory agents (anticholinergics or H_2 blockers) has offered only transient healing of the hole without modifying the course of duodenal ulcer diathesis. Moreover, this mode of therapy may provide sometimes potential toxicity. Therefore, present day gastro-enterologists are becoming more interested in strengthening the defensive mechanism of gastroduodenal mucosa with various newly developed drugs such as carbenoxolone, gefarnate, sucralfate, tripotassium dicitrat bismuthate (TDB) and certain other cytoprotective agents like prostaglandin (PGE_2).

Satavari may strengthen the mucosal resistance and thus, reduces H+ ion back diffusion. It may prolong the life span of mucosal cells, may increase the secretion and viscosity of mucus may strengthen the mucosal barrier, thereby reducing H+ ion back diffusion into mucosa, like carbenoxolone, a derivative of an Ayurvedic plant drug well known for the treatment of *Parinama Sula* namely *Madhuyasthi*. *Satavari* has been found to maintain the continuity and thickness of aspirin treated gastric mucosa with a significant increase in mucosal mucin (Mishra et al, 1985). Secondly, *Satavari* may form a complex with mucus of other substances at the base of ulcer which may protect the ulcer form the corrosive and proteolytic effects of acid-pepsin, like colloidal bismuth. Furthermore, as *Satavari* heals the duodenal ulcer without inhibiting the acid secretion, it may have cytoprotective action like that of prostaglandins. Other possible mechanisms may be deactivation and binding of pepsin or binding of bile salts. It warrants further investigation to evaluate the mechanism of ulcer healing effect of *Satavari*.

None of the patient complained of adverse effects. In fact, *Satavari*, a plant medicine contrary to toxic effects of modern chemical drugs exerts many beneficial effects on general body system, such as delayed aging and increased longevity and virility owing to its *Rasayana*, *Vajikarana* and *Balya* effects, and appears absolutely safe for long term use even during pregnancy (*Astanga-Hrdaya*, Sa. 2: 547) and lactation.

References:

Vagbhata	1982	Astanga Hrdayam (Vagbhata Tantra), Sarvanga Sundari (Aruna Dutta) and Ayurveda Rasayana (Hemadri; Chaukhamba Sanskrit series office, Varanasi, Reprinted, Sarira, 2:54.
Cakrapanidutta	1976	Cakradutta (Cakrapanidutta Viracita), Bhavartha Sandipani. Chaukhamba Sanskrit Series Office, Varanasi, Ed. IV, 26:28, 27:35-37, 49-53.
Chaturvedi, G.N.; Mahadeva Prasad; Agrawal, A.K. and Gupta J.P.	1979	Some clinical and experimental studies on whole root of Glycyrrhiza glabra Linn. (Yastimadhu) in peptic ulcer. Ind. Med. Gaz. CXaIII (6): 200-5.
Chaturvedi, G.N.; Mahadeo Prasad; Rai, N.P. and Tiwari, S.K.	1979	Certain studies on non-ulcer and ulcer dyspepsia-An Ayurvedic correlation. Nagarjuna, XIII (i): 1-3.

Gupta, V.K.; Jain, A.K.; Gupta, J.P.; Chaturvedi, G.N. and Udupa, K.N.	1982	Evaluation of indigenous drugs in management of peptic ulcer. M.D.(General Medicine) Thesis, I.M.S., BHU, Varanasi.
Kay, A.W.	1953	Effect of large doses of histamine on gastric secretion of HCl. An augmented histamine test. Br. Med. J. 51: 469-81.
Kolmer, J.A.; Spaulding, E.H. and Robinson, H.W.	1969	Fraction gastric analysis, In Apporved Laboratory Technique, Scientific Book Agency, 22 Raja Woodmunt Street, Calcutta, pp. 211-21.
Maheshwari, C.M. and Chaturvedi, G.N.	1977	Clinical correlation of Parinama-shoola and its treatment with an indigenous drug Satavari (Asparagus recemosus). M.D. (Ay.) Thesis, Department of Kayachikitsa, I.M.S., B.H.U., Varanasi.
Mishra, A., Tripathi, S.N., Gupta, J.P. et al.	1985	Peptic ulcer-an experimental and clinical study; Ph.D. Thesis, Zoology-Kayachikitsa, B.H.U., Varanasi.
Sarangadhar	1981	Sarngadhar Samhita, Hindi commentary, Chaukhamba Amar-Bharati Prakashana, Ed. VI, Ma. 1: 15, Varanasi.
Singh, K.P.; Singh, R.H. and Gupta, J.P.	1985	Role of certain indigenous drugs and diets in peptic ulcer (Parinama-Sula) and in gastric secretion, Ph.D. Thesis, Kayachikitsa, I.M.S., B.H.U., Varanasi.
Varma, M.D.; Singh, R.H.; Gupta, J.P. and Udupa, K.N.	1977	Amalaki Rasayana in the treatment of peptic ulcer. Jour. Res. Ind. Med. Yoga. Homeo. 12(4): 19.

हिन्दी सारांश

परिणामशूल में शतावरी (एस्पैरागस रैसोमोसस) के प्रभाव का आतुरोय अध्ययन

के.पी.सिंह एवं आर. एच. सिंह

परिणामशूल (ड्यूओडिनल अल्सर डिजीज) के ३२ रोगियों में शतावरी के प्रभाव का आतुरीय अध्ययन किया गया। १८.७५ प्रतिशत रोगियों ने अन्त तक औषधि का सेवन नहीं किया। शतावरी मूल चूर्ण (१२.० ग्राम प्रति दिन चार समान मात्राओं में) लगभग ६ सप्ताह तक दिया गया। आमाशयगत अम्लता ह्रास, विकिरण विज्ञानीय गृहयान्तदर्शी सुधार आदि एवं मापदण्डों के आधार पर औषधि के प्रभाव का निर्णय किया गया।

शतावरी मूल चूर्ण से प्रारम्भ से ही सभी रोगियों के अधिकतर लक्षणों में महत्वपूर्ण सुधार हुआ। शतावरी में अम्लविरुद्ध (एन्टासिड) प्रभाव नहीं पाया गया। शतावरी में तीव्र स्राव रोधी (एन्टीसिक्रेटरी) प्रभाव भी नहीं प्रतीत होता है क्योंकि इसके द्वारा बेसल अम्ल स्राव में केवल ४८ प्रतिशत, हिस्टामिनजनित स्राव में ३८ प्रतिशत एवं अल्कोहलजनित स्राव में मात्र ३२ प्रतिशत ह्रास हुआ। विकिरण विज्ञानीय अध्ययन एवं गृहयान्तदर्शी अवलोकन से लगभग तीन चौथाई रोगियों में सुधार पाया गया। संभवतः शतावरी का व्रण रोपण प्रभाव, उसके अपने प्रत्यक्ष व्रणरोपण प्रभाव, तथा श्लैष्मिक कला अवरोध या कोशकीय संरक्षा को बल प्रदान करने से होता है।