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An Official Journal of

BHARATIYA SANGYAHARAK ASSOCIATION

(Association of Anaesthetists of Indian Medicine)

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EDITORIAL

Ayurved in 21st Century: My Dream

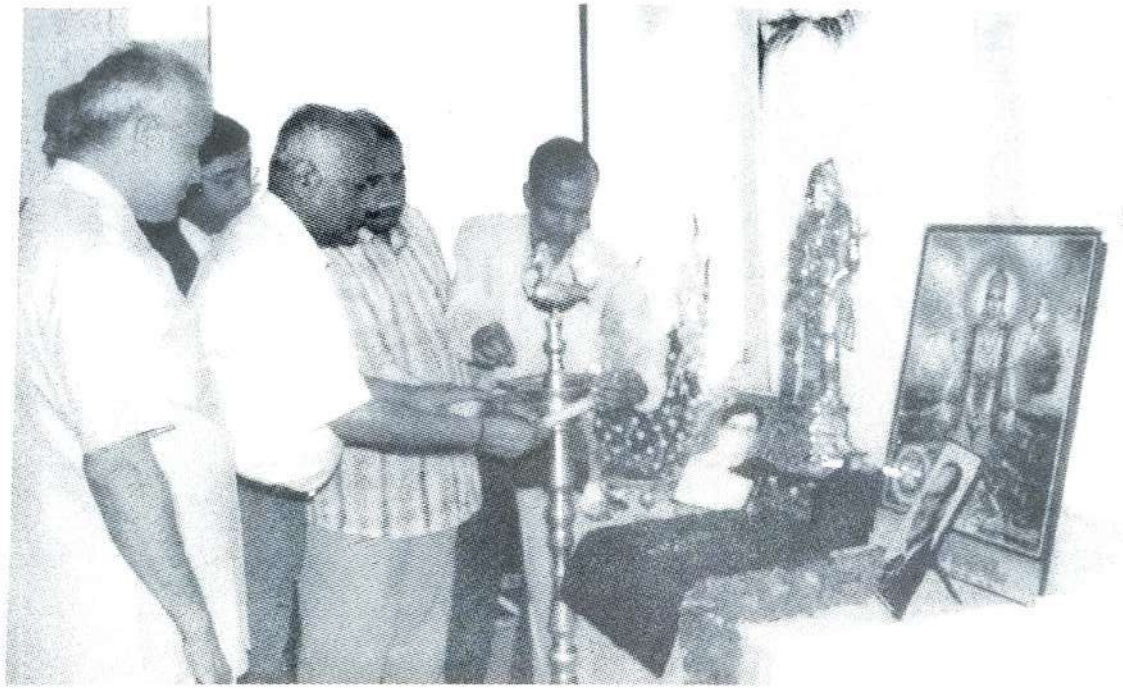
Ayurved- the science of life-the oldest medical system in world, proved as a first systematic presentation of diseases and their cure with a special attention to preventive measures. The principle and observations made on the basis of personal experiences during thousands of years are tested and are never failed. But it does not mean that it closes the possibility to explore and include newer principles, approaches, procedures or drugs. The investigators/ the inventors of Ayurvedic medical system accepted a principle of continuing medical education where is a definite chance of discussion and to search out newer remedies / procedures and their applications. It is why after all the sabotage against this system during Mughal and British period, the system remain in existence. Keeping in view these foresightedness of our Acharya we should also try to explore the possibilities to grow and to develop this system as per need of the time and society. We should not take it as there is no scope for further branching or incorporating the newer researches. Researches are being done with motive to help the humanity not to any single cost /Race/Community or Country. Therefore every research/development carried out all over the world are property of the humanity and this should be utilized for betterment of the humanity without any compartmentalize. Keeping in view this ideology we should be prepare to incorporate all the development occurred during 20th century in the medical sciences in our own medical system- **Ayurved**, so that it can provide **health for all** with full efficacy.

Branching is earnestly required to fulfill the demand of the present day hurried worried peoples. Astang Ayurved is now divided into 22 specialty but still there is scope to spread. I am really thankful to the C.C.I.M. which had taken a positive approach to strengthen Ayurved by updating the syllabus for U.G. & P.G. both with incorporating several new disciplines in P.G. courses. This is a sign of development and progress. One step further C.C.I.M. is going to introduce 2 years P.G. Diploma courses in several disciplines. This will also serve our goal- **Health for all**. Many specialist will be ready to serve the society by their specific skill in these branches. It will also serve the other specialty branches which were defunct in absence of these specialties like Sangyaharan and Vikiran. I hope that in 21st century our course curriculum will be so advance and updated that our own postgraduate will take up the most super specialized field like Neurology/Neurosurgery/Cardiothoracic surgery etc. We have to strengthen Ayurveda in such a way that our country would have only onee system -Ayurved. All the other system would be incorporated in Ayurved. It does not mean that Ayurved will disappear but it will remain alive with its own holistic approach.

Jai Hind **Jai Ayurved** **Jai Sangyaharan**

Devendra Nath Pande
Chief Editor

Glimpses of 12th National Conference of A.A.I.M.
Rajeev Gandhi Post Graduate Ay. College, Paprola, Kangra



Inaugurated by Hon'ble Dr. S.K. Sharma, Advisor, AYUSH

Lamp Lighting - Dr. S.K. Sharma accompany with Dr. K.K. Pandey, Dr. A. Dutt & Dr. S. Sharma

Presentation of Memento to the Chief Guest – Dr. S.K. Sharma

Presentation of Ashwinau Award to Dr. Anil Dutt

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2nd International Congress of

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Last Date for Submission of Abstract: 31/12/2009

Effects of an Herbal Drug Compound (Dashmula Ghanvati) on Oxygen Saturation- During Anesthesia

* Pande D.N. ** Pandey K.K. *** MishraY.K.

Abstract:

The resultant of surgical trauma, anesthetic drugs and pain creates a lot of Physiological alterations during anesthesia.. Pain being subjectiv phenomena- it becomes more difficult to monitor the damage due to it and to manage it in patients. No doubts there is a list of large number of synthetic and semi-synthetic preparation of analgesic drugs to pacify the pain during post operative period, but till date none of these has been proved to be devoid of their untoward effects. Hence a through search was made to evaluate some of the indigenous drugs possessing analgesics and anti-inflammatory properties, (Vedanahara, Shothahara) mentioned in text of Ayurveda.

A clinical trial was carried out on Dashamula, prepared in the form of dried decoction tablet (Ghanvati) and compared with well known analgesic drug Diclofenac Sodium . During the study the Oxygen saturation with other parameters were assessed.

Keywords: Analgesics, Vedanahara, Sothahar, Vatashamak, Damula Ghanvati, Post operative, MBP = Mean blood pressure,$EtCO_2$ = End Tidal CO_2, PR = Pulse rate, SPO_2 = Oxygen saturation

INTRODUCTION

Literary evidences reveal that people in ancient days were quite conversant with pain relieving drugs. Acharya Sushruta has mentioned the use of medicated alcohol (Madya) before surgical procedure and during delivery to relieve pain and allaying apprehension. Pain is the basic and most challenging problem for surgeons from primitive era.

The primary requirement of safe and satisfactory surgery is to abolish the pain during operation. Keeping these views we have evaluated the efficacy of herbal drug compound Dasamula Ghanvati as anti-inflammatory and analgesic activity.

A clinical trial was carried out on Dasamula, prepared in the form of dried decoction tablet (Ghanvati) and compared with well known analgesic drug Diclofenac Sodium During the study the Oxygen saturation with other parameters were assessed.

No. of patients = 40

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MATERIAL AND METHODS

Operation - Elective surgery below umbilicus except GIT surgery.
Anesthesia - Lumber subarachnoid block with Bupivacaine 0.5% heavy

OBSERVATIONS AND RESULTS

1. Groping of the Patient:

Table No. 1:

Trial group -I (Dasmula Ghanvati)	No. of Patient - 20	Premedication - Tab. Dasamula Ghanvati 1 gm at 10 P.M. of previous night and 2 hours before operation with sips of plain water orally and Injection Glycopyrolate 0.2 mg IM 1 hours before Operation.
Control group -II (Diclofenac Sodium)	No. of Patient - 20	Premedication - Tab. Diclofenac sodium 50 mg at 10 P.M. of previous night and 2 hours before operation with sips of plain water orally and Injection Glycopyrolate 0.2 mg IM 1 hours before Operation.

2. AGE, WEIGHT AND HEIGHT

Table 2:

The statistical comparison of mean age, mean weight and mean height between the groups.Group		Age (years) Mean ± SD	Weight (Kg) Mean ± SD	Height (cm) Mean ± SD
Group I / Trial		41.75 ± 11.79	59.10 ± 8.48	164.80 ± 3.82
Group II / Control		40.00 ± 14.05	58.00 ± 7.65	164.60 ± 4.76
Comparison between groups unpaired 't' test	t value	t = 0.43	t = 0.43	t = 0.15
	p-value	p > 0.05	P > 0.05	P > 0.05
Remark		NS	NS	NS

It is obvious from the above table that mean age, weight and height are statistically comparable and identical (p > 0.05) in the patients of both the groups.

3.EFFECT ON RESPIRATORY RATE

Table 3A: The statistical comparison of mean respiratory rate per minute changes before premedication (A), after premedication (B), during subsequent anaesthesia(C) and after recovery from anaesthesia (D) between the two groups at corresponding time by applying student t-test and p-values and remarks are as follows.

Group		Mean Respiratory Rate/min; (Mean ± SD)			
		Before premedication (A)	After premedication (B)	During subsequent anaesthesia (C)	After recovery from anaesthesia (D)
Group I (Trial)		17.45 ± 1.93	17.20 ± 2.06	17.90 ± 3.02	17.00 ± 1.34
Group II (Control)		19.90 ± 2.20	16.75 ± 2.34	17.56 ± 3.34	16.95 ± 1.53
Comparison between groups unpaired 't' test	t value	t = 0.84	t = 0.64	t = 0.34	t = 0.11
	p-value	p > 0.05	p > 0.05	p > 0.05	P > 0.05
Remark		NS	NS	NS	NS

From Table 3A, it is observed that difference of mean respiratory rate per minute when compared in between group-I and group-II at corresponding four different timings, it is statistically insignificant and identical.

Table 3b.: Comparison within the groups:

Comparison within the groups	Group I			Group II		
	Mean ± SD	t-value p-value	Remark	Mean ± SD	t-value p-value	Remark
A vs. B	0.25± 1.33	t = 0.84,p >0.05	NS	0.15± 1.95	t = 0.34,p > 0.05	NS
A vs. C	-0.45± 2.06	t = 0.97p > 0.05	NS	-0.65± 2.39	t = 1.22,p > 0.05	NS
A vs. D	0.45 ± 1.54	t = 1.31 p > 0.05	NS	-0.05 ± 2.01	t = 0.11 p > 0.05	NS

From Table 3B, it is observed that changes in respiratory rate are insignificant in both groups at the levels of before premedication vs after premedication, before premedication vs during subsequent anaesthesia and before premedication vs after recovery from anaesthesia

4. EFFECT ON OXYGEN SATURATION

Table 4A: Mean oxygen saturation

		Mean oxygen saturation (%) (Mean ± SD)			
		Before premedication (A)	After premedication (B)	During subsequent anaesthesia (C)	After recovery from anaesthesia (D)
Group I (Trial)		97.85 ± 0.93	97.70 ± 0.73	97.75 ± 0.94	98.00 ± 0.64
Group II (Control)		97.65 ± 0.93	97.40 ± 0.75	97.65 ± 1.04	97.80 ± 0.69
Comparison between group unpaired 't' test	t value	t = 0.68	t = 0.22	t = 0.16	t = 0.94
	p-value	p > 0.05	p > 0.05	p > 0.05	P > 0.05
Remark		NS	NS	NS	NS

From Table 4A, it is observed that difference of mean SPO2 percentage when compared between group-I and group-II at corresponding four different timings it is insignificant.

Table 4B: Statistical comparison of difference in the mean SPO2 percentage before premedication (A), after premedication (B), during subsequent anaesthesia (C), and after recovery from anaesthesia (D), within the groups by applying paired t-test, p-values and remarks are as follows

Comparison	Group I			Group II		
	Mean ±	t-value		Mean ±	t-value	

Table 5A: The statistical comparison of difference of mean $ETCO_2$ in mmHg

Group		Mean ETCO2(mmHg); (Mean ± SD)			
		Before premedication (A)	After premedication (B)	During subsequent anaesthesia (C)	After recovery from anaesthesia (D)
Group I (Trial)		33.25 ± 2.67	33.30 ± 2.92	33.05 ± 3.06	33.30 ± 2.31
Group II (Control)		33.85 ± 2.20	33.70 ± 2.17	33.65 ± 2.64	34.25 ± 1.88
Comparison between groups unpaired 't' test	t value	t = 0.77	t = 0.49	t = 0.66	t = 1.42
	p-value	p > 0.05	p > 0.05	p > 0.05	P > 0.05
Remark		NS	NS	NS	NS

From Table 5A, it is observed that difference of mean $ETCO_2$ (mmHg) when compared in between group-I and group-II at corresponding four different timings it is insignificant.

Table 5B:-Statistical comparison of difference in the mean $ETCO_2$ (mmHg)

Comparison within the groups	Group I			Group II		
	Mean ± SD	t-value p-value	Remark	Mean ± SD	t-value p-value	Remark
A vs. B	0.15 ± 0.81	t = 0.82 p > 0.05	NS	-0.05 ± 0.94	t = 0.24 p > 0.05	NS
A vs. C	0.20 ±1 .36	t = 0.66 p > 0.05	NS	0.20 ± 1.47	t = 0.61 p > 0.05	NS
A vs. D	-0.40 ± 2.56	t = 0.79 p > 0.05	NS	-0.05 ± 1.39	t = 0.16 p > 0.05	NS

From Table 5B, It is observed that difference of mean $ETCO_2$ (mmHg), at the level of before premedication and after premedication and before premedication, during subsequent anaesthesia and before premedication and after recovery from anaesthesia is insignificant in group I and group-II.

6. DESIRABLE EFFECTS AND UNDESIRABLE EFFECTS

Table 6: Incidence of desirable effects and undesirable effects in patients of both groups after premedication.

Effects	Incidence	Group-I		Group-II		Z-value between Group-I vs. Group-II	Remarks
		No.	%	No.	%		
Sedation	Present	0	0	0	0	0	NS
	Absent	20	100	20	100		
Apprehension	Present	3	15	5	25	z=0.79 p > 0.05	NS
	Absent	17	85	15	75		
Excitement	Present	0	0	0	0	0	NS
	Absent	20	100	20	100		
Dizziness	Present	0	0	0	0	0	NS
	Absent	20	100	20	100		
Nausea	Present	0	0	0	0	0	NS
	Absent	20	100	20	100		
Vomiting	Present	0	0	0	0	0	NS
	Absent	20	100	20	100		

Z-value is two proportions form independent groups.

Z value is calculated by

The comparison between the group-I and group-II regarding sedation, apprehension and excitement is statistically insignificant.

The statistical comparison of undesirable effects like dizziness, nausea, vomiting, in between group-I and group-II at the level of after premedication is insignificant.

7. POST ANAESTHETIC SEQUEL

Table 7: The incidence of post-anesthetic sequel observed between Group I and Group **II**

Side Effects	Incidence	Group-I		Group-II		Z-value between Group-I vs. Group-II	Remarks
		No.	%	No.	%		
Sedation	Present	0	0	0	0	0	NS
	Absent	20	100	20	100		
Nausea	Present	1	5	2	10	Z = 0.60 p > 0.05	NS
	Absent	19	95	18	90		
Vomiting	Present	0	0	0	0	0	NS
	Absent	20	100	20	100		
Dizziness	Present	0	0	0	0	0	NS
	Absent	20	100	20	100		
Dyspepsia	Present	0	0	0	0	0	NS
	Absent	20	100	20	100		
Gastric Irritation	Present	0	0	0	0	0	NS
	Absent	20	100	20	100		
Increased Peristalsis	Present	0	0	0	0	0	NS
	Absent	20	100	20	100		
Haematemesis	Present	0	0	0	0	0	NS
	Absent	20	100	20	100		
Malena	Present	0	0	0	0	0	NS
	Absent	20	100	20	100		
Precipitation of Asthma	Present	0	0	0	0	0	NS
	Absent	20	100	20	100		
Respiratory depression	Present	0	0	0	0	0	NS
	Absent	20	100	20	100		
Headache	Present	2	10	1	5	Z = 0.60 p > 0.05	NS
	Absent	18	90	19	95		
Backache	Present	3	15	3	15	Z=0	NS
	Absent	17	85	17	85		

Nausea: Incidence of nausea in group I (trial) was 5% and in group II (control) was 10% which is also statistically insignificant.

Headache: Incidence of headache in group-I was 10% and in group-II it was 5%. On statistical comparison, incidence of headache is insignificant.

Backache: Incidence of backache was 3 in group-I, i.e. 15% and in group-II it was i.e. 15%.Statistically they are identical.

Sedation, vomiting, dizziness, dyspepsia, gastric irritation, increased peristalsis, haematemesis, malena, precipitation of asthma, respiratory depression and other side effects were noted meticulously in both groups and was found to be absent in all the groups.

REQUIREMENT TIME OF 1ST DOSE OF ANALGESIC

Table 8: The mean of the 1st analgesic dose requirement time (in minutes) of all patients in group-I and group-II were recorded and statistically compared.

Groups	Mean ± SD	t-value	p-value	Remark
Trial Group –I	**218.75 ± 29.77**	**t = 1.51**	**p > 0.05**	**NS**
Control Group – II	**202.200± 38.93**			

It is obvious from the above table that requirement of the first dose analgesic time in patients of both the groups was almost equal and identical time intervals the statistical comparison of first dose analgesic requirement time between the groups is insignificant .

CONCLUSION

On the basis of observations made on 20 patients in each groups, it can be concluded that in these groups:

The trial drug Dasamula Ghanvati has Shothahar (anti-inflammatory) and Vedanahar (analgesic) properties.

The trial drug Dasamula Ghanvati (1000 mg) is almost equally effective as anti-inflammatory and analgesic in comparison to control drug Diclofenac sodium (50 mg).

No alteration was seen ON OXYGEN SATURATION

No alteration was seen ON END TIDAL CARBON DIOXIDE ($ETCO_2$)

Thus the herbal compound can be used safely during anesthesia(under regional block)

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THE NEWS

Date	Venue	Conference	Contact
6th to 8th Feb. 2010	K.N. Udupa Auditorium, IMS, BHU, Varanasi	**13th National and 2nd International Conference of** *Association of Anesthesiologists of Indian Medicine*	The Organizing Secretary, AAIMCON-2010 Section of Sangyaharan,Department of Shalya Tantra, Institute of Medical Sciences, Banaras Hindu University Varanasi – 221005, **Tel.: 0542 (O) 6702194, (R) 2575092 Mobile No. 09415256461 Telefax : 91 - 542 - 367568, E-mail: dnpande@gmail.com** Web-www.aaim.synthasite.com
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5th to 7th Oct. 2009	Bangalore India	3rd Annual TEE Workshop under the Aegis of IACTA	Organizing Secretary Dr. Muralidhar. K Tel: 080-27835000 to 27835018 Fax: 080-27835222/27832648 E-mail: kanchirulestheworld@gmail.com / kanchi_rules_300a@lycos.com
9th to 11th Oct. 2009	Delhi Cant – 110010 INDIA	Annual National Conference of Research Society of Anesthesiology Clinical Pharmacology (RSACPCON-2009)	Organizing Secretary Col. (Dr.) Mukul Kapoor Tel: +91-11-23338260, 23338257 Mob: +91-9971888773, 9818779937 E-mail: rsacp2009@gmail.com
20th to 23rd Nov. 2009	Brisbane AUSTRALIA	ASURA 2009 Anstralasian Symposium on Ultrasound & Regional Anaesthesia	Cassandra Hargreaves Tel: +61 (O) 29302 2709 e-mail: chargreaves@fediasa.org.an Web: www.asura2009.org.an
25th to 29th Dec. 2009	Chennai INDIA	57th Annual National Conference of ISA ISACON 2009	Organizing Secretary Dr. Ganapathy Asokan e-mail isacon2009@hotmaol.com Web: www.isacon2009chennai.com

Endometriosis: An Agony

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Abstract- Endometriosis is the disease affecting women world wide, is increasing cause of concern. The name comes from the word 'endometrium' which is the tissue that lines the inside of the uterus and is build up and sloughed off each month during the menstrual cycle. Common locations of endometriosis growths are ovaries, fallopian tubes, outer surface of uterus, lining of the pelvic cavity, cervix, vagina, rectovaginal septum. Endometrial growths are generally not malignant. The most common symptoms of endometriosis includes heavy or irregular bleeding, painful intercourse, chronic pelvic pain, pain before and during periods, painful urination or bowel movements, repeated abortion and infertility. The exact etiology is still unknown but some common theories are described in modern texts. There is need to bring the ayurvedic treatment into limelight for endometriosis because the allopathic treatment limitations and many side effects. Ayurveda can give a better solution for endometriosis through a varied range of treatments which does not pass any hazards to patient. Panchkarma specially vasti and virechan are best known treatment for it along with other ayurvedic formulation/herbs.

Keywords- Endometrium, Vasti, Virechan, Yonivyapad, dosha

Introduction- Endometriosis is defined as the presence of functioning uterine glands and stroma in any site outside the uterine cavity[1]. It is a complex and painful disease affecting women in the reproductive year. The termed endometriosis was first coined by Sampson (1921) when he 'discovered' some chocolate cyst of the ovary[2]. The name of comes from the word 'Endometrium' which the tissue that lines the inside of uterus and is build up and sloughed of each month during menstrual cycle. In endometriosis, the endometrial tissue develops into what are called growth, implants, nodules, lesion or tumors[3].

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The most common sites for endometriosis are in abdomen involving ovaries[4] the fallopian tubes, outer surface of uterus, the bladder and the lining of pelvic cavity[3]. Some times bowel may be involve along with other side of pelvis. The rectum is involved at rectovaginal septum[4]. Some uncommon lesions in the cervix and vagina bluish in color and cystic also found[4]. Rarely endometriosis growth also have been found in urinary tract and umbilical scars, lungs, arms and thigh[3]. Endometrial lesion first appear as clear vesicles, then become red and progress to black over a period of 7-10 years. Clear lesions are seen at average age of 21.5 while black lesion is seen at 31.9yers. This relationship between age and colour of lesions confirms the progressive nature of disease. In 47-64% of woman, this disease will progress without therapy[5]. Active endometriosis is seen most commonly between the ages 30 and 40 years. It can however occur at any time between menarche and menopause even before the age of 20 years[6].

Most common symptoms of endometriosis includes chronic pelvic pain, pain before and during periods, painful intercourse, heavy or irregular bleeding, repeated miscarriage and infertility[6].Natures of pain depends largely on site and extent of lesion[4]. If the uterosacral ligaments are involved then the chronic pelvic pain on one side is found[6]. The pain may also felt in the rectum, perineum or vagina. Typically the dysmenorrheal begins a few days prior to menstruation and continues throughout period. There may be associated menorrahagia due to adenomyosis or if ovarian function is altered. Due to bilateral endometriomas irregular menstruation also result[4]. In a survey, 20-29 years old women being treated for endometriosis, 90% reported menstrual pain, 80% infertility, 71% pelvic pain and 46% menstrual irregularities[7]. Endometriosis can be categorized in four stages based on the area of adhesions

Stage I (mild) =1 -5 cm, Stage II (moderate) = 6- 15cm, Stage III (severe) =16 - 30cm, Stage IV (extensive) = 31- 54 cm (American Fertility Societies Scoring System).

Aetiopathogenesis- There has been numerous theories explain the occurrence of endometriosis, though exact etiology is not known. Endometriosis can occur due to retrograde menstruation[8], narrow cervics favoring ante grade menstruation or immunological factors[9]. Women who have not a full term pregnancy are more likely to have endometriosis because apparently, the longer a women does not have a child, the more estrogen she is exposed to during her life time[9]. Environmental toxins and use of alcohol is also liable for development of endometriosis[10]. A study shows that women who drink alcohol have a 50% higher risk of developing endometriosis than those who abstain[11]. Endometrial tissue get implanted else where through retrograde menstruation, coelomic metaplasia, direct implantation, lymphatic or vascular routes. This ectopic endometrium has got potentiality to undergo changes under the action ovarian hormones. Proliferative changes are constantly seen but the secretory changes are absent due to deficiency of steroid receptor in the ectopic endometrium. Cyclic growth and shedding continue till menopause. The periodically shed blood may remain encysted and the cyst may some time become tense and ruptures. As the blood is an irritant. there is dense tissue reaction surrounding the lesion with fibrosis.

Diagnosis- Abdominal palpitation may not reveal any abnormalities. A mass may be felt in lower abdomen in the case of enlarge chocolate cyst, due to endometriosis adhesion, the mass is tender with restricted mobility. Speculum examination reveals vaginal/cervical deposits which are bluish in color and tender in touch. Bimanual examination reveals nodular and tender uterosacral ligaments. Uterine mobility is restricted and it may be fixed in retroversion. Progressively increasing secondary dysmenorrheal, dyspareunia, infertility are some classical clinical symptoms for endometriosis.

Endometriosis should be suspected in women with subfertility, dysmenorrhoea, dyspareunia or chronic pelvic pain. Surprisingly, there may be no symptoms even when the endometriosis is widespread and advanced. In adult woman dysmenorrohea may be especially suggestive of endometriosis, if it begins after years of pain free menses. The dysmenorrheal often starts before the onset of menstrual bleeding and continues through out the menstrual periods. The distribution of pain is bilateral. Local symptoms can arise from rectal, ureter and bladder involvement. The infertility is caused due to involvement of the ovaries and causing adhesion that block tubo-ovarian motility and ovum pick up. Diagnosis of endometriosis is considered uncertain until proven by laproscopy[5]. Ovarian cancer some times has the same symptoms as of endometriosis and treatment with hormones particularly with estrogen, could cause a cancer grow faster[5]. Ca 125 is a cellular protein found in pelvic organs that appears to be elevated in cases of moderate to severe endometriosis[12]..

Ayurvedic concept- In ayurveda disorders of female reproductive system are explained in the classics under the entity of 'yoni vyapad' and 'artav vyapad'. The world 'yoni' refers to the structural aspects i.e. organs of female reproductive system all together and world artav refers to the functional aspect of female reproductive system. Among the 'vimshati yoni vyapad' and 'asthartava vyapad' described in all the classics, not any single entity equated with endometriosis. Vatic disorder has qualities like roughness, instability, dislocation, division, attachment and piercing pain (C.Su. 20/3,4)[13]. Among these qualities dislocation (dislocation of cells from inside to outside of endometrium), attachment (of ectopic endometrial cells to other organs) and piercing pain are the main characteristic of endometriosis. Haemorrahagic patches and thickening of skin are paittik disorder. Chronic nature of endometriosis refers to kaphaja involvement. Based on the classical symptoms of endometriosis i.e dysmenorrhoea, infertility and dyspareunia, some of the yoni vyapad and artav vyapad can be considered in a group as the disease endometriosis. Udavartini (dysmenorrhoea), paripluta (dyspareunia), vatala yoni vyapad (chronic pelvic pain) and other condition like vandyatva (infertility), antarmukhi yoni vyapad (excessive ante version causing ante grade menstruation) in a group can be considered as endometriosis.

Udavartini- Due to movement of natural urges in reverse direction the apan vayu moving in reverse direction fills yoni (uterus). This yoni seized with pain, initially throws raja upwards and then discharges it with great difficulty. The lady feels immediately relief following discharge of menstrual blood. This condition is also characterized by painful frothy menstruation, general malaise and discharge of clotted blood (C.Chi.30/25,26[13]; S.S.U.39/11[14])

Vatala yoni vyapad- Women of vata prakruti when consume diet and indulge in other activity capable of aggravating vata. It gets provoked and reaches yoni and produces pricking

pain stiffness, roughness, numbness and fatigue. Chakrapani says that the painful frothy and thin menstrual blood occurs even in inter menstrual period. This condition is characterized with pain which is more in magnitude. (C.Chi.30/11[13], S.S.U.38/11[14])

Paripluta yoni vyapad- A woman having predominance of pitta withholds her natural urges at the time of coitus, the vitiated pitta, getting attached with vayu reaches yoni and produces its abnormalities. The yoni becomes inflammed and she gets painful menstruation. She also suffers from pain in lumbo sacral and groin region. This condition is also characterized with severe dyspareunia.(C.Chi.30/23,24)[13].

Antarmukhi yoni vyapad- When a woman after meal indulge in coitus, sleeping in abnormal posture, then the vayu situated in her yoni getting pressed by food produces different types of pains .There is dyspareunia also (C.Chi.30/29-31[13], A.S.U.38/39[15], A.H.U.33/35,36[16]).

Bandhyatva - In Harita classification of bandhyatva, kakvandhya (one child sterility), anapatya (primary infertility) and garbhsravi (repeated abortion) having similarities with endometriosis (H.S.Tri.48/1[17]) because endometriosis may result in infertility.

According to Acharya Charak in classification of infertility **saparja** and **apraja** also show similarity towards endometriosis (C.Sh.2/5)[13].

Yonikand- In vataj yonikand mass is rough, discoloured and fissured, pittaj type of yonikand is red coloured, accompanied with fever and burning sensation, blue and resembling flower of linseed and having itching in that, is of kapha origin (M.N. 62/3,4)[18]. These masses or nodules have resemblance with endometriotic lesions.

Asrigdara- General symptoms of asrigdar includes excessive bleeding during menstrual or inter menstrual period (A.S.Sh. 1/11)[15]. Pain and bodyache are common features in all type of Asrigdar (M.N.61/2[18], B.P.Chi.68/3[19]). Dalhan has described clinical features of asrigdara as burning sensation in lower portion of groin, pelvic region, back, kidney and flank region, and severe pain in uterus (S.S.U.45/44)[14].

Treatment of endometriosis- The medical treatment of endometriosis is aimed as controlling the pain and/or shrinking the endometrial tissue[20]. Long term use of low estrogen, high progestin birth control pills are recommended in women who are not trying to get pregnant[12]. Danazol is another powerful drug, works by reducing FSH and LH levels and is taken for 6-9 months at a time[21]. However women who use it may have serious side effects including pseudo-menopause, hot flushes, vaginal dryness, joint pains, muscle cramps, weight gain, irritability, depression and acne[20]. Gonadotropin releasing hormone agonists are most commonly used hormonal drugs. These drugs shut down the production of FSH and LH hormone and thus produce a condition like menopause[12,21] .In addition to hormonal drugs, pain killers like nonsteroidal, anti inflamatory drugs (NSAIDs) and natrosyn, rufen, motrin are also used. Laproscopic electrocautery is a quality surgical approach for treatment endometriosis and laprotomy involves the opening up of abdominal cavity and is considered major surgery.

The goal of ayurvedic approach is to enliven the body's natural self healing abilities to not only treat endometriosis but also to prevent the disease in general and create a state of health and well being. Since endometriosis is the condition of ama accumulation, the treatment

should focus on detoxification (Shodhan therapy) in order to remove the ama and get the dosha's back to their original position therefore panchkarma is indicated along with agni therapy. Ayurvedic oil massage daily loosen up the ama, collect it from different part of the body and bring back to the digestive system for elimination[22]. Even though pitta is anubandha dosha, the treatment should be vata hara as apana vata is the main controlling factor of the female reproductive system. Uttarbasti is best and complete treatment for vatavyadhi so for endometriosis. Rasayan and prajasthapak chikitsa are other useful treatment for it. The importance of rasayan chikitsa is to treat vaigunya of artava vaha srotas as the artav is updhatu of rasa dhatu and rasayan chikitsa is best for rasa dhatu. Prajasthapak treatment is important as the condition is usually associated with primary infertility in modern aspects. Virechan is beneficial with trivruth lehya per oral in a dose of 10-15 gm after menstrual period. Yoni prakshalan with panchvalkal kwath, bala tail or neelothpaladi tail uttar vasti for 7 days in ritukal i.e. from 5th to12th day is useful in married women suffering from endometriosis. Unmarried patients should be given sneha vasti with guduchyadi tail. Few ayurvedic formulations are beneficial in these cases like Phala ghrit 2 TSF twice daily with milk, Chandraprabha vati 2 tab twice daily (a potential drug in yoni and artav vyapad as it contains guggulu, a rasayan, vedana sthapak and vandhyatvahar and shilajeet, a proven drug in reproductive disorders), ashokarisht 15 ml twice daily. In pateint with extra pelvic endometriosis kshara karma may be fruitful[23]. Uttarvasti and per oral administration of prishnaparni has shown good results in cases of dysfunctional uterine bleeding[24], it may be also useful in cases of endometriosis.

Therefore ayurvedic approach is cost effective, with minimal side effects and a boon specially to young unmarried and nulliparous, infertile females.

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PARIJAT GHANSATVA AS PREMEDICANT AND BIOCHEMICAL STATUS

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ABSTRACT: Literary evidences are present in our classics that thousands years ago in our Indian civilization one thing was very popular that was the Science of Sammohan. At that time it was known as SAMMOHAN VIDYA. This science consist of some pharmacological and some non-pharmacological procedures for creating unconsciousness without any hazard to the subject.

Previous workkers Dr. R.K. Jaiswal, Dr. S.K. Singh and Dr. G.S. Shah explored that Parijat is a good analgesic and is free from apparent clinical side effects. Now we tried to study the effect of Parijat ghansatva at biochemical level.

For the present study we have selected Parijat ghansatva to evaluate its efficacy under intrathecal anaesthesia at biochemical level. The clinical trial was conducted over sixty patients of both, posted for minor to moderate operations under LSAB with 0.5% Bupicacaine-Heavy.

Three blood samples(F.B.S., B.U., Serum Cholesterol and Liver Function Test) were taken:–1.Before premedication 2. Two hours after premedication (30 minutes after spinal anaesthesia) and 3. After sensory recovery from spinal anaesthesia.

No significant hazardous biochemical changes were seen during the whole study. Therefore it is concluded that Parijat Ghansatva can be used safely under Intrathecal Anaesthesia.

Key words: Parijat, Ghanatva, Intrathecal Anaesthesia, Biochemical, Premedication, Pain.

INTRODUCTION :

This is a fact that none of the analgesic available in the market is free from side effects. This is why the Section of Sangyaharan tried to explore a possibility to search out an indigenous analgesic free from side effect or having least side effect.

Aim & Object:

1. to evaluate the Biochemical changes during intrathecal anaesthesia with use of Parijat Ghansatva as premedicant-trial drug.
2. to evaluate the the Biochemical changes during intrathecal anaesthesia with use of Diclofenac odium-control drug.

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Material and Methods:.

Collection and Preparation of Drugs

The crude drug was collected from the Ayurvedic Garden of Institute of Medical Sciences, Banaras Hindu University, Varanasi and after confirming its validity, coarse powder was prepared (after the drug was dried completely under the shade).

Preparation of Ghansatva :

The process of preparation of ghansatva is divided into two steps.

Step I: Preparaion of Decoction 500 gms of coarse powder of dried leaves was mixed with 2 liters of water and boiled. When one fourth of initial content was remained, I was filtered thus the decoction was prepared.

Step II: Preparation of Ghansatva The decoction was boiled again upto change in its form from liquid to semisolid. Then dried under shade and thus Ghansatva was prepared. After preparation of ghansatva a fine powder was made by triturating. The complete procedure was done in the laboratory of Department of Shalya-Shalakya, Institute of Medical Sciences, Banaras Hindu University, Varanasi by well trained team of assistants and doctors. Its quality was checked by Department of Dravyaguna, Institute of Medical Sciences, Banaras Hindu University, Varanasi.

Drug Presentation

It was presented in the form of capsule, Each capsule has 500 mg Parijat ghansatva.

Dose of Parijat Ghansava :Two capsules (1000 mg) 90 min. before operation P.O.

Grouping of the Patients For the present study we selected sixty patients of both sexes with age and weight distribution proposed for lower abdominal surgery going under spinal anaesthesia.

Groups of patients	No. of Patients	Premedicant
Group I: Control Group	30	Previous night (at 10 P.M.):Tab. Vaveran 50 mg P.O. On the Day of Operation (90 min. before operation): Inj. Glycopyrolate 0.2 mg IM + Cap. Parijat ghansatva 1000 mg P.O
Group II: Trial Group	30	Previous night (at 10 P.M.):Cap. Parijat ghansatva 1000 mg P.O On the Day of Operation (90 min. before operation): Inj. Glycopyrolate 0.2 mg IM + Cap. Parijat ghansatva 1000 mg P.O

Neither the observer nor the patient was aware of the types of drug administered. Before administering the scheduled premedicant Pulse rate, Blood pressure, Respiratory rate and other physiological and psychological conditions were recorded. After 60 to 90 minutes of premedicant Pulse rate Blood pressure and Respiratory rate were recorded, then anaesthesia was induced. During subsequent coarse of anaesthesia Pulse rate, Blood pressure, Respiratory

rate and other squeal were noted. After the end of one hour of surgery, samples were collected for Intra-operative biochemical changes. Same observation and collection of sample was taken during recovery period (i.e., complete reversal of sensory block) .

Inclusion criteria:

Sixty patients with a narrow age and weight difference scheduled for elective surgery were taken for the study and were randomly divided into two groups consisting of 30 patients in each group.

EXCLUSION CRITERIA:

The following classes of patients were excluded from the study-

1. those who were out of 18 to 50 years of age.

2. who were out of A.S.A. GP. 1& 2.

3. who were pregnant.

4. Patient suffering from Respiratory, cardiac, hepatic, renal, sensitive to aspirin, diclofenac sodium, bleeding disorders and peptic ulcer.

Technique of Anaesthesia: Uniform anesthetic technique –S.A. with 25 WG needle at L3-4 interspaces in lateral position with 0.5% Bupivacain heavy -3ml.

Parameters used:

In this study Clinical and Biochemical parameters both were taken. For clinical study changes in Pulse rate, Blood pressure, Respiratory rate and Temperature were recorded during each step like as before premedication, after premedication, during anaesthesia and after recovery from anaesthesia. For the biochemical study we selected following parameters.

1. Fasting blood sugar
2. Blood urea
3. S. Cholesterol
4. L.F.T.
5. Free fatty acids

For this we collected the blood sample in three steps:

1. Before induction of anaesthesia
2. During anaesthesia
3. After recovery from anaesthesia

PREMEDICATION: AS SHOWN IN THE ABOVE TABLE- GROUPING OF THE PATIENT

OBSERVATION & RESULT WITH DISCUSSION:

Age and Weight

Table 1. Comparison of Mean age and weight:

	Group I	Group II	Statistical Comparison		
			t	p	Remark
Mean age (years)	29.77 ± 6.09	33.10 ± 6.92	-1.97	<0.05	N.S.
Mean weight (kg)	49.53 ± 5.30	51.00 ± 4.65	-1.14	<0.05	N.S.

Age and Weight is identical.

Table 2 a. Effects on Pulse Rate changes per minute

Group	Mean pulse rate (mean ± S.D.)			
	A	B	C	D
Group – I Control group	84.03 ± 4.46	84.63 ± 3.86	84.93 ± 4.52	83.66 ± 4.02
Group II – II Trial group	83.87 ± 2.96	84.30 ± 3.14	83.93 ± 3.16	83.97 ± 2.83

A= Pulse rate before premedication , B= Pulse rate after premedication

C= Pulse rate during intrathecal anaesthesia , D= Pulse rate after recovery from anaesthesia

Table 2 b. Statistical comparison within the group

	Group I – Control group			Group II – Trial group		
	A vs B	A vs C	A vs D	A vs B	A vs C	A vs D
Mean S.D.	0.6 ± 2.53	0.9 ± 3.46	-0.37 ± 2.46	0.43 ± 1.25	0.07 ± 1.20	0.10 ± 0.80
S.E.	0.46	0.63	0.45	0.23	0.22	0.15
t-value	1.30	1.42	-0.82	1.89	0.30	0.68
p-value	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Remarks	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.

Table 2 c. Statistical comparison between the groups

Group I vs Group II

	A vs B	A vs C	A vs D
t-value	0.33	1.24	-1.00
p-value	<0.05	<0.05	<0.05
Remarks	N.S.	N.S.	N.S.

Pulse rate changes at different label are identical.

Table 3a. Effects on Blood Pressure changes (M.B.P.)

Group	Mean Blood Pressure (mean ± S.D.)			
	A	B	C	D
Group – I Control group	92.33 ± 2.30	92.22 ± 2.23	91.42 ± 2.74	92.13 ± 2.12
Group II – II Trial group	92.26 ± 2.35	92.25 ± 2.35	91.78 ± 1.77	91.98 ± 2.18

Table 3b. Statistical comparison within the group

	Group I – Control group			Group II – Trial group		
	A vs B	A vs C	A vs D	A vs B	A vs C	A vs D
Mean S.D.	-0.11 ± 0.46	-0.64 ± 1.86	-0.09 ± 0.55	-0.01 ± 0.51	-0.54 ± 1.86	-0.18 ± 0.52
S.E.	0.09	0.34	0.1	0.09	0.34	0.1
t-value	-1.32	-1.89	-0.89	-0.12	-1.61	-1.87
p-value	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Remarks	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.

Table 3 c. Statistical comparison between the groups

Group I vs Group II

	A vs B	A vs C	A vs D
t-value	-0.77	-0.21	0.64
p-value	<0.05	<0.05	<0.05
Remarks	N.S.	N.S.	N.S.

Mean Blood Pressure changes at different label are identical

Table 4a. Effects on Respiratory Rate changes (per minute)

Group	Mean respiratory rate (mean ± S.D.)			
	A	B	C	D
Group – I Control group	17.97 ± 1.22	18.17 ± 1.15	18.23 ± 1.10	17.77 ± 1.22
Group II – II Trial group	17.7 ± 1.02	17.73 ± 1.14	17.93 ± 1.11	17.73 ± 0.98

Table 4b. Statistical comparison within the group

	Group I – Control group			Group II – Trial group		
	A vs B	A vs C	A vs D	A vs B	A vs C	A vs D
Mean S.D.	0.2±1.06	0.2±1.35	-0.23±0.97	0.03±0.81	0.17±1.18	0±0.79
S.E.	0.19	0.25	0.18	0.15	0.22	0.14
t-value	1.03	0.8	-1.29	0.23	0.78	0
p-value	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Remarks	**N.S.**	N.S.	N.S.	N.S.	N.S.	N.S.

Table 4 c. Statistical comparison between the groups

Group I vs Group II

	A vs B	A vs C	A vs D
t-value	0.71	1	-1
p-value	<0.05	<0.05	<0.05
Remarks	N.S.	N.S.	N.S.

Respiratory rate was found insignificant at both the level.

Table 5 a. Effects on Body Temperature changes (0^F)

Group	Mean Temperature (mean ± S.D.)			
	A	B	C	D
Group – I Control group	98.53±021	98.77±0.21	96.78±0.66	98.23±0.21
Group II – II Trial group	98.5±0.23	98.68±0.19	96.41±0.2	98.21±0.15

Table 5 b. Statistical comparison within the group

	Group I – Control group			Group II – Trial group		
	A vs B	A vs C	A vs D	A vs B	A vs C	A vs D
Mean S.D.	0.24±0.23	-1.92±0.37	-0.25±0.22	0.18±0.21	-2.03±0.42	-0.287±0.21
S.E.	0.04	0.07	0.04	0.04	0.08	0.04
t-value	5.85	-27.4	-6.33	4.86	-26.7	-7.5
p-value	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
Remarks	H.S.	H.S.	H.S.	H.S.	H.S.	H.S.

Table 5 c. Statistical comparison between the groups

Group I vs Group II

	A vs B	A vs C	A vs D
t-value	1.07	1.04	0.71
p-value	<0.05	<0.05	<0.05
Remarks	N.S.	N.S.	N.S.

The rise in mean temperature after premedication level in both the groups were statistically significant where as were statistically insignificant between the group I and II.

Table 6 a. Effect on Fasting Blood Sugar

Group	Mean Fasting Blood Sugar (mean ± S.D.)		
	A	B	C
Group – I Control group	81.13±8.37	79.4±8.59	79.93±8.46
Group- II	79±8.97	81.3±9.02	80±5.38

Table 6 b. Statistical comparison within the group

	Group I – Control Group		Group II – Control Group	
	A vs B	A vs C	A vs B	A vs C
Mean S.D.	-1.67±12.48	-1.13±12.57	2.3±14.92	1±10.16
t-value	-0.73	-0.49	0.85	0.54
p-value	<0.05	<0.05	<0.05	<0.05
Remarks	N.S.	N.S.	N.S.	N.S.

Table 6 c. Statistical comparison between the groups – Group I vs Group II

	A vs B	A vs C
t-value	1.12	0.72
p-value	<0.05	<0.05
Remarks	N.S.	N.S.

Table 6a, b & c shows that mean fasting blood sugar variation between the groups were statistically insignificant.

Table 7 a. Effect on Blood Urea

Group	Mean Blood Urea (mean ± S.D.)		
	A	B	C
Group – I Control group	22.9±4.11	22.3±3.15	22.7±3.02
Group- II Trial group	22.7±3.64	22.3±2.84	22.4±2.22

Table 7 b. Statistical comparison within the group

	Group I – Control Group		Group II – Control Group	
	A vs B	A vs C	A vs B	A vs C
Mean S.D.	-0.6±2.81	-0.2±2.68	-0.4±3.76	-0.3±3.01
t-value	-1.18	-0.41	-0.58	-0.55
p-value	<0.05	<0.05	<0.05	<0.05
Remarks	N.S.	N.S.	N.S.	N.S.

Table 7 c. Statistical comparison between the groups – Group I vs Group II

	A vs B	A vs C
t-value	-0.23	0.14
p-value	<0.05	<0.05
Remarks	N.S.	N.S.

Table 7a, b & c shows that mean blood urea variation between the groups were statistically insignificant.

Table 8 a. Effect on Serum Cholesterol

Group	**Mean Serum Cholesterol (mean ± S.D.)**		
	A	B	C
Group – I Control group	152.5±23.77	151.9±21.04	152±19.62
Group- II Trial group	131.4±12.44	130.8±14.15	132±11.99

Table 8 b. Statistical comparison within the group

	Group I – Control Group		**Group II – Control Group**	
	A vs B	A vs C	A vs B	A vs C
Mean S.D.	-0.6±6.47	-0.5±7.82	-0.6±4.82	0.6±4.2
t-value	-0.51	-0.35	-0.68	0.78
p-value	<0.05	<0.05	<0.05	<0.05
Remarks	N.S.	N.S.	N.S.	N.S.

Table 8 c. Statistical comparison between the groups – Group I vs Group II

	A vs B	**A vs C**
t-value	**0**	**-0.68**
p-value	<0.05	<0.05
Remarks	N.S.	N.S.

Table 8a, b & c shows that mean Serum cholesterol variation between the groups were statistically insignificant.

Table 9 a. Effect on Serum Bilirubin

Group	Mean Serum Bilirubin (mean ± S.D.)		
	A	B	C
Group – I Control group	0.74±0.27	0.7±0.2	0.72±0.21
Group- II Trial group	0.68±0.19	0.67±0.19	0.69±0.17

Table 9 b. Statistical comparison within the group

	Group I – Control Group		Group II – Control Group	
	A vs B	A vs C	A vs B	A vs C
Mean S.D.	-0.04±0.12	-0.02±0.12	-0.01±0.03	0.01±0.055
t-value	-1.82	-0.93	-1.82	1
p-value	<0.05	<0.05	<0.05	<0.05
Remarks	N.S.	N.S.	N.S.	N.S.

Table 9 c. Statistical comparison between the groups – Group I vs Group II

	A vs B	A vs C
t-value	0	-0.68
p-value	<0.05	<0.05
Remarks	N.S.	N.S.

Table 9a, b & c shows that mean Serum Bilirubin variation between the groups were statistically insignificant.

Table 10 a. Effect on Serum Alkaline Phosphatase

Group	Mean Serum Alkaline Phosphatase (mean ± S.D.)		
	A	B	C
Group – I Control group	5.2±1.63	5.3±1.82	5.3±1.51
Group- II Trial group	6.7±2.14	7.05±1.12	6.9±1.79

Table 10 b. Statistical comparison within the group

	Group I – Control Group		Group II – Control Group	
	A vs B	A vs C	A vs B	A vs C
Mean S.D.	0.1±1.73	-0.01±1.47	0.35±2.06	0.2±0.207
t-value	0.31	-0.37	0.92	0.53
p-value	<0.05	<0.05	<0.05	<0.05
Remarks	N.S.	N.S.	N.S.	N.S.

Table 10 c. Statistical comparison between the groups – Group I vs Group II

	A vs B	A vs C
t-value	-0.5	-0.6
p-value	<0.05	<0.05
Remarks	N.S.	N.S.

Table 10a, b & c shows that mean Serum Alkaline Phosphatase variation between the groups were statistically insignificant.

Conclusion

The indigenous drug Parijat (Nyctanthes arbor-tristis. Linn.) ghansatva is capable to produce following effects –

a. maintain cardiovascular system stable.

b. maintain respiratory system stable.

c. Produce good analgesic, antipyretic and anti-inflammatory effects.

d. Maintain the fasting blood sugar approximately normal.

e. Produce no adverse effect on liver function or renal function.

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VIVIDHA VEDANA

* Dr. S.S.Mishra

Abstract- In Ayurvedic classics, words Vedana, Ruja and Pida are having the meaning of pain or unpleasant feeling or sensation. A person feels or experience verities of unpleasant sensations or Vedana. Such different kinds of feeling of pain are called as "Vividha Vedana" or character of the pain. The terms colicky, gripping and throbbing are mentioned in modern medical literatures and Toda, Manthana and Choasa etc in Ayurvedic classics to express the kinds of pain.

Such words depend on doshik involvement, end organ, patients intelligence, vocabulary and concepts of what is taking place.)

Key word- Vedana, Shambukavarta, Bahula.

In Ayurvedic classics word Vedana, Ruja and Pida are having the meaning of pain or unpleasant feeling or sensation. Feeling or experience of vedana may be of several kinds or in other words a person feels or experiences varieties of unpleasant sensations or vedana. Such different kinds of feeling of pain are called as vividha vedana or character of the pain.

It is likely that for each type of sensation there are specific end organs, nervous pathways and sensory receptor cells in the thalamus and the cortex of the cerebrum, and that each sensation is produced by the application of a specific and adequate stimulus. Much reliance is put on the patient's choice of word and intensity of the pain. The expression of feeling of particular pain in words depends on the patients intelligence, vocabulary awareness and concept of what is taking place. It is better to give him a choice of useful terms such as colicky, griping stabbing, burning or aching.

The following types of pain are commonly experienced-

Colicky Pain- This pain is rhythmically intermittent in nature with brief periods of intense pain of several seconds duration, followed by longer intervals of remission. Colicky pain arises due to obstruction or distention of any of hollow viscera.

Gripping Pain- Such pain most characteristically arises from the heart, and is felt in the chest as impending its expansion. Biliary colic and impaction of stone in ureter are often gripping type of pain.

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Throbbing Pain- Arterial pulsation or disease gives rise to the painful stimuli to produce the throbbing pain.

Stabbing Pain- It is common as a symptom of anxiety. It occurs in Tabes dorsalis. The transient stabbing pain of left mammary region or in either iliac fosse is common in anxious people. Such pain may arise in the iliac fosse with ovarian dysfunction. Stabbing pain may be felt in hypochondria or iliac fosse in pleurisy. Perihepatitis and perisalpingitis cause a similar sharp unilateral pain worse on breathing, sneezing or coughing.

Aching Pain- This describes the quality of pain arising from deep tissues and is used by patient to indicate only moderate severity and describe mild colic or peritoneal pain, having no close pathological correlation.

Agonizing Pain-It is the characteristic of acute pancreatitis and of torsion of testis.

Burning Pain- The searing pain of peritonitis is some times described as "burning". It is steady, persistence without intervals of relief. In perforated peptic ulcer burning pain as positive sing is present.

Gnawing Pain- The pain of peptic ulcer is frequently designated as "Gnawing" but again the deep, steady quality is more important to denote it.

According to Ayurvedic literatures vividh vedana or character of pain mainly depends on doshic involvement or doshik predominancy and kriyakala awastha at the site of causative of the pain. The various kinds of vedana may be classified according to doshic predominance as follow.

Vatik Vedana

Such kind of Vedana appears mainly due to provoke vata dosha at different sites and in different diseases. Vatik pain may occur without any appreciable causes and appears or disappears repeatedly. The following Vedana experienced by the patient in Vatik dosha provoking diseases.

Toda-Pain felt like

Bheda- Pain felt like the

Tadana- Pain felt like th

Chhedana- Pain perceived like the cutting of the part by a sharp weapon.
Aayaman- Pain felt like the expanding or stretching of that part.
Manthana-Pain felt like the churning or gnawing of the part.
Vikshepana- Pain experienced like the throwing or shooting of structure.
Chumchumayana- Pain felt like the irritating or tingling of the part or pain feels like plastering of Rajika or Sarshapa.
Nirdahana- Pain felt with burning sensation.
Avabhanjana- Pain felt like the breaking of that structure.
Sphotana- Pain felt like the bursting of the part.
Vidarana- Pain felt like the tearing of the part.
Utpatana- Pain felt like extracting or uprooting of the part.

Kampana- Pain as the trembling sensation.
Shoola- Pain felt like the piercing of the spike.
Vishleshana- Pain felt like the dislocating or disintegrating of that part.
Vikiran- Pain experienced like the radiating or shifting of the part.
Stambhana-Pain felt like the stiffening of the part.
Poorana- Pain felt like the filling of that part.
Swapna- Pain experienced like numbening in that part.
Aakunchana- Pain felt like the contraction in that part.
Ankushika- Hooking or twitching type of pain.
Nistoda- Like Toda.
Parikartana- Pain felt like the cutting by scissor.
Angamarda- Body ache like pain.
Krityata or kartna- As Parikartana.
Aaddyalyata- Pain felt like grinding of the part.
Avadarana- Pain felt like the breaking of the part.
Chatchatayansheela- Pain felt like the cracking of the part.
Vyathyata- Pain felt like shaking of the part.
Pratyasyata-Pain felt like throbbing in that part.
Aayama-As Aayamana.
Swapa-*As swapna.*
Dashana- Pain such as biting of ants.
Sansarpaya- Pain such as roaming over the body ants.
Pidana- Pain felt such as pressing of the part with hand.
Ghatana-*Pain felt such as that part rubbing with finger or scraped round with finger.*
Viddha- Pain such as stung by scorpion.

Paittik Vedana

Paittik Vedana appears mainly due to Pitta dosha provocation at different sites and in different paittik diseases. Such pain also appears also in Raktaja kind of the diseases. The following Vedana experienced by the patients in provoked Paittika diseases.
Oasha- Pain felt with localised heat.
Choasha-Pain felt like sucking of the part.
Paridah-Pain with generalized heat or feel like body is burnt.
Dhumayan-Pain with feeling like smoke emitting or inhaling heat or vapor.
Pachan-Pain felt as if strewn with pieces of glowing charcoal, or pain as if caustic alkali is put in open wound.
Ushma Abhivriddhi- Pain with increase of heat at wound side.
Dahan- Pain as burning with fire.

Kaphaja Vedana

Kaphaja Vedana appears mainly due to kapha dosha provocation at different site and in various kaphaja kinds of diseases. Such pains are of low intensity. The following Vedana feels by the patient in Kaphaja provoke conditions-
Kandu- *Pain felt like itching.*
Gurutwa- Pain felt like heaviness in that part.
Upadehatwa- Pain, which seems as if that part has been plastered over with a paste.
Supttatwa-Which seems as numbness.

Stambha-Pain felt with stiffness.
Shaitya- Pain with feeling of coldness.
Vata, Paittika Toda, Deha, Dhumayan etc. Mix Vedana of Vataj and Paittaja Vedana.
Vata, Kaphaja- Kandu, Nistoda, Darunah (intensive pain) etc. Mix Vedana of Vataj and Kaphaja Vedana.
Pitta, Kaphaja- Mix Vedana of paittika and Kaphaja Vedana. Such as Daha, Kandu etc.
Vata, Raktaja- Toda bahula (intensive needle pricking pain), Suptatwa etc.
Kapha, Raktaja - Guru and Kandu etc.
Vata, Pitta, Raktaja -Mix Vedana of Vataj and Paittik such as Sphurana, Toda, Daha, and Dhumayana etc.
Vata, Shleshma, Shonitaja- Kandu, Sphurana and chumchumayaman etc.
Pitta Shleshma Shonitaja- Deha and Kandu etc.
Vata, Pitta, Kaphaja-Trividha or mix Vedana of Vata, Pitta, and Kapha
Poorna Nadi Shambukavarta vat Samuttishthanti Vedana- Pain appears like whirlpool in Full River or spiral in snail shell.
Vata, Pitta, Kapha, Shonitaja-
Nirdahana- Pain with burning as making ash.
Nirmanthana- Churning like pain sphurana,
Toda, Kandu, Daha, Swapa and various special types of pain feeling which may not be possible to express in word etc.

Even then many pain feelings do not have proper words and called as strange pain.

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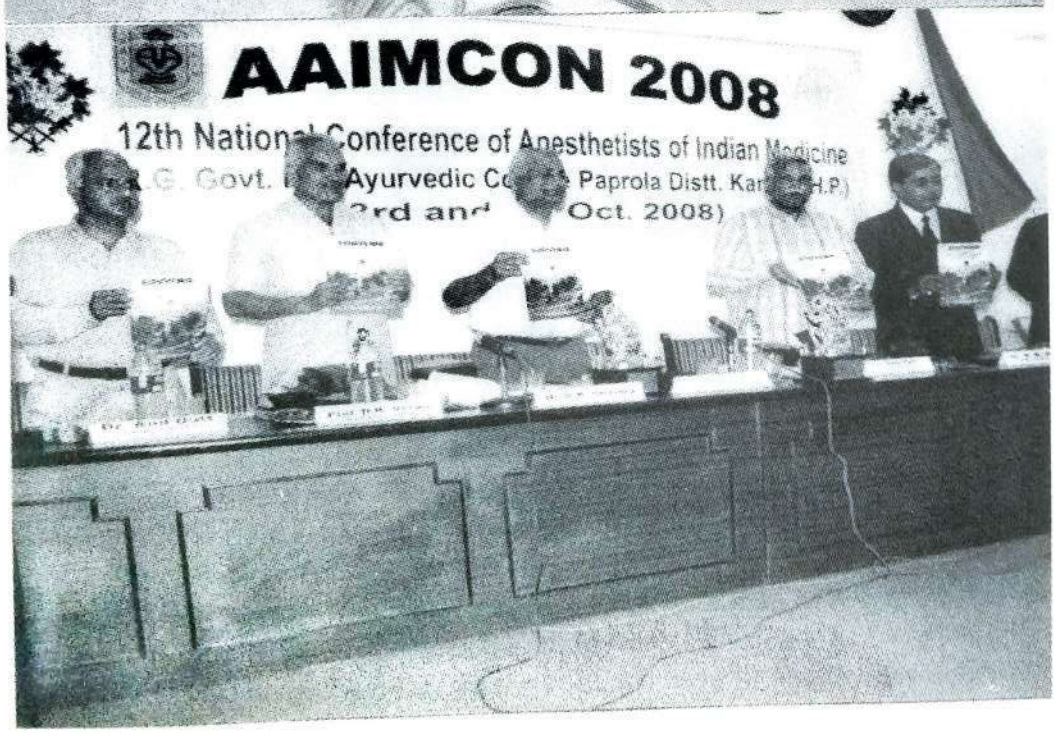

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Amavata roga- a literary review

***Dr. Murlidhar Paliwal**

Abstract-
Healthy long life is the first and foremost quest of every individual. Nobody wants ill-health, that's why **Acharya Charaka** has mentioned ***Praneshana*** (pursuit of life) at the first place among all the three pursuits ***(tisreshanas)*** & many ways to get long life is well mentioned. But unluckily either by intellectual blasphemy or by unavoidable factors like ***kala viparyaya*** (unhealthy seasons) etc. the diseases take place.
Amavata roga is one of the common and painful ailments caused by vitiation of ***vata dosha*** due to production of ***Ama dosha*** (improper digested part of food).It is mainly because of unwholesome contacts of sense organs with their objects and intellectual blasphemy. It affects the whole body but mainly ***Marmasthisandhi*** (vital parts of the body and bony joints) and aggravated during excessive cold and rainy season. The first Ayurvedic approach to treat this disease is ***Amapachan*** (digestion of the improper digested part of food).
Key words - ***Ama, Vata dosha, Shleshma sthan, stabdagatrata, Amavata***, Rheumatoid arthritis.

Introduction-
All the diseases affect the health (equilibrium state of the body) & Amavata is one of them. Although it affects all the systems of the body but it affects ***Marmasthisandhi*** (vital parts and bony joints) mainly. ***Sandhivata Roga & vatarakta roga*** also affect the bony joints but ***Amavata*** is long standing, very painful and ultimately crippling disease. This disease occurs in all the age group and both the sex (male and female).
Amavata contains two words- ***Ama & Vata. Ama*** means improper digested part of food[1] and Vata is vitiated ***Vata dosha*** (a type of body humors). Initially because of improper indigestion ***Amarasa*** takes place and then it obstructs the channels which ultimately vitiate ***Vata Dosha.*** Due to ***Amarasa*** and vitiated ***Vata Dosha*** pain and swelling in the joints and other associated symptoms occur. So ***Amarasa*** is main cause of this disease according to Ayurveda.
If we look towards it's historical review, it is not described in ***Vedas, Upanishadas, Puranas*** and ***smriti granthas.***
In Ayurvedic literature, it is not described in ***'Brihat-trayee'*** (Charaka Samhita, Sushruta Samhita& Vagbhata Samhita).Although the term ***Amavata*** has been used at different places while claiming the efficacy of certain compound drugs and in other contexts in ***'Brihat-trayee,***[2] yet it is not described as a separate disease entity.
Description regarding ***Nidana, Roopa, Bheda*** and ***chikitsa*** of ***Amavata is*** given first time in ***Harita Samhita***[3], but its authenticity is doubtful among many more scholars.
Detail description of ***Amavata*** is presented by ***Acharya Madhavakar*** in ***Madhava Nidan*** (7th century A.D.)[4]. After Madhava Nidan, Amavata is mentioned in many more classics as- Vrinda vaidyak[5], Chakradatta[6], Bangasen Samhita[7],Sharangadhar Samhita [8],Bhavaprakash [9],Gada Nigraha[10],Rasendrasarsamgraha[11],Yogaratnakar[12],Yogatarangini[13],Bhaishajya ratnavali[14] and so on.

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Etiology of *Amavata roga*

Aetiological factors of ***Amavata*** can be classified into two types mainly-

A. causative factor includes

I. Production of ***Ama***

Ii. Vitiation of ***Vata Dosha***

B. Predisposing factors include

I. ***Viruddha ahar*** (incompatible diet)

Ii. ***Viruddha vihar*** (incompatible regimen)

iii. ***Mandagni*** (hypo functioning of digestive fire)

iv. ***Nishchalata*** (sedentary life)

V. ***Vyayam*** with ***snigdha annapan*** (physical exercise after taking fatty diet)[15]

Almost all the scholars of Ayurveda accepted the same causes of this disease.

Samprapti (pathogenesis) of Amavata-

When a person of sedentary habits with hypo functioning digestive mechanism indulges in incompatible diet and regimen or does physical exercise after taking fatty food, the Ama is formed and propelled by ***Vayu*** and reaches to the ***shleshmasthyan*** (site of ***Kapha dosha)***. The ***Amarasa*** being incompletely processed and very much vitiated by ***vata, pitta*** and ***Kapha*** is circulated all over the body through the vessels. It then takes on multiple colours, becomes excessively mucoid and accumulates in the small channels. It renders the patient weak in no time and produces a feeling of heaviness in the pericardial region. This substance named ***Ama*** is the cause of so many distressing diseases. Simultaneously provoked ***Vata*** and ***Kapha*** enter into ***Triksandhi*** (sacral region) as well as all the joints of body and produces ***stabdhagatrata*** (stiffness of the body). This condition is known as ***Amavata*** in Ayurveda[16].

Factors responsible for pathogenesis can be summarized like this-

A. ***Nidan-***	Incompatible diet and regimen
B. ***Dosha-***	***Vata-Kapha*** dominant
C. ***Dushya-***	***Rasa Dhatu***
D. ***Srotas-***	***Annavaha srotas, Rasavaha srotas***
E. ***Adhishthan-***	***Shleshma sthan*** mainly joints
F. ***Ashaya-***	***Amashaya samuttha***
G. ***Vyadhi swabhav-***	***Chirkari*** (Chronic)
H. ***Srotodushti*** Type-	***Sang*** (obstruction)
I. ***Rogamarga-***	***Madhyam rogamarga***

Types of Amavata-

1. According to Harita Samhita, it is of four types[17]-

A. ***Vishtambhi ama***
B. ***Gulmakrit ama***
C. ***Snehi ama***
d. ***Pakwam***

2. According to clinical manifestation of the disease, it can be divided in three types-

A. Features in General-

B. Features in the stage of exacerbation-

C. Features according to Doshic dominance-

3. Acharya Sharangadhar described four types of Amavata on the basis of ***dosha*** predominance[18]-

A. ***Vataj***-B. ***Pittaj***-C. ***Kaphaja***-D. ***Sannipataj-***

Clinical manifestation –Clinically Amavata is well described by Acharya Madhavkar in Madhav Nidan .Stepwise description of the disease is given in Madhav Nidan which is very useful for a physician to diagnose and treat it. Such as-

General features of Amavata[19]-

- Body ache,Anorexia,Thirst,Lethargy,Feeling of heaviness,Fever,Indigestion,Swelling of the body parts etc.

Features in the stage of exacerbation[20]-

- Painful swellings such as in the joints of hand and feet, cervical region, knees and thighs.
- Affected part is excessively painful like scorpion bite
- Weaker digestive system
- Excessive salivation
- Anorexia
- Feeling of the heaviness
- Loss of the drive
- Bad taste in mouth
- Polyuria
- Burning sensation
- Hardness in abdomen
- Colicky pain
- Reversal of the sleeping habits
- Thirst
- Vomiting
- Vertigo
- Fainting
- Precordial discomfort
- Constipation
- Stiffness
- Gurgling intestinal sound
- Meteorism and other troublesome complications etc..

Features according to Doshic dominance[21]-

- Severe pain due to ***vata dosha***
- Redness and heat (locally) due to ***pitta dosha***
- Feeling of being covered with wet clothes, heaviness and itching due to ***Kapha dosha***

Modern view-

In modern medicine the word ***Amavata*** is not used for any disease but on the basis of similarities of signs & symptoms, most of the scholars correlate it with rheumatoid arthritis, an auto-immune disease. This disease influence 0.3 to 2.1% of the population of the world[22].although this disease occurs in all the age group and both the sex(male & female) but it is seen in females three times more than male.

Proper etiology of the RA is not known.

Regarding signs and symptoms of RA, we see that there are mainly seven criteria for classification of Rheumatoid arthritis established by American college of Rheumatology in 1987.As-

i. Morning stiffness
ii. Arthritis of three or more joint area
iii. Arthritis of hand joint
iv. Symmetric arthritis
v. Rheumatoid nodules
vi. Serum rheumatoid factor
vii. Radiographic changes

Four of seven are required to classify a patient as having RA. Criteria (I) and (IV) must be present for at least six weeks.

Differential diagnosis-D.D. is essential for proper diagnosis of the any disease. Amavata is not an exception in this sense. So D.D. with the diseases having most of the common features (e.g.Sandhivata, Vatarakta etc.) is given below with the help of Ayurvedic as well as modern view-

S.NO.	Name of the sign & symptom	Amavata	Sandhivata	Vatarakta
1.	Joint affected	Hand,feet,head,ankle, sacral, knee, thigh	Weight bearing joints	Small joints of hand & feet
2.	Nature of pain	Migratory pain	No migratory pain	No migratory pain
3.	onset	May be start with childhood	Generally after 40 years of age	Generally after 40 years of age
4.	Type of pain	As if Scorpion- bite	Pain in the joints due to vitiation of *vata dosha*	Spreads like virulent rat poison
5.	*Dosha* dominance	*Vata-Kapha*	*Vata*	*Vata*
6.	*Dushya*	*Rasa,rakta,mansa, snayu,asthisandhi*	*Asthisandhi*	*Rakta,twak,mansa*
7.	*Upashaya*	*Ruksha swedan deepan,pachan*	*Snehan,Vatahar Ahar evam aushadha*	*Raktamokshana,Vatahar evam raktashodhak aushadha*
8.	Heart involvement	Endocarditis or pericarditis may be	None	none
9.	Type of *Shotha*(swelling)	*Apakva*	*apakva*	May be *pakva*
10.	X-ray finding	Osteoporosis, erosion at bone margins	Narrowing of joint space,osteophytes can be found	Punched out areas(due to bone erosion, gouty tophi,sometimes lipping and osteophytes
11.	Laboratorial findings	RA factor, CRP may be positive, ESR raised	No specific test	Serum uric acid increases, ESR raised during attacks

Prognosis-Prognosis of the Amavata depends upon the Doshic status. If one dosha is involved, it is curable. In the involvement of two Doshas it is palliable. When all the Doshas are involved and there is an inflammation all over the body, the condition is very difficult to cure or incurable[23].

Any disease having maximum of the aetiological factors, prodromata ,signs & symptoms, involvement of all the ***Doshas,*** season, ***prakriti*** and ***Dushya*** having similar qualities to ***Doshas***, lack of body strength and unavailability of good quality chatushpad (four basic limbs of treatment) is almost incurable.

Discussion & conclusions-

After review of whole literature of Ayurveda it is clear that this disease is not described in Samhita period as a separate disease entity. It may be because of less prevalence of the disease at that time. Although in Harit Samhita it is mentioned, but authenticity of this Samhita is doubtful among most of the scholars.

Systematic clinical description of the disease is available in Madhav Nidan (7th century A.D.) for first time. Later on it is discussed in almost all the text books by different scholars, may be because of increasing prevalence in due course of time.

Main cause of the disease is ***Amarasa*** (improper digested part of food).

Prodromal signs & symptoms of the disease is not discussed anywhere in Ayurvedic literature.

Amavata affects all the systems of the body but mainly bony joints. In modern medicine also joint involvement is considered as a main feature of Rheumatoid arthritis. It is seen in all the age groups and both the sex.

Genetic predisposition is not very clearly mentioned in Ayurveda but it is observed in some cases. Modern medicine advocates it's genetic predisposition.

Amavata is a disease of ***yapya*** (relievable) nature and treated with ***langhan*** (lightning therapy like- fasting etc.), ***ruksha swedan*** (dry fomentation), ***Amapachan*** (digestives), ***shulhar*** (analgesics), ***virechan*** (purgation), ***Basti*** (medicated enema) etc.

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EVALUATION OF ANALGESIC EFFECT OF INDIGENOUS DRUG *KADAMBA* IN POST OPERATIVE PAIN MANAGEMENT UNDER EPIDURAL ANAESTHESIA

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ABSTRACT:

Pain is an extraordinarily complex sensation which is difficult to define and equally difficult to measure in an accurate, objective manner. The main role of the anesthetist is to enable patients to undergo surgical and other painful or uncomfortable procedure without pain or distress. Keeping in view these problems a thorough search was made in texts of ayurveda, to minimize untoward side effects of commonly used premedicant. A large number of indigenous drugs mentioned in Ayurvedic literature were experimentally screened on the animals and also studied clinically on the patients as pre anesthetic medicant such as Brahmi, Jatamansi, Mandukparni, Shigru and Dashmool etc.

The encouraging results of their studies prompted us to work on this line and a well known Vednasthapaka drug, KADAMBA (Anthocephalus indicus) was selected to evaluate its analgesic and anti-inflammatory activity in practice of Sangyaharan as premedicant. The trial drug Kadamba was used in the form of Ghanvati and compared with tab. Diclofenac sodium.

In the present clinical trial 16 patients were randomly divided into two equal & identical groups (8 in each group) i.e. group- I & II. These patients were planned surgical procedures under lumbar epidural anaesthesia. The entire patient evaluated before premedication, after premedication, during subsequent anaesthesia and during post anesthetic period.

KEYWORDS: Analgesics, vedanahar, kadamb ghanwati, Anthocephalus MBP=Mean Blood Pressure, ETCO2=End Tidal CO2, PR=Pulse Rate, Spo2=Oxygen saturation.

INTRODUCTION:

Pain is defined by the International Association for the Study of Pain (IASP) as an unpleasant sensory and emotional experience associated with actual or potential tissue damage mainly due to tissue trauma , sterile inflammation and tissue hypoxia but at times may be due infection which is bacterial in nature . It starts with the surgical tissue trauma and ends with healing. It has been defined as the sensory appreciation of afferent nociceptive stimulation which elicits an automatic component; both are subjected to rational interpretation by the patient.

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Relief of anxiety and apprehension is an important goal of Premedication in anaesthesia.In spite of lot of work done in the field of anaesthesia, no single drug amongst the presently available premedicants can fulfil the demand of an ideal premedicant.

Various clinical and experimental studies have been done previously by using different medicinal plants and indigenous drugs. In the present study well known Vednasthapaka drug, KADAMBA (Anthocephalus indicus) was selected to evaluate its analgesic and anti-inflammatory activity in practice of Sangyaharan as premedicant. The trial drug Kadamba was used in form of Ghanvati and compared with tab. Diclofenac sodium.

MATERIAL AND METHOD:

Number of patients: 16

Operation: lower abdominal surgeries.

Anaesthesia: lumber epidural anaesthesia.

Table 1.

Groups	No. of Patients	Premedication
Group I (Control)	8	1. Tab. of Diclofenac 50mg at 10 pm (previous night) and one hour before operation with an ounce of plain water orally. 2.Inj. Glycopyrrolate 0.2mg I.M. 1 hour before the induction anaesthesia
Group II (Trial)	8	1.Two tablet Kadamba Ghanvati (1000mg each) at 10.00 pm (previous night) and one hours before operation with an ounce of plain water orally 2.Inj. Glycopyrrolate 0.2 mg IM 1 hour before the induction of anaesthesia

OBSERVATION AND RESULT:

1. AGE, WEIGHT AND HEIGHT-

Table 2 .The statistical comparison of mean age, mean weight and mean height of the patients between the groups.

Group		Age (years) Mean ± SD	Weight (Kg) Mean ± SD	Height (cm) Mean ± SD
Group I (Control)		52.87 ± 12.98	53.75 ± 6.34	164.75 ± 7.40
Group II (Trial)		41.87± 19.12	57.00 ± 6.48	164.25 ± 6.08
Comparison between groups unpaired 't' test	t value	t = 1.35	t =-1.02	t = 0.15
	p-value	p > 0.05	P > 0.05	P > 0.05
Remark		NS	NS	NS

It is obvious from the above table that mean age, weight and height are statistically comparable and identical (p > 0.05) in the patients of both the groups.

2. EFFECT ON BLOOD PRESSURE

Table 3a: The statistical comparison of difference in mean of mean blood pressure (mm Hg) between the groups at corresponding time i.e. before premedication (W), after premedication (X), during subsequent anaesthesia (Y) and after recovery from anaesthesia (Z), by applying student t-test, p-values and remarks are as follows.

Group		Mean of MBP ± SD			
		Before premedication (W)	After premedication (X)	During subsequent anaesthesia (Y)	After recovery from anaesthesia (Z)
Group I (Control)		92.00 ±2.17	91.38 ± 5.45	89.88 ± 5.30	92.13 ± 3.94
Group II (Trial)		92.13 ± 3.94	92.25 ± 3.92	91.00 ± 4.31	92.00 ±2.27
Comparison between groups unpaired 't' test	t value	t = -0.08	t = -0.37	t = -0.47	t = 0.08
	p-value	p > 0.05	P > 0.05	p > 0.05	p > 0.05
Remark		NS	NS	NS	NS

The above statistical comparison represents that difference in mean of mean blood pressure between group I and group II at corresponding four different timings are statistically insignificant

Table 3b: The statistical comparison of mean of MBP (mmHg)

Comparison within the groups	Group I (Control)			Group II (Trial)		
	Mean ± SD	t-value p-value	Remark	Mean ± SD	t-value p-value	Remark
W vs. X	0.60 ±3.88	t = 0.45 p > 0.05	NS	-0.12 ± 2.16	t = -0.16 p > 0.05	NS
W vs. Y	2.12 ± 3.68	t = 1:63 p > 0.05	NS	1.12 ± 4.05	t = 0.79 p > 0.05	NS
W vs. Z	-0.12 ± 3.60	t = 0.31 p > 0.05	NS	0.12 ± 3.60	t = 0.10 p > 0.05	NS

From Table 3b it is observed that changes in MBP are insignificant in both groups at the levels of before premedication vs. after premedication, before premedication vs. during subsequent anaesthesia and before premedication vs. after recovery from anaesthesia.

4.EFFECT ON PULSE RATE

***Table 4a*: The statistical comparison of difference of mean pulse rate/min**

Group		Mean Pulse Rate/min; (Mean ± SD)			
		Before premedication (W)	After premedication (X)	During subsequent anaesthesia (Y)	After recovery from anaesthesia (Z)
Group I (Control)		79.00 ± 3.21	87.37 ± 2.56	78.50 ± 4.24	77.50 ± 3.51
Group II (Trial)		77.25 ± 7.01	86.62 ± 4.37	78.25 ± 4.83	76.50 ± 3.82
Comparison between groups unpaired 't' test	T value	t =0.64	t =0.42	t =0.11	t = 0.17
	p-value	p > 0.05	p > 0.05	p > 0.05	P > 0.05
Remark		NS	NS	NS	NS

From Table 4a, it is observed that difference of mean pulse rate when compared in between group- I and group- II at corresponding four different timings it is insignificant.

***Table 4b:* The statistical comparison of mean of pulse rate/min**

The statistical comparison of mean of pulse rate/min before premedication (W), after premedication (X), during subsequent anaesthesia (Y), and after recovery from anaesthesia (Z), within the group by applying paired t-test, p-values and remarks are as follows:Comparison within the groups	Group I (Control)			Group II (Trial)		
	Mean ± SD	t-value p-value	Remark	Mean ± SD	t-value p-value	Remark
W vs. X	- 5.00 ± 1.85	t = -7.64 p > 0.05	S	-5.50 ± 2.07	t = -7.51 p < 0.05	S
W vs. Y	-0.50 ± 3.50	t = 0.40 p > 0.05	NS	-2.40 ± 8.55	t =-1.08 p > 0.05	NS
W vs. Z	1.50 ± 2.07	t = 2.05 p > 0.05	NS	1.50 ± 4.14	t = 0.55 p >0.05	NS

.From Table 4b, it is observed that difference of mean pulse rate, at the level of before premedication and after premedication is significant in group- I and group II.

5. EFFECT ON RESPIRATORY RATE

Table 5a: **The statistical comparison of mean respiratory rate per minute**

Group		Mean Respiratory Rate/min: (Mean ± SD)			
		Before premedication (W)	After premedication (X)	During subsequent anaesthesia (Y)	After recovery from anaesthesia (Z)
Group I (Control)		16.00 ± 1.51	16.25 ± 1.67	16.50 ± 1.31	15.63 ± 1.30
Group II (Trial)		16.25 ± 1.39	16.25 ± 1.28	16.00 ± 1.07	15.87 ± 1.25
Comparison between groups unpaired 't' test	t value	t = 0.34	t = 0.00	t = 1.34	t = -0.60
	p-value	p > 0.05	p > 0.05	p > 0.05	P > 0.05
Remark		NS	NS	NS	NS

From Table 5a, it is observed that difference of mean respiratory rate per minute when compared in between group- I and group- II at corresponding four different timings; it is statistically insignificant and identical.

Table 5b:. **The statistical comparison of mean respiratory rate per minute**

Comparison within the groups	Group I (Control)			Group II (Trial)		
	Mean ± SD	t-value p-value	Remark	Mean ± SD	t-value p-value	Remark
W vs. X	-0.25 ± 1.98	t = -0.36 p > 0.05	NS	0.00 ± 1.41	t = 0.00 p > 0.05	NS
W vs. Y	-0.50 ± 2.13	t = 0.66 p > 0.05	NS	0.25 ± 1.38	t = 0.51 p > 0.05	NS
W vs. Z	0.37 ± 1.06	t = 1.00 p > 0.05	NS	0.37 ± 0.74	t = 1.43 p > 0.05	NS

From Table 5b, it is observed that changes in respiratory rate are insignificant in both groups at the levels of before premedication vs. after premedication, before premedication vs. during subsequent anaesthesia and before premedication vs after recovery from anaesthesia.

6. EFFECT ON TEMPERATURE

Table 6a: **The statistical comparison of difference in the mean axillary temperature (°F)**

Group		Mean Axillary Temperature (mean ± SD)			
		Before premedication (W)	After premedication (X)	During subsequent anaesthesia (Y)	After recovery from anaesthesia (Z)
Group I (Control)		98.40 ± 0.37	99.45 ± 0.49	98.58 ± 0.27	98.45 ± 0.37
Group II (Trial)		98.43 ± 0.38	99.80 ± 0.91	98.48 ± 0.21	98.58 ± 0.28
Comparison between groups unpaired 't' test	t value	T = 0.438	t = 0.89	t = 0.00	t = 0.31
	p-value	P > 0.05	p > 0.05	p > 0.05	P > 0.05
Remark		NS	NS	NS	NS

From table 6a, it is observed that difference of mean Axillary temperature (°F), when compared group- I and group- II at corresponding four different timings it is statistically insignificant.

Table 6b: **The statistical comparison of mean Axillary temperature (°F) within the groups**

Comparison between the groups	Group I (Control)			Group II (Trial)		
	Mean ± SD	t-value p-value	Remark	Mean ± SD	t-value p-value	Remark
W vs. X	-1.05 ± 0.34	t=-9.39 p<0.05	S	-1.37 ± 0.78	t =-4.98 p > 0.05	S
W vs. Y	-0.17 ± 0.32	t = -1.51 p>0.05	NS	-0.05 ± 0.43	t =-0.32 p>0.05	NS
W vs. Z	-0.05 ± 0.14	t =-1.00 p>0.05	NS	0.03 ± 0.70	t = -1.00 p > 0.05	NS

From Table 6b, when comparison is done for mean Axillary temperature (°F), within the both groups at the level of before premedication with after premedication is significant and difference of mean temperature before premedication and during subsequent anaesthesia and after recovery from anaesthesia is Insignificant in group I and group- II

7. EFFECT ON OXYGEN SATURATION-

Table 7a: **The statistical comparison of difference in SPO2 percentage between the two groups**

Group		Mean oxygen saturation (%) (Mean ± SD)			
		Before premedication (W)	After premedication (X)	During subsequent anaesthesia (Y)	After recovery from anaesthesia (Z)
Group I (Control)		99.25 ± 0.89	99.38 ± 0.74	99.00 ± 0.93	99.38 ± 0.74
Group II (Trial)		99.00 ± 1.20	99.00 ± 0.93	98.87 ± 0.83	99.25 ± 0.89
Comparison between group unpaired 't' test	t value	t = 0.48	t = 0.89	t = 0.00	t = 0.31
	p-value	p > 0.05	p > 0.05	p > 0.05	P > 0.05
Remark		NS	NS	NS	NS

From Table 7a, it is observed that difference of mean SPO2 percentage when compared between group- I and group-II at corresponding four different timings it is insignificant.

Table 7b: **Statistical comparison of difference in the mean SPO2 percentage**

Comparison within the groups	Group I(Control)			Group II(Trial)		
	Mean ± SD	t-value p-value	Remark	Mean ± SD	t-value p-value	Remark
W vs. X	-0.12 ± 1.35	t = -0.26 p > 0.05	NS	0.00 ± 1.51	t = 0.00 p > 0.05	NS
W vs. Y	0.25 ± 0.88	t = 0.80 p > 0.05	NS	0.12 ±1.95	t =-0.12 p > 0.05	NS
W vs. Z	-0.12 ± 1.24	t = -0.28 p > 0.05	NS	-0.25 ± 1.48	t =-0.48 p > 0.05	NS

From Table 7b it is observed that difference of SPO_2 percentage at all the level is insignificant in both groups.

8. EFFECT ON END TIDAL CARBON DIOXIDE ($ETCO_2$)

Table 8a: **The statistical comparison of difference of mean $ETCO_2$ in mmHg, between the two groups**

Group		Mean ETCO2(mmHg); (Mean ± SD)			
		Before premedication (W)	After premedication (X)	During subsequent anaesthesia (Y)	After recovery from anaesthesia (Z)
Group I (Control)		31.50 ± 1.41	31.25 ± 1.49	31.00 ± 1.07	31.50 ± 1.41
Group II (Trial)		31.75 ± 1.28	31.25 ± 1.83	31.50 ± 1.77	31.00 ± 1.07
Comparison between groups unpaired 't' test	T value	t =0.00	t = -0.63	t = -0.03	t = 0.00
	p-value	p > 0.05	p > 0.05	p > 0.05	P > 0.05
Remark		NS	NS	NS	NS

From Table 8a, it is observed that difference of mean $ETCO_2$ (mmHg) when compared in between group-I and group- II at corresponding four different timings it is insignificant.

Table 8b: **Statistical comparison of difference in the mean $ETCO_2$ (mmHg) within the groups**

Comparison within the groups	Group I (Control)			Group II (Trial)		
	Mean ± SD	t-value p-value	Remark	Mean ± SD	t-value p-value	Remark
W vs. X	0.25 ± 0.70	t = 1.00 p > 0.05	NS	0.50 ±1.41	t = 1.00 p > 0.05	NS
W vs. Y	0.50 ±1.41	t = 1.00 p > 0.05	NS	0.25 ± 2.49	t = 0.28 p > 0.05	NS
W vs. Z	0.25 ± 0.70	t = 1.00 p > 0.05	NS	0.75 ± 1.48	t = 1.43 p > 0.05	NS

From Table 8b, It is observed that difference of mean $ETCO_2$ (mmHg), at all the level is insignificant in group I and group- II.

9. DESIRABLE EFFECTS AND UNDESIRABLE EFFECTS

Table 9 a : **Incidence of desirable and undesirable effects in patients of both groups after premedication.**

Effects	Incidence	Group- I (Control)		Group-II (Trial)		Z-value between Group-I vs Group-II	Remarks
		No.	%	No.	%		
Sedation	Present	0	0	0	0	0	NS
	Absent	8	100	8	100		
Apprehension	Present	1	12.5	1	12.5	z=0	NS
	Absent	7	87.5	7	87.5		
Excitement	Present	0	0	0	0	0	NS
	Absent	8	100	8	100		
Dizziness	Present	0	0	0	0	C	NS
	Absent	8	100	8	100		
Nausea	Present	0	0	0	0	0	NS
	Absent	8	100	8	100		
Vomiting	Present	0	0	0	0	0	NS
	Absent	8	100	8	100		

The comparison between the group- I and group- II regarding sedation, apprehension and excitement is statistically insignificant.

The statistical comparison of undesirable effects like dizziness, nausea, vomiting, in between group-I and group-II at the level of after premedication is insignificant.

10. SURGICAL TIME AND DURATION OF ANAESTHESIA

Table 10 a: **Mean surgical time and mean duration of anaesthesia**

Parameters	Group-I(Mean ± SD)	Group-II(Mean ± SD)	t-value	p-value	Remarks
Total Surgical Time (min)	69.38 ± 14.25	66.88 ± 20.34	t = 0.28	>0.05	NS
Duration of Anaesthesia (min)	182.50 ± 13.89	180.63 ± 17.83	t = 0.23	>0.05	NS

Mean surgical time in group- I and group- II expressed in minutes were 69.38 ± 14.25 and 66.88 ± 20.34, respectively. The statistical comparison between the groups is insignificant.

Mean duration of anaesthesia in minutes in group- I and II were 182.50 ± 13.89 and 180.63 ± 17.83 respectively. The statistical comparison between the groups is found to be insignificant.

11. POST ANAESTHETIC SEQUEL

Table 11 a: **The incidence of post-anesthetic sequel**

Side Effects	Incidence	Group- I (Control)	Group- II (Trial)	Z-value between Group-I vs. Group-II	Remarks		
		No.	%	No.	%		
Sedation	Present	0	0	0	0	0	NS
	Absent	8	100	8	100		
Nausea	Present	0	0	0	0	0	NS
	Absent	8	100	8	100		
Vomiting	Present	0	0	0	0	0	NS
	Absent	8	100	8	100		
Dizziness	Present	0	0	0	0	0	NS
	Absent	8	100	8	100		
Dyspepsia	Present	0	0	0	0	0	NS
	Absent	8	100	8	100		
Gastric Irritation	Present	0	0	0	0	0	NS
	Absent	8	100	8	100		
Increased Peristalsis	Present	0	0	0	0	0	NS
	Absent	8	100	8	100		
Haematemesis	Present	0	0	0	0	0	NS
	Absent	8	100	8	100		
Malena	Present	0	0	0	0	0	NS
	Absent	8	100	8	100		
Precipitation of Asthma	Present	0	0	0	0	0	NS
	Absent	8	100	8	100		
Respiratory depression	Present	0	0	0	0	0	NS
	Absent	8	100	8	100		
Headache	Present	0	0	0	0	0	NS
	Absent	8	100	8	100		
Backache	Present	0	0	0	0	0	NS
	Absent	8	100	8	100		

Nausea, backache, headache, Sedation, vomiting, dizziness, dyspepsia, gastric irritation, increased peristalsis, haematemesis, malena, precipitation of asthma, respiratory depression and other side effects were noted meticulously in both groups and was found to be absent in all the groups.

12.REQUIREMENT TIME OF 1ST DOSE OF ANALGESIC

Table 12 a: **The mean of the 1st analgesic dose requirement time (in minutes)**

Groups	Mean ± SD	t-value	p-value	Remark
Control Group – I	291.88 ± 17.72	t = -1.76	p > 0.05	NS
Trial Group – II	306.25± 14.82			

It is obvious from the above table that requirement of the first dose analgesic time in patients of both the groups was almost equal and identical time intervals the statistical comparison of first dose analgesic requirement time between the groups is insignificant.

CONCLUSION

On the basis of observations, it can be concluded that in these groups:

- The trail drug Kadamba in the form of ghanvati has Vednahar and Shothhar properties.
- Kadamba Ghanvati did not produce any significant side effects when used as premedicant.
- No significant changes were observed in mean blood pressure,pulse rate,respiratory rate,temperature,oxygen saturation and end tidal carbon-di-oxide.
- The trail drug Kadamba in the form of Ghanvati (1000mg) is almost equally effective as analgesic and anti-inflammatory in comparison to control drug Diclofenac sodium(50mg).

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HIMRATAN OIL (हिमरतन तैल)

Indication: For local application in Shirahshool (Headache)/muscular spasm/low backache and Arthritis.

Method: Take 2-5 ml of Himratan oil and massage gently on the effected part.

हिम रत्न (आयुर्वेदिक शीतल तैल – हिमालय की जड़ी–बूटियों से निर्मित)

आयुर्वेदिक दवाओं के शास्त्रीय सिद्धान्तों का अनुसरण करते हुए, हिमालय के वनों से प्राप्त प्राकृतिक जड़ी–बूटियों का प्रयोग कर, आधुनिक वैज्ञानिक अन्वेषणों और प्रयोगों के अनुसार निर्माण कर हिमरतन तैल को जनसाधारण तक पहुँचाना ही हमारा उद्देश्य है।

हिम रत्न शीतल तेल – इसका प्रयोग सिर दर्द दूर करता है। यह सिर को ठंडा और दिमाग को तरोजाता रखने में विशेष उपयोगी है।

इसका मधुर गंध चित्त को प्रसन्न करता है तथा साधारण तेलों की तरह इसमें कोई रासायनिक तत्व नहीं हैं। इस तेल को आयुर्वेदिक चिकित्सकों के परीक्षण और उपयोग करने वालों के प्रामाणिकतानुसार बालों की विभिन्न समस्याओं में अत्यन्त उपयोगी पाया गया है। हिमरतन शीतल तैल चिपचिपाहट रहित, भीनी–भीनी सुगन्ध वाला बालों का पोषक है। इसके नियमित इस्तेमाल से बालों का प्राकृतिक सौन्दर्य सदैव कायम रहता है। बालों की लम्बाई बढ़ती है, बाल और सिर की त्वचा स्वस्थ रहती है। रुसी और जू दूर होता है। यह बालों की जड़ो तक पहुँचकर उन्हें पुष्ट करता है जिससे बालों का झड़ना रुक जाता है। आलोपेशिया (गंजापन) दूर होता है। असमय बाल पकना रूकता है। मामूली जलने कटने में भी यह तैल जल्द असर करता है।

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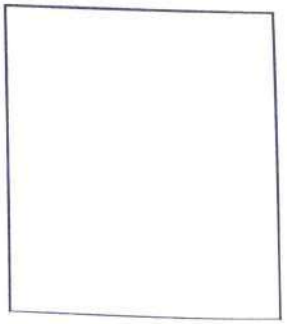

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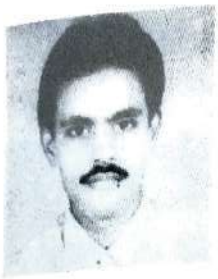

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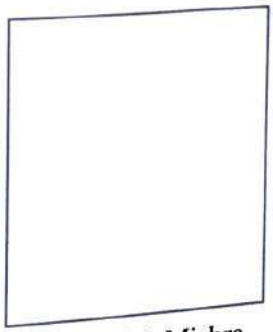

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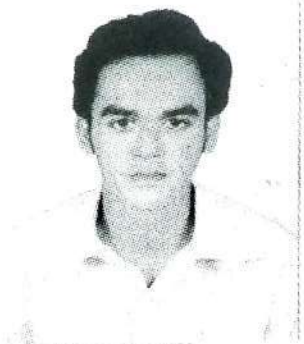

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