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RANDOMIZED OPEN LEVEL PARALLEL CLINICAL TRIAL OF SHUNTHI CHURNA, RASNADI GUGGULU AND RUKSHA VALUKA SVEDA IN CASES OF RHEUMATOID ARTHRITIS VIS-À-VIS AMAVATA UNDER THE INFLUENCE OF DOMINANT SETS OF DEHA PRAKRITI

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ABSTRACT

Background: Rheumatoid arthritis (RA) is a prevalent rheumatological condition in the society. It affects 1-3% of women in their life during middle age. It affects 1% of the population in India. The causes, diathesis and management of RA are still evolving in conventional system of medicine. Introduction of corticosteroids had brought big hope for the patients but because of its greater side effects. The overall role of steroids, Non steroidal anti-inflammatory drugs (NSAIDs) and Disease modifying anti-rheumatoid drugs (DMRDs) in the management of RA is very limited. Ayurvedic scholars in the medieval period have been vividly described the aetiopathogenesis and management of *Amavata*, which is comparable to RA of the conventional system of medicine. In the last few decades, researches have been conducted by research scholars but all of them did not show any major breakthrough. In this emerging scenario, the author has decided to launch a clinical study to evaluate

the efficacy of *Shunthi Churna*, *Rasnadi Guggulu* and *Ruksha (Valuka) Sveda* in a series of patients of RA vis-à-vis *Amavata* under the influence of dominant sets of *Deha Prakriti*. *Ayurveda* strongly believes that based on *Prakriti* every individual has its genetic makeup and it varies from person to person. So, the response of drugs and therapeutic measures is also different from person to person either it is belonging to Ayurvedic or modern drugs.

Methods: The present clinical study was launched to conduct an open parallel clinical trial in 60 patients (20 in each group) diagnosed (based on EULAR 2010 revised criteria) with Rheumatoid arthritis (*Amavata*), allocated randomly in the (ratio of 1:1:1) three parallel groups based on three dominant sets of *Deha Prakriti* viz. – Group A - *Vata* dominant, Group B - *Pitta* dominant and Group C - *Kapha* dominant, respectively. Each group was treated with a package of Ayurvedic drugs (*Shunthi churna* &*Rasnadi Guggulu*) and *Ruksha (Valuka) Sveda*.

Result: The patients of *Vata* and *Pitta* predominant *Prakriti* receiving Ayurvedic drugs and *Ruksha Sveda* have shown mild to moderate degree of relief in clinical symptoms, pain on Visual analogue scale (VAS), Erythrocyte sedimentation rate (ESR) & C - reactive protein (CRP). While the patients of *Kapha* predominant receiving Ayurvedic drugs and *Ruksha Sveda* have shown minimal relief in clinical symptoms, pain on VAS, ESR and CRP.

Conclusion: These drugs and measures are completely safe and effective; especially in *Vata* and *Pitta* predominant of *Deha Prakriti*. No unwanted effects were observed during the trial treatment.

Keywords: ***Amavata*, Ayurveda, *Deha Prakriti, Ruksha Sveda*, Rheumatoid Arthritis, *Shunthi churna*, *Rasnadi Guggulu***

INTRODUCTION

The worldwide prevalence of RA has been estimated as 0.24 percent based upon the Global Burden of Disease 2010 Study **[1]**. RA generally starts between the ages of 30 and 60 in women and somewhat later in life in men. The lifetime risk of developing RA is 3.6 percent for women and 1.7 percent for men. However RA can strike at any age, even small children can get it. Arthritis affects 15% of people i.e. over 180 million people in India and its prevalence is much higher than many well-known diseases such as diabetes, AIDS, and Cancer **[2]**.

It is a long-lasting autoimmune induced inflammatory disorder that primarily affects joints. The causes and diathesis of RA is not clear in the conventional system of medicine. It is believed to involve a combination of genetic and environmental factors. Ayurvedic scholars in the medieval period have been described *Amavata* in detail in their respective texts, which is highly evolved and comparable to recent concept of RA as known today in conventional system of medicine. In recent years RA is emerging as one of the challenging health disorders for the physicians due to its chronicity,

incurability, complications, morbidity spontaneous remission, and exacerbation. Concurrent stressful situations in life and lifestyle are known to precipitate exacerbation of RA. Every exacerbation leaves some deformities with progressive crippling, which leads to the economic burden for the patients, the family, society, and the nation.

In this emerging scenario, the author has decided to launch a clinical study to evaluate the safety and efficacy of *Shunthi churna, Rasnadi Guggulu* and *Ruksha valuka Sveda* in a series of patients of RA via-a-vis Ama*vata* under the influence of dominant sets of *Deha Prakriti* (body constitution). Ayurveda strongly believes that based on *Prakriti* every individual has its own genetic makeup and it varies from person to person. So, the response of drugs and therapeutic measures are also different from person to person. In recent years, this true knowledge of ancient scholars of *Ayurveda* gaining momentum by the researcher confined to human genetic.

MATERIALS AND METHODS [3]:

- **Aims & Objectives**

- To clinically evaluate the safety & efficacy of trial drugs and measures on subjective & objective parameters of RA vis-à-vis *Amavata*.
- To assess the role of *Deha Prakriti* on therapeutic response.

- **Hypothesis**

- **H_0-** *Rasnadi Gugglu, Shunthi Churna* and*RukshaSveda* have no effect on *Vata*, *Pitta*, and *Kapha Prakriti* patients of RA (*Amavata*)
- **H_1-***Rasnadi Gugglu, Shunthi Churna* and *Ruksha (Valuka) Sveda* have some effect on *Vata*, *Pitta*, and *Kapha Prakriti* patients of RA (*Amavata*).

- **Selection of Subject**

A total 60 Patients of RA (*Amavata*) was taken for the study from OPD & IPD of Department of Kayachikitsa, S.S.H., IMS, BHU, Varanasi, after detail history taking, clinical and laboratory examination, subjective and objective assessment. Besides, it also follows the diagnostic criteria of EULAR & clinical feature of *Amavata* as described in *Madhava Nidana* **[4]**

Study design: After following the exclusion & inclusion criteria, the *Amavata* patients of both sex was allocated (1:1:1 ratio) randomly into three parallel groups (20 patients in each group).The patients registered after detail interrogation, examination and relevant investigations were carefully recorded in the case record proforma especially prepared for this purpose.

Sampling method: Stratified randomized sampling

Type of study: Randomized open level parallel clinical trial.

Duration of study: 90 days.

Selection of drug: In the present study *Shunthi Churna, Rasnadi Guggulu,* were selected as trial drugs and *Ruksha Sveda* (dry fomentation) with *Valuka* (sand particle) as local therapeutic measures for fomentation.

Preparation of the Trial Drug: The useful part of *Shunthi Churna & Rasnadi Guggulu* were taken from original sources and identified by the experts of the department of *Dravyaguna* and *Rasa Shastra & bhaisajya kalpana*, Faculty of Ayurveda, IMS, BHU Varanasi. The crude fine powder of *Shunthi Churna was* prepared by BHU Ayurvedic Pharmacy and the *Rasnadi Guggulu* was prepared in the form of 500 mgs as per directive of Ayurvedic classics, which is procured from Swasthyavardhaka Comp. Pvt. Ltd, Varanasi. The genuineness of these drugs was confirmed by the expert of above mentioned department and department of medicinal chemistry.

Dosage and Duration

The prepared *ShunthiChurna* was given 2 gms bid with jaggery and *Rasnadi Guggulu* was given in the dose of 500 mg bid with Luke warm water after meal for 90 days in respective three trial groups.

Procedure and duration of *Valuka Sveda*:

The warm sand bolus of the required temperature applied on the affected parts of the body. The *Sveda* conducted (like *Pinda Sveda*) either in sitting posture or in whichever posture the patient feels comfortable and done comfortably in circular manner.

The temperature of the bolus maintained uniformly so that the patient should not feel discomfort either by more heat or less heat. If the sand becomes cold, the bolus must be changed and again a warm bolus should be applied on the affected part, till the local symptoms are reduced, or when the patient feels satisfied. In each affected part, usually *Sveda* is done for 10-15 minutes. The *Sveda* was done one or two or more times daily depending upon the severity of the disease.

Drug trial schedule

The selected patients for trial were randomly allocated (1:1:1 ratio) into the following three groups:

A) **Group A-** Patients *Vata* predominant sets of *Prakriti* were treated with combined therapy of *Rasnadi Guggulu* -500mg BID with Luke warm water & *Shunthi Churna*-2gm BID with jaggery after meal & *Ruksha Sveda* .

b) **Group B-** Patients *Pitta* predominant sets of *Prakriti* were treated with combined therapy of *Rasnadi Guggulu*-500mg BID with Luke warm water & *Shunthi Churna*-

2gm BID with jaggery after meal & *Ruksha Sveda*.

c) **Group C-** Patients *Kapha* predominant sets of *Prakriti* were treated with combined therapy of *Rasnadi Guggulu*-500mg BID with Luke warm water & *Shunthi Churna*-2gm BID with jaggery after meal & *Ruksha Sveda*.

Besides, no additional dietary and lifestyle intervention were enforced during the course of trial treatment.

Inclusion Criteria	Exclusion Criteria	Criteria for withdrawal-
Patients fulfilling the diagnostic criteria of RA and *Amavata* in *Ayurveda.*	The patients having severe degree joints deformities.	Personal matters
Both seropositive and sero-negative cases were included in present study.	The patients of both sexes having age <20 and >65 years were excluded.	Intercurrent illness
The patients of both sexes and middle age group (between >20 and <65 years) were registered.	Patients having duration of illness < 1 years and >5 years were excluded from the study.	Aggravation of complaints
Patients having duration of illness > 1 years and <5 years were recruited for the study.	Patients having severe Ankylosed joints.	Any other difficulties
	The patients of RA having major systemic complications were also excluded from the study.	
	Patient of rheumatic arthritis having other infective and non-infective joint disorders	

ASSESSMENT OF RESULT:-

Effects of the therapies were compared before and after the treatment on the basis of self-formulated scoring scales based on subjective parameters associated with the RA (*Amavata*).

PARAMETERS OF ASSESSMENT

Scale for *Amavata* symptoms as subjective parameters

To assess the subjective features of *Amavata*, the clinical symptomatology was graded into four grades i.e. 0, 1, 2, and 3 on the basis of severity and duration of illness and the same is applied in each patient of *Amavata*. The change in the gradations of each symptom was assessed the effect of given treatment. The eight points were used in this thesis from clinical features of *Amavata* as described in Ayurveda and their rating procedures are in **Table 1**.

Parameters of assessment of the Therapeutic Response:

- **Clinical Parameters**: In the present study assessment of drug response was done in terms of grade scores of clinical symptoms. Mean percentage fall in RA and CRP were also designed.
- **Laboratory Parameters**: Laboratory observations in terms of ESR at 30 days interval and quantitative RA & CRP, Blood urea, serum creatinine and liver function test are done before and after the treatment for assessment of safety of trial drugs.

Besides this, also design criteria to assess the shift of % change in quantitative RA & CRP in different trial groups.

Variants: Shift of percentage change in RA and CRP (in IU/ml): No improvement/Mild improvement/Moderate improvement/marked improvement.

A. Subjective assessment of improvement in clinical symptoms of RA

In *Amavata* (RA) improvement in clinical symptoms was assessed by a grade score system (based on shifting of patients from abnormal to normal given below (**Table 2**).

Table 1: Grading of *Amavata* symptoms

i. *Angamard (*stiffness*)*	GRADE
no stiffness	0
stiffness less than 1 hour	1
stiffness 1-2 hour	2
stiffness more than 2 hour	3

ii. *Aruchi* (lack of eating desire)	GRADE
normal eating desire	0
mild complained but not presented with associated features	1
complained for loss of appetite & also for associated features	2
complained and presented with associated clinical features	3

iii. *Trishna* (thirst)	GRADE
Absent	0
only feeling is present	1
marginally excess water intake	2
more excess water intake	3

iv. *Alasya* (lethargy)	GRADE
Absent	0
patient feel fatigability on exertional/heavy work	1
patient feel fatigability on moderate work	2
patient feel fatigability on mild work	3

v. *Gauravam* (heaviness of the body and joints)	GRADE
no heaviness	0
mild (present for < half hour)	1
moderate (present for >half hour but <one hour)	2
severe (for one hour or more)	3

vi. *Jvar* (fever)	GRADE
normal body temperature (98-99°F)	0
only feeling of fever	1
temperature above normal but <100°F	2
temperature > 100°F	3

vii. *Apaka* (Indigestion)	GRADE
absent (normally digestion of food in 3- yaam (9 hour)	0
transiently present with no associated symptoms	1
present for long period with less associated symptoms	2
regular present with much associated symptoms	3

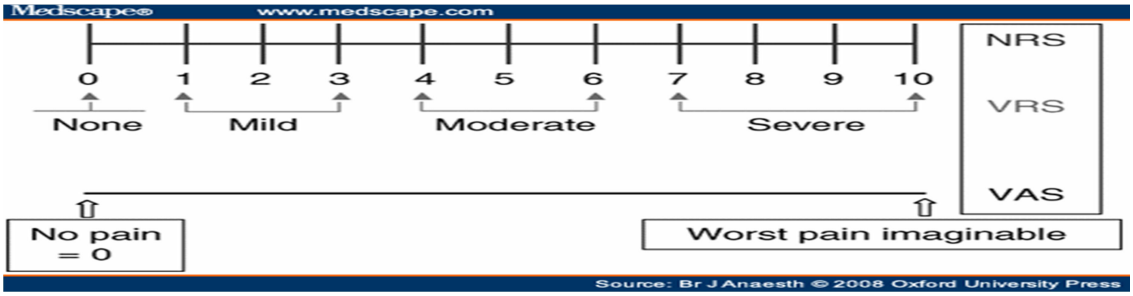
viii. Shuntanganam (Swelling over body part)	GRADE
No swelling/normal joint contour	0
swelling complaint but no apparent (mild swelling)	1
swelling obvious but not generalized, only localized to few joints(Moderate swelling)	2
obvious swelling localized to multiple joints and also body parts(Severe swelling)	3

ix. *Sandhisula* (Joints pain):	GRADE
No pain	0
mild pain (pain precipitating with heavy work)	1
moderate pain (continuous pain during movement)	2
severe pains (unable to do any work)	3

Table 2

Complete remission	**symptoms shifted from severity of symptoms towards normalcy**
Moderate remission	**symptoms shifted from severity of symptoms towards minimal**
Mild remission:	**symptoms shifted from severity of symptoms towards mild**
No relief	**No shift of clinical symptoms**

B. Pain assessment on VAS:



Deha Prakriti: A proforma was specially designed based on classical texts to identify an individual of a particular *Prakriti* as per methods described by Dubey and Singh (1970) for *Prakriti* assessment.

Statistical Analysis

The collected data was transferred on master chart showing various items/variables in columns and subjects in rows. The analysis of data was done using statistical software SPSS version 13.0.

Intra-group (within the group) comparison:

To test the significance of mean of difference of paired observations (BT versus AT) paired t test was applied.

Inter-group comparison (Between the groups):

To test the significance of difference of means of two independent groups, unpaired t test (independent sample t test) was applied.

Corresponding to t value, p-value was determined.

$p < 0.01$ or $p < 0.01$: highly significant

$p < 0.05$: significant

$p > 0.05$: not significant

In the present study intra group, comparison was done at base line (BT) and at the end of trial (F3), whereas for between the groups comparison difference of BT and F3 was used instead of separately comparing means at BT and F3. It is to be noted here that for intergroup comparison of more than two groups one-way ANOVA (F-test) followed by appropriate Post-hoc multiple comparison test is used. Similarly, for intra group comparison ANOVA may be used as generalization of paired t test. When the observed data do not satisfy the assumptions of parametric test then corresponding non-parametric test may be applied or data transformation may be done. Non-parametric test corresponding to paired t test and unpaired t test are

Wilcoxon signed rank test and Wilcoxon-Signed Ranks test respectively. Similarly, for one-way ANOVA and repeated measure ANOVA and non-parametric test are Kruskal-Wallis test and Friedman test respectively.

OBSERVATION

Table 3: Clinical symptoms presented during patient enrolment -

Clinical symptoms	Group A	Group B	Group C	Total
Pain	20 (100%)	17 (85%)	15 (75%)	52 (86.66%)
Angamarda	19 (95%)	19 (95%)	18 (90%)	56 (93.33%)
Aruchi	13 (65%)	16 (80%)	12 (60%)	41 (68.33%)
Trishna	10 (50%)	13 (65%)	12 (60%)	35 (58.33%)
Àlasyam	17 (85%)	20 (100%)	20 (100%)	57 (95%)
Gauravam	13 (65%)	18 (90%)	18 (90%)	49 (81.66%)
Jvara	14 (70%)	14 (70%)	12 (60%)	40 (66.66%)
Apaka	11 (55%)	8 (40%)	8 (40%)	27 (45%)
Shunyatanganama	18 (90%)	20 (100%)	19 (95%)	57 (95%)

RESULT

Table 4: Change in *Angamard* (stiffness) in patients of *Amavata* (n=54)

Group (N=54)	Grading	*Angamard*								Within the group comparison (Friedman-test)
		BT		F_1		F_2		F_3		
		No.	%	No.	%	No.	%	No.	%	
A n=18	0	1	5.56	0	0.00	8	44.44	14	77.78	χ^2 = 46.706 p = 0.000 (HS)
	1	1	5.56	9	50.00	8	44.44	4	22.22	
	2	6	33.33	8	44.44	2	11.11	0	0.00	
	3	10	55.56	1	5.56	0	0.00	0	0.00	
B n=18	0	1	5.56	3	16.67	4	22.22	10	55.56	χ^2 = 32.523 p = 0.000 (HS)
	1	1	5.56	6	33.33	8	44.44	8	44.44	
	2	5	27.78	7	38.89	6	33.33	0	0.00	
	3	11	61.11	2	11.11	0	0.00	0	0.00	
C n=18	0	2	11.11	4	22.22	2	11.11	10	55.56	χ^2 = 25.783 p = 0.000 (HS)
	1	3	16.67	9	50.00	10	55.56	8	44.44	
	2	9	50.00	4	22.22	6	33.33	0	0.00	
	3	4	22.22	1	5.56	0	0.00	0	0.00	
Between the group comparison (Chi-square test)		χ^2 = 6.840 P = .336		χ^2 = 6.333 P = .387		χ^2 = 6.593 P = .159		χ^2 = 2.541 P = .281		

Table 5: Change in *Aruchi* in patients of *Āmavāta* (n=54)

Group (N=54)	Grading	*Aruchi*								Within the group comparison (Friedman-test)
		BT		F_1		F_2		F_3		
		No.	%	No.	%	No.	%	No.	%	
A n=18	0	7	38.89	11	61.11	15	83.33	17	94.44	χ^2 = 25.326 p = .000 (HS)
	1	5	27.78	5	27.78	3	16.67	1	5.56	
	2	5	27.78	2	11.11	0	0.00	0	0.00	
	3	1	5.56	0	0.00	0	0.00	0	0.00	
B n=18	0	4	22.22	6	33.33	13	72.22	16	88.89	χ^2 = 23.496 p = .000 (HS)
	1	8	44.44	11	61.11	5	27.78	1	5.56	
	2	3	16.67	1	5.56	0	0.00	1	5.56	
	3	3	16.67	0	0.00	0	0.00	0	0.00	
C n=18	0	8	44.44	10	55.56	10	55.56	16	88.89	χ^2 = 11.495 p = .009 (HS)
	1	4	22.22	6	33.33	8	44.44	2	11.11	
	2	4	22.22	1	5.56	0	0.00	0	0.00	
	3	2	11.11	1	5.56	0	0.00	0	0.00	
Between the group comparison (Chi-square test)		χ^2 = 4.398 p.=.623		χ^2 = 6.874 P = .333		χ^2 = 3.375 P = .185		χ^2 = 2.541 P = .637		

Table 6: Change in *Trishna* index in 54 patients of *Amavata*

Group (N=54)	Grading	*Trishna*								Within the group comparison (Friedman-test)
		BT		F_1		F_2		F_3		
		No.	%	No.	%	No.	%	No.	%	
A n=18	0	10	55.56	11	61.11	14	77.78	16	88.89	χ^2 = 13.373 p = .004 (S)
	1	6	33.33	6	33.33	3	16.67	2	11.11	
	2	2	11.11	1	5.56	1	5.56	0	0.00	
	3	0	0.00	0	0.00	0	0.00	0	0.00	
B n=18	0	7	38.89	11	61.11	14	77.78	17	94.44	χ^2 = 24.592 p = .000 (HS)
	1	7	38.89	7	38.89	4	22.22	1	5.56	
	2	4	22.22	0	0.00	0	0.00	0	0.00	
	3	0	0.00	0	0.00	0	0.00	0	0.00	
C n=18	0	8	44.44	12	66.67	13	72.22	17	94.44	χ^2 = 17.074 p = .001 (S)
	1	8	44.44	6	33.33	5	27.78	1	5.56	
	2	2	11.11	0	0.00	0	0.00	0	0.00	
	3	0	0.00	0	0.00	0	0.00	0	0.00	
Between the group comparison (Chi-square test)		χ^2 = 1.846 p= .764		χ^2 = 2.164 p= .706		χ^2 = 2.549 p= .636		χ^2 = 0.540 p= .763		

Table 7: Showing change in *Alasya* index in patients of *Amavata* (n=54)

Group (N=54)	Grading	*Ālasyam*								Within the group comparison (Friedman-test)
		BT		F_1		F_2		F_3		
		No.	%	No.	%	No.	%	No.	%	
A n=18	0	3	16.67	4	22.22	8	44.44	10	55.56	χ^2 = 15.200 p = .001 (s)
	1	10	55.56	11	61.11	10	55.56	8	44.44	
	2	3	16.67	3	16.67	0	0.00	0	0.00	
	3	2	11.11	0	0.00	0	0.00	0	0.00	
B n=18	0	0	0.00	5	27.78	9	50.00	11	61.11	χ^2 = 20.578 p = .000 (HS)
	1	12	66.67	8	44.44	7	38.89	6	33.33	
	2	3	16.67	5	27.78	2	11.11	1	5.56	
	3	3	16.67	0	0.00	0	0.00	0	0.00	
C n=18	0	0	0.00	5	27.78	8	44.44	10	55.56	χ^2 = 19.633 p = .000 (HS)
	1	10	55.56	7	38.89	6	33.33	6	33.33	
	2	5	27.78	6	33.33	4	22.22	2	11.11	
	3	3	16.67	0	0.00	0	0.00	0	0.00	
Between the group comparison (Chi-square test)		χ^2 = 7.227 p= .300		χ^2 = 2.143 p= .710		χ^2 = 5.210 p= .266		χ^2 = 7.820 p= .098		

Table 8: Showing changes in *Gauravam* (feeling of heaviness) in patients of *Amavata* (n=54)

Group N=54	Grading	*Gauravam*								Within the group comparison (Friedman-test)
		BT		F_1		F_2		F_3		
		No.	%	No.	%	No.	%	No.	%	
A n=18	0	7	38.89	8	44.44	14	77.78	17	94.44	χ^2 = 25.352 p = 0.000 (HS)
	1	5	27.78	8	44.44	3	16.67	1	5.56	
	2	6	33.33	2	11.11	1	5.56	0	0.00	
	3	0	0.00	0	0.00	0	0.00	0	0.00	
B n=18	0	2	11.11	5	27.78	7	38.89	12	66.67	χ^2 = 34.091 p = 0.000 (HS)
	1	3	16.67	7	38.89	6	33.33	5	27.78	
	2	13	72.22	6	33.33	5	27.78	1	5.56	
	3	0	0.00	0	0.00	0	0.00	0	0.00	
C n=18	0	2	11.11	6	33.33	8	44.44	9	50.00	χ^2 =27.291 p = 0.000 (HS)
	1	4	22.22	5	27.78	5	27.78	8	44.44	
	2	12	66.67	7	38.89	5	27.78	1	5.56	
	3	0	0.00	0	0.00	0	0.00	0	0.00	
Between the group comparison (Chi-square test)		χ^2 = 4.237 p= .375		χ^2 = 6.875 p= .143		χ^2 = 8.865 p= .065		χ^2 = 1.475 p= .831		

Table 9: Change in *Jvara* (fever) in patients of *Amavata* (n=54)

Group (n=54)	Grading	*Jvara*								Within the group comparison (Friedman-test)
		BT		F_1		F_2		F_3		
		No.	%	No.	%	No.	%	No.	%	
A (n=18)	0	6	33.33	9	50.00	15	83.33	16	88.89	χ^2 = 26.037 p = .000 (HS)
	1	7	38.89	9	50.00	3	16.67	2	11.11	
	2	5	27.78	0	0.00	0	0.00	0	0.00	
	3	0	0.00	0	0.00	0	0.00	0	0.00	
B(n=18)	0	6	33.33	7	38.89	14	77.78	14	77.78	χ^2 = 13.255 p =.004 (S)
	1	9	50.00	9	50.00	2	11.11	2	11.11	
	2	3	16.67	1	5.56	2	11.11	1	5.56	
	3	0	0.00	1	5.56	0	0.00	1	5.56	
C(n=18)	0	8	44.44	10	55.56	16	88.89	16	88.89	χ^2 = 14.392 p = .002 (S)
	1	7	38.89	7	38.89	1	5.56	1	5.56	
	2	3	16.67	0	0.00	1	5.56	1	5.56	
	3	0	0.00	1	5.56	0	0.00	0	0.00	
Between the group comparison (Chi-square test)		χ^2 = 1.475 p= .831		χ^2 = 3.858 p= .696		χ^2 = 3.133 p= .536		χ^2 = 3.574 p= .734		

Table 10: Change in *Apaka* index in patients of *Amavata* (n=54)

Group (n=54)	Grading	*Apaka*								Within the group comparison (Friedman-test)
		BT		F_1		F_2		F_3		
		No.	%	No.	%	No.	%	No.	%	
A(n=18)	0	9	50.00	12	66.67	13	72.22	17	94.44	χ^2 = 15.000 p = .002 (S)
	1	4	22.22	5	27.78	4	22.22	1	5.56	
	2	4	22.22	1	5.56	1	5.56	0	0.00	
	3	1	5.56	0	0.00	0	0.00	0	0.00	
B(n=18)	0	12	66.67	16	88.89	17	94.44	18	100.00	χ^2 =8.778 p = .032 (S)
	1	5	27.78	2	11.11	1	5.56	0	0.00	
	2	1	5.56	0	0.00	0	0.00	0	0.00	
	3	0	0.00	0	0.00	0	0.00	0	0.00	
C(n=18)	0	12	66.67	16	88.89	17	94.44	18	100.00	χ^2 =37.667 p = .000 (HS)
	1	5	27.78	2	11.11	1	5.56	0	0.00	
	2	1	5.56	0	0.00	0	0.00	0	0.00	
	3	0	0.00	0	0.00	0	0.00	0	0.00	
Between the group comparison (Chi-square test)		χ^2 = 5.688 p=.459		χ^2 = 4.727 p=.316		χ^2 = 5.681 p= .224		χ^2 = 2.038 p=.361		

Table 11: Change in *Shuntangam* index in patients of *Amavata* (n=54)

Group (n=54)	Grading	*Shuntangam*								Within the group comparison (Friedman-test)
		BT		F_1		F_2		F_3		
		No.	%	No.	%	No.	%	No.	%	
A(n=18)	0	2	11.11	3	16.67	14	77.78	16	88.89	χ^2 = 23.282 p = .000 (HS)
	1	2	11.11	8	44.44	4	22.22	2	11.11	
	2	8	44.44	6	33.33	0	0.00	0	0.00	
	3	6	33.33	1	5.56	0	0.00	0	0.00	
B(n=18)	0	0	0.00	1	5.56	6	33.33	13	72.22	χ^2 = 11.857 p = .008 (S)
	1	3	16.67	5	27.78	7	38.89	4	22.22	
	2	7	38.89	8	44.44	4	22.22	1	5.56	
	3	8	44.44	4	22.22	1	5.56	0	0.00	
C(n=18)	0	1	5.56	1	5.56	4	22.22	11	61.11	χ^2 = 12.767 p = .005 (S)
	1	2	11.11	6	33.33	7	38.89	4	22.22	
	2	7	38.89	5	27.78	6	33.33	3	16.67	
	3	8	44.44	6	33.33	1	5.56	0	0.00	
Between the group comparison (Chi-square test)		χ^2 = 1.254 p= .974		χ^2 = 4.036 p= .672		χ^2 = 17.096 p= .009 (HS)		χ^2 = 5.126 p= .275		

Table 12: Changes in Joints pain in patients of *Amavata* (n=54)

Group (N=54)	Grading	Joints pain (subjective)								Within the group comparison (Friedman-test)
		BT		F_1		F_2		F_3		
		No.	%	No.	%	No.	%	No.	%	
A n=18	0	0	0.00	0	0.00	5	27.78	7	38.89	χ^2 = 35.261 p = .000 (HS)
	1	4	22.22	8	44.44	7	38.89	8	44.44	
	2	8	44.44	8	44.44	6	33.33	3	16.67	
	3	6	33.33	2	11.11	0	0.00	0	0.00	
B n=18	0	3	16.67	5	27.78	7	38.89	9	50.00	χ^2 = 35.738 p = .000 (HS)
	1	5	27.78	6	33.33	6	33.33	7	38.89	
	2	3	16.67	5	27.78	5	27.78	2	11.11	
	3	7	38.89	2	11.11	0	0.00	0	0.00	
Cn=18	0	5	27.78	5	27.78	5	27.78	6	33.33	χ^2 =45.0 p = .000 (HS)
	1	3	16.67	3	16.67	4	22.22	5	27.78	
	2	8	44.44	6	33.33	6	33.33	4	22.22	
	3	2	11.11	4	22.22	3	16.67	3	16.67	
Between the group comparison (Chi-square test)		χ^2 = 9.241 P = .322		χ^2 = 19.653 p = .058		χ^2 = 4.494 p = .610		χ^2 = 22.069 P= 0.089		

3. Therapeutic Profile

Table 13: Change in Pain (VAS) titer in patients of *Amavata* (n=54)

Groups	PAIN (VAS)		Mean ± SD	Within the group comparison Paired t-test (BT-AT)
	BT	AT	% decrease in mean	
A(n=18)	6.647 ± 1.169	2.058 ± 0.747	69.03	4.588 ± 1.371 t = 13.789, p = 0.000(HS)
B(n=18)	5.500 ± 1.977	1.555 ± 1.756	71.72	3.944 ± 1.764 t = 9.483, p = 0.000(S)
C(n=18)	4.866 ± 1.552	1.866 ± 0.833	61.65	3.000 ± 0.845 t = 13.748, p = 0.00(S)
Between the group comparison on way ANOVA	F = 5.077 P = 0.010	F = 0.747 P = 0.479		
Post Hoc Test for significant pairs	C, A	-	-	

Table 14: Change in RA titer in patients of *Amavata* (n=54)

Change in RA titer in patients of *Amavata* (n=54)Group	RA titer ± SD		Mean	Within the group comparison Wilcoxon Signed ranks test (BT-AT)
	BT	AT	% decrease in mean	
A(n=18)	90.42 ± 65.62	25.78 ± 17.16	71.48	Z=-3.682 p = 0.000
B(n=18)	65.42 ± 55.57	21.89 ± 18.31	66.53	Z=-3.463 p = 0.001
C(n=18)	59.99 ± 56.64	31.64 ± 38.84	47.25	Z=-2.912 p = 0.004
Between the group comparison on way Kruskal-Wallis test	χ2= 225182 P = 0.325 (NS)	χ2= 0.179 P = 0.9145 (NS)		

Table 15: Change in CRP titer in 54 patient of *Amavata*

Groups	CRP titer Mean ± SD			Within the group comparison Paired t-test (BT-AT)
	BT	AT	% decrease in mean	
A(n=18)	4.56 ± 3.38	0.569 ± 0.426	87.52	Z=-3.726 p = 0.000
B(n=18)	5.67 ± 6.04	0.947 ± 0.784	83.29	Z=-3.376 p = 0.001
C(n=18)	5.91 ± 6.68	2.32 ± 5.46	60.74	Z=-3.575 p = 0.000
Between the group comparison One way Kruskal-Wallis test	χ^2= 0.102 P = 0.950	χ^2= 1.738 P = 0.232		

Table 16: Change in ESR titer in patients of *Amavata* (n=54)

Groups	ESR Mean ± SD			Within the group comparison Wilcoxon Signed ranka test (BT-AT)
	BT	AT	% decrease in mean	
A(n=18)	58.39 ± 16.14	21.83 ± 6.13	62.61	Z=-3.728 p = 0.000
B(n=18)	58.28 ± 11.79	21.94 ± 8.228	62.35	Z=-3.728 p = 0.000
C(n=18)	57.44 ± 21.57	23.72 ± 16.59	58.70	Z=-3.724 p = 0.000
Between the group comparison one way Kruskal-Wallis test	χ^2 = 0.268 P = 0.874	χ^2 = 0.908 P = 0.635		

Table 17: Improvement in terms of percentage in clinical symptoms of RA

S.No.	Symptoms	Group	Complete remission		Moderate remission		Mild remission		No remission	
			No.	% of improvement	No.	% of improvement	No.	% of improvement	No.	% of improvement
1.	Joint pain	A (100%)	7	38.89	8	44.44	3	16.67	0	0.00
		B (83.33%)	6	33.33	7	38.89	2	11.11	0	0.00
		C (72.22%)	1	5.56	5	27.78	4	22.22	3	16.67
2.	*Angamarda*	A (94.44%)	13	72.22	4	22.22	0	0.00	0	0.00
		B (94.44%)	9	50.00	8	44.44	0	0.00	0	0.00
		C (88.89%)	8	44.44	8	44.44	0	0.00	0	0.00
3.	*Aruchi*	A (61.11%)	10	55.56	1	5.56	0	0.00	0	0.00
		B (77.78%)	12	66.67	1	5.56	1	5.56	0	0.00
		C (55.56%)	8	44.44	2	11.11	0	0.00	0	0.00
4.	*Trishana*	A (44.44%)	6	33.33	2	11.11	0	0.00	0	0.00
		B (61.11%)	10	55.56	1	5.56	0	0.00	0	0.00
		C (55.56%)	9	50.00	1	5.56	0	0.00	0	0.00
5.	*Alasyam*	A (83.33%)	7	38.89	8	44.44	0	0.00	0	0.00
		B (100%)	11	61.11	6	33.33	1	5.56	0	0.00
		C (100%)	10	55.56	6	33.33	2	11.11	0	0.00
6.	*Gauravam*	A (61.11%)	10	55.56	1	5.56	0	0.00	0	0.00
		B (88.89%)	10	55.56	5	27.78	1	5.56	0	0.00
		C (88.89%)	7	38.89	8	44.44	1	5.56	0	0.00
7.	*Jvara*	A (66.67%)	10	55.56	2	11.11	0	0.00	0	0.00
		B (66.67%)	8	44.44	2	11.11	1	5.56	1	5.56
		C (55.56%)	8	44.44	1	5.56	1	5.56	0	0.00
8.	*Apaka*	A (50%)	8	44.44	1	5.56	0	0.00	0	0.00
		B (33.33%)	6	33.33	0	0.00	0	0.00	0	0.00
		C (33.33%)	6	33.33	0	0.00	0	0.00	0	0.00
9.	*Shuntaangana m*	A (88.89%)	14	77.78	2	11.11	0	0.00	0	0.00
		B (100%)	13	72.22	4	22.22	1	5.56	0	0.00
		C (94.44%)	10	55.56	4	22.22	3	16.67	0	0.00

Table 4, 5, 8, 12 shows the distribution of subjects at initial level and subsequent follow- ups according to severity of ***Angamard, Aruchi, Gauravam,*** **joint Pain** respectively grade among the study group.In all three groups **Group A, Group B, Group C;** the change in severity of ***Angamard, Aruchi, Gauravam,*** **joint Pain**was statistically **highly significant(P<0.001)**.

Intergroup comparison of above parameters (***Angamard, Aruchi, Gauravam,*** **joint Pain**) was statistically **notsignificant** initially as well as at each follow up.

Table 6 shows the distribution of subjects at initial level and subsequent follow-ups according to severity of ***Trishna***grade among the study group.In **Group A and Group C** showed relief at different follow ups and at the third followup the change in severity of *Trishna* was statistically **significant (P<0.005)**. But, in **Group B**the change in severity of *Trishna* was statistically **highly significant (P<0.001)**.

Intergroup comparison was not statistically significant initially as well as at each follow up.

Table 7 shows the distribution of subjects at initial level and subsequent follow-ups according to severity of ***Alasyam***grade among the study group.In **Group B** and **Group C** showed relief at different follow ups and at the third followup the change in severity of ***Alasyam*** was statistically **highly significant (P<0.001)**. But, in **Group A** the change in severity of *Alasyam was* statistically **significant (P<0.005)**.

Intergroup comparison was not statistically significant initially as well as at each follow up.

Table 9 shows the distribution of subjects at initial level and subsequent follow-ups according to severity of ***Jvara***grade among the study group.In **Group A**the change in severity of *Jvara*was statistically **highly significant (P<0.001)**. In **Group B**and **Group C** the change in severity of *Jvara* was statistically **significant (P<0.005)**. Intergroup comparison was not statistically significant initially as well as at each follow up.

Table 10 shows the distribution of subjects at initial level and subsequent follow-ups according to severity of *Apaka* grade among the study group.In **Group A and Group B** showed relief at different follow ups and at the third followup the change in severity of ***Apaka*** was statistically **significant (P<0.005)**. But, in **Group C** the change in severity of *Alasyam was* statistically **highly significant (P<0.001)**.

Intergroup comparison was not statistically significant initially as well as at each follow up.

Table 11 shows the distribution of subjects at initial level and subsequent follow-ups according to severity of ***Shunatanganam*** grade among the study group.In **Group A** showed relief at different follow ups and at the third followup the change in severity of ***Shunatanganam*** was statistically **highly significant (P<0.001)**. But, in **Group B and Group C** the change in severity of *Alasyam was* statistically **significant (P<0.005)**.

Intergroup comparison was not statistically significant initially as well as at each follow up except at second follow up, which showed statistically significant difference.

Table 13: Mean decrease in Pain (VAS) in **Group A** was 4.588 which were statistically mild to moderate **significant**. Similarly in **Group B & Group C** it was 3.944 & 3.000 respectively & both were statistically (less than Group A) significant. **(p= <0.05).** The percentage improvement in mean of pain was higher in group B (71.72%), then in group A (69.03%) followed by group C (61.65%). Thus the efficacy of treatment given to different trial groups was in order of **group B > group A > group C.**

The intergroup comparison on difference of BT & AT was **significant** in all the study groups.

Table 14 Before treatment RA factor in terms of mean ± SD Group A, Group B and Group C was 90.42±65.62, 65.42±55.57 and 59.99±56.62, respectively which were reduced after treatment to 25.78±17.16, 21.89±18.31 and 31.64±38.84 in Group A, Group B and Group C, respectively.

Table 15 Before treatment CRP in terms of mean ± SD in Group A, Group B and Group C was 4.56±3.38, 5.67±6.04 and 5.91±6.68, respectively which were reduced after treatment to 0.569±0.426, 0.947±0.784 and 2.32±5.46 in Group A, Group B and Group C, respectively.

Table 16 Before treatment ESR in in terms of mean ± SD in Group A, Group B and Group C was 58.39±16.14, 58.28±11.79 and 57.44±21.57, respectively which reduced after treatment to 21.83±6.13, 21.94±8.228 and 23.72±16.59 in Group A, Group B and Group C, respectively.

The intergroup comparison of RA Factor, CRP, ESR at BT & AT showed statistically not significant difference at any of the follow up

The percentage improvement in mean of RA titer, CRP and ESR was higher in group A then in group B followed by

group C (47.25%). Thus the efficacy of treatment given to different trial groups was in order of **group A > group B > group C.**

DISCUSSION

Probable Mode of Action of *ShunthiChurna*

Based on the Pharmacological Action

In Ayurvedic classic *Sunthi Churna* is described as *Katu rasa*, *Ushna Virya* and *Madhur Vipaka* and having *Laghu*, *Snigdha* properties which is well known for their *Vedanasthapana*, *Vatanulomana*, *Hridya*, *Kapha ghna*, *Swasaghna*, *Vrisya*, *Balya*, *Rucikaraka*, *Pandughna*.

Ginger *(Shunthi)* [5,6,7,8,9] suppresses prostaglandin synthesis through inhibition of cyclooxygenase- 1 and cyclooxygenase-2. An important extension of this early work was the observation that ginger also suppresses leukotriene biosynthesis by inhibiting 5-lipoxygenase. This pharmacological property distinguishes ginger from non-steroidal anti-inflammatory drugs. This discovery preceded the observation that dual inhibitors of cyclooxygenase and 5-lipoxygenase may have a better therapeutic profile and have fewer side effects than non-steroidal anti-inflammatory drugs.In an experimental trial of male swiss mice and male Wistar rats for the evaluation of antinociceptive and anti inflammatory effects respectively, they found that ginger essential oil showed significant activities on the same. These recent evidences suggest that *Sunthi* play an important role to reduce the progression of disease.

Probable Mode of Action of *Rasnadi Guggulu* in *Amavata* [10]

Rasnadi Guggulu (contents Rasna, *Guduchi*, *Erand*, *Devdaru*, *Shunthi Guggulu*) is well known for their *Sothaghna*, *Svasahara*, *Vataraktaghna*, *Vatashulahara*, *Udarrogahara*, *Kasaghna*, *Jvaraghna*, *Visaghna*, *Vrana*, *Bhagandar* and *shiroroga* **[11,12]**. So considering all the above properties of each drug mentioned above in work effectively as a whole in the disease *Amavata*.

***Rasna*[13,14,15]**-The ethanolic extract of Pluchea lanceolata exhibited significant anti-inflammatory activity. An important clinical difference was that the plant extract suppressed the delayed periarticular changes more as compared to the acute inflammatory phase. Another study on the therapeutic aspect of Pluchea lanceolata was the comparison of the water-soluble fraction of the alcoholic extract with the nonsaponifiable steroidal fraction, the test system being carrageenan-produced hind paw swelling in albino rats. While the former extract did not show significant activity, the latter steroidal fraction was significantly anti-inflammatory in action. However, it had not much effect on the granuloma pouch. The anti-inflammatory

potential of some Ayurvedic compositions containing Pluchea lanceolata extract was tested on experimental arthritis and granuloma pouch. They showed marked anti-inflammatory activity in both models. In experimental arthritis, a decoction of the plant has been reported to prevent the swelling of joints.

Erand **[16,17]**:The methanolic extract of *Erand* leaves (250 and 500 mg/kg) had shown the anti inflammatory activities in both the acute and sub chronic models in wistar rats. Anti-inflammatory and anti-arthritic activity effect might be speculated due to phytochemicals present such as flavanoid and saponin.

Guggulu **[18]**: *The* aqueous extract of *Guggulu* significantly inhibited both the maximal oedema response and the total oedema response during six hours; fraction containing gum *Guggulu* in experimental arthritis decreased the thickness of the joint swelling during the course of drug treatment. *Guggulu* sterone also appear to reduce circulating levels of pro-inflammatory cytokines and markers such as IL-1, IL-2 and TNF α (Manjula N *etal*., 2000). It also able to reduce cyclo-oxygenase-2 mRNA levels and suppress its TNFα mediated induction (Ly N *etal*. 2008 and Sisodia S, Agarwal BB, 2004)

Deodaru **[19,20,21,22]**: Chemically standardized isolates from *Cedrus deodaru* stem wood having anti-cancerous and anti-inflammatory activity. The volatile oil extract showed significant inhibition of carrageenan induced rat paw edema.

Guduchi **[23]**:A study showed that the aqueous extract of Tinospora cordifolia has significant analgesic and anti-inflammatory activities. In another study, the extract of aerial parts of Tinospora cordifolia produced a significant increase in pain threshold in hotplate and tail flick tests in a dose dependent manner. In acetic acid-induced writhing the extract produced significant inhibition of writhing reaction.

Probable Mode of Action of *Valuka Sveda* [24,25] in *Amavata*

The *Valuka Sveda* does three main actions by its *Ruksha* and *Ushna Guna*.

- *Svedna* does the *Pakwata* of *Ama*.
- *Sroto Mukha Vishodana* i.e. it helps the *Pakwa Dosha* to come to *Kostha* from *Shakha*.
- *Vayuscha Nigraha* i.e. it regulates movements of *Vata*.

With these main functions *Valuka Sveda* does *Amapachana*, *Sandhi Shoola Nashak*, *Sandhi Shotha Nashak*, *Gatra Stabdhata Nashak* etc. in the disease ***Amavata***.

While concluding, the drugs were selected in a view of its traditional use and also on the basis of its Ayurvedic pharmcodyanamic properties in terms of

Rasa, Guna, Virya and *Vipaka*. The selected drug *Shunthi* **[26]** *(Gingiber officinale*) imparts *Rasayana* effect in the body, pacify the vitiated *Ama, Vata* and *Kapha dosha* and calm down pain and fever, while in the *Rasnadi Guggulu* most of the contents having *Katu & Tikta rasa* imparting *Amapacana, Vatanulomana & Shulaghna* properties along with *Rasayana* effect. Under local therapeutics measures, *Ruksa Sveda* is chosen for this purpose because it locally digests *Ama & Kapha Dohsa*, removed the stiffness of the joints and widely prescribed by learned scholars of *Ayurveda*.

The whole study is comprises into three groups on the basis of three dominant sets of *Deha Prakriti*. At the end of trial, it was observed that patients of *Vata* and *Pitta* dominant sets of *Deha Prakriti* responded better in clinical symptoms, pain on VAS, RA, CRP, and ESR, while the patients of *Kapha* dominant set of *Prakriti* responded least. Probably this is due to biophysical similarity of *Ama* with the some involvement of *Dushya* along with similarity of *Kapha* dominant of *Prakriti*.

CONCLUSION

The selected trial drugs and measures are effective in cases of RA (*Amavata*). The drug is ideally suited to bring down the observed clinical symptoms of RA, elevated level of RA, CRP, ESR and pain on VAS. Thus it exerts *Dosha Shamak* effect and attainment towards normalcy. Thus, by virtue of its pharmacological properties as described by ancient scholars the selected drugs and measures possess ability to improve the symptoms of *Amavata* patients. This indicates and warrants Ayurvedic fraternity to manage *Amavata* with different angle to add or reduce the drugs and measures on the basis of *Prakriti* of the individual. Probably this is the reason that *Ayurveda* has advocated for personalized medicine for thousands of years. The major concept of health and disease revolve around uniqueness of the individual i.e. *Purusham Purusham Veekshya Sa Geya Bhisgottamama* **[27]**.

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REFERENCE

[1] The Global Burden of Rheumatoid Arthritis: Estimates From the Global Burden of Disease 2010 Study - PubMed. Accessed July 2, 2020. https://pubmed.ncbi.nlm.nih.gov/24550173/

[2] Epidemiology of, risk factors for, and possible causes of rheumatoid arthritis - UpToDate. Accessed June 30, 2020. https://www.uptodate.com/contents/epidemiology-of-risk-factors-for-and-possible-causes-of-rheumatoid-arthritis

[3] Singh A. K, Pandey A.K.Clinical observation of effect of Rasnadi Guggulu, Shunthi churna and Ruksha sweda in the management of Amavata vis-a-vis RA under the influence of dominant sets of Deha Prakriti, MD (Ay) Kayachikitsa thesis, IMS, Banaras Hindu University, Varanasi, UP, India. (2014)

[4] Madhavakara, Madhav Nidana, Madhukosha Sanskrit Commentary by Vijayraksita, Srikanthadatta and Vidyotini Hindi Commentary by Sudarsana Sastri . Revised Edition Reprint-2009Edited by Yadunandana Upadhyaya, Chaukhambha Prakashana Varanasi, Nidana Sthana 25/5, Pg no.509

[5] Young H, Luo Y, Cheng H,WH-J of, 2005 undefined. Analgesic and anti-inflammatory activities of -gingerol. Elsevier. Accessed July 3, 2020. https://www.sciencedirect.com/science/article/pii/S0378874104004404

[6] Srivastava K, hypotheses TM-M, 1992 undefined. Ginger (Zingiber officinale) in rheumatism and musculoskeletal disorders. Elsevier. Accessed July 3, 2020. https://www.sciencedirect.com/science/article/pii/030698779290059L

[7] Thomson M, Al-Qattan K, acids SA-S-… and essential fatty, 2002 undefined. The use of ginger (Zingiber officinale Rosc.) as a potential anti-inflammatory and antithrombotic agent. Elsevier. Accessed July 3, 2020. https://www.sciencedirect.com/science/article/pii/S0952327802904412

[8] Grzanna R, Lindmark L, Frondoza CG. Ginger - An herbal medicinal product with broad anti-inflammatory actions. J Med Food. 2005;8(2):125-132. doi:10.1089/jmf.2005.8.125

[9] Mahto R, Shukla V, Dave A. A comparative study of Rasona Rasnadi Ghanavati and Simhanada Guggulu on Amavata with special reference to Rheumatoid arthritis. AYU (An Int Q J Res Ayurveda). 2011;32(1):46. doi:10.4103/0974-8520.85724

[10] Sen T, Chaudhuri A. Anti-Inflammatory and Antiulcer Actions of Pluchea indica. Planta Med.

1989;55(07):647-647. doi:10.1055/s-2006-962229

[11] Sharma PV. *Dravyaguṇavijñāna, Vol-II*, p331-335 Varanasi, India: Publisher Chaukhambha Bharati Academy, reprint 2011.

[12] The Ayurvedic Pharmacopeia of India, part 1, vol.1, p138-139

[13] Sen T, Chaudhuri A. Anti-Inflammatory and Antiulcer Actions of Pluchea indica. Planta Med [Internet]. 1989 [cited 2020 Jul 3];55(07):647–647. Available from: https://www.thieme-connect.com/products/ejournals/pdf/10.1055/s-2006-962229.pdf

[14] Buapool D, Mongkol N, Chantimal J, SR-J of, 2013 undefined. Molecular mechanism of anti-inflammatory activity of Pluchea indica leaves in macrophages RAW 264.7 and its action in animal models ofinflammation. Elsevier. Accessed July 3, 2020. https://www.sciencedirect.com/science/article/pii/S0378874113000251

[15] Barros I, Lopes L, MB-J of, 2006 undefined. Anti-inflammatory and anti-nociceptive activities of Pluchea quitoc (DC.) ethanolic extract. Elsevier. Accessed July 3, 2020. https://www.sciencedirect.com/science/article/pii/S0378874106000456

[16] Chand G, Morya K. Ayurvedic Approach of Eranda (Ricinus communis Linn.) on Vata Vyadhi for Green Pharmacology. researchgate.net. Accessed July 3, 2020. www.researchtrend.net

[17] Rana M, Dhamija H, Prashar B, Sharma S. Ricinus Communis L.—a Review. Vol 4. Accessed July 3, 2020. https://www.doc-developpement-durable.org/file/Plantes-Medicinales-Aromatiques/FICHES_PLANTES/ricin/RICINUS COMMUNIS_Research Journal Chemistry.pdf

[18] Tomar R, Kaur G, Sannd R, Singh H, Sarkar B. A Review on Guggulu Formulations Used in Ayurveda. Vol 3.

[19] KT S, Gupta S, sciences JL-I journal of pharmaceutical, 1998 undefined. Pharmacodynamic effects of Cedrus deodara wood essential oil. ijpsonline.com. Accessed July 3, 2020. https://www.ijpsonline.com/articles/pharmacodynamic-effects-of-cedrus-deodara-wood-essential-oil.pdf

[20] Liu D-Y, Shi X-F, Wang D-D, Qu-Huan MA, Zhang J-M, Chong LI. A New Flavonoid in Pine Needles of Cedrus deodara. Chinese Herb Med. 2011;3(1):5-6. doi:10.3969/j.issn.1674-6384.2011.01.002

[21] Chaudhary AK, Ahmad S, Mazumder A. P H c o g J. Cedrus deodara (Roxb.) Loud.: A Review on its Ethnobotany, Phytochemical and Pharmacological Profile. Pharmacogn J. Published online 2011:23. doi:10.5530/pj.2011.23.2

[22] Shinde U, Phadke A, … AN-J of, 1999 undefined. Studies on the anti-inflammatory and analgesic activity of *Cedrus deodara* (Roxb.) Loud. wood oil. Elsevier. Accessed July 3, 2020. https://www.sciencedirect.com/science/article/pii/S0378874198001500

[23] Mittal J, Sharma MM, Batra A. Tinospora cordifolia: a multipurpose medicinal plant-A review (chemical constituents). J Med Plants Stud Year J Med Plants Stud. 2014;2(2):32-47. www.plantsjournal.com

[24] Lakkundi SS. "A Comparative Study Of Valuka Sweda And Shatapushpadi Lepa In Janu Sandhi Shula, Shotha And Stabdata W.S.R Amavata" Department of Post Graduate Studies in Panchakarma, Government Ayurvedic Medical College Bangalore Department Of Post Graduate Studies In Panchakarma Government Ayurvedic Medical College. Published online 2012. Accessed July 3, 2020. http://52.172.27.147:8080/jspui/handle/123456789/7359

[25] Basotra S, Proff A, Sharma R. A Case Study On Role of Baluka Sweda And Ajmodadi Churna In The Management of Amavata (Rheumatoid Arthritis). worldwidejournals.org. doi:10.36106/ijar

[26] Rudrappa Harisha C, Shukla VJ. A Pharmacognostical and Physico-Chemical Analysis of Shunthi Churna (*Zingiberofficinale*) and Physico-Chemical Analysis of Erand Tail (Castor Oil).; 2019. Accessed July 3, 2020. www.thepharmajournal.com

[27] Charaka Samhita Vidyotini Hindi Commentary by Pt. Kashinatha Shastri, , 6th Ed. Edited by Dr. Gangasahayapandey Sutra sthana. 1. Vol. 1. Varanasi: Choukhambha Bharati Academy; 2000.