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"AN OBSERVATIONAL STUDY TO EVALUATE THE ROLE OF ASATHMYAJA NIDANA IN TAMAKA SWASA (ALLERGIC BRONCHIAL ASTHMA)"

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ABSTRACT

Tamaka Swasa is a most common disorder in this era, disturbing daily activities of life, having an impact on physical and mental health causing distress. The disease TamakaSwasa is an age old disease and has been documented in almost all Ayurvedic text. It is one of the main diseases developing due to vitiation of Pranavaha Srotas. Bronchial asthma simulates TamakaSwasa in relation to etiology and clinical presentation. Dyspnoea being the prime manifestation. Vata and Kapha are the two key pathological factors involved in Samprapti of TamakaSwasa. "HridayasyaRasadhinamDhathunamChaUpashoshanou"[2] mentioned in CharakaChikitsaSthana implies the "Corpulmonale" which is a common complication of severe allergic bronchial asthma. It is the development of right ventricular failure due to pulmonary hypertension. This clinical condition has been identified and detailed thousands of years ago by our Acharyas.The AsathmyajaNidanasof SwasaRoga are described as Raja-Dhuma-Pragvata-SheethaSthana-Megha-AnilaSevana. This disease resembles the Allergic Bronchial Asthma in modern science affecting millions of people each year and frequent visits in day to day practice. The environment has greatest influence on the allergic asthma due to the urban trajectory of increasing air .Looking at the alarming rate of Allergic Bronchial Asthma in our society and relative rate of hospital visits, an effective understanding on the AsathmyajaNidana which provoke TamakaSwasa becomes inevitable.The objectives of the study were to evaluate and analyse the AsathmyajaNidana of TamakaSwasa and to study the concept of allergy (Asathmya) in Ayurveda.

INTRODUCTION

With a boon of advancement in science and technologies the 21stcentury is on the surge of newer diseases. The biggest global health treats of this century is the uprising trends in the incidence of allergic conditions and auto- immune diseases. Many epidemiological surveys demonstrate a two-fold increase in the prevalence of allergic conditions during the past two decades. In India, an approximate of 20-30% of the total population suffers from at least one or the other allergic diseases.One among them is the Allergic bronchial asthma a syndrome truly encompassing several distress. The environmental interactions are important to develop asthma. The most recent comprehensive analyses of the GBD (Global Burden of Disease) undertaken in 2008-2010 estimates the number of asthmatics in world as high as 334 millions.Prevalence varies widely between countries and between centre's and was 8.5%.Asthma prevalence is higher in high income countries. Globally asthma is ranked 16th among the leading cause of years lived with disability and 28th among the leading causes of burden of disease. Around 300 million people have asthma worldwide and it is likely that by 2025 a further 100 million may be affected[1].Researchers says that cause of intrinsic asthma is more similar to the cause of intrinsic asthma with the major difference ie) extrinsic asthma is triggered by allergens with elevated IgE whereas intrinsic asthma is non-allergic with less IgE.

The whole sequel of allergic phenomenon is best explained under the concept of Asatmya. Satmya and Asatmya are the two phenomena related to the body response against

external factors either related to food , behavior or environmental factors.Spontaneous or habitual exposure to external factors beget a response which is complimentary to the health is comprehended as Sathmya. The complementary response either immediate or in a long run both are referred by the term Sathmya. A person accustomed to the exposure of different environmental factors either since birth or later in the life.Such factors when complement the health status suggests the Satmya.

Contrary to this the external factors when exhibit a response that are derogatory to the health is called as Asathmya[3]. AsathmyaNidanas has influence over body immune system. Allergens can be included in Asathmya which disturbs the internal homeostasis. The SatmyataBhavavaries person to person and even in Swastha and Rogi. The effect depends upon the SatmyajaBhava of that person. SatmyajaBhava (AtmaSatmya)[4] is unique to each person. National Institute of Environmental Health science says " Subtle differences in genetic factors cause people to respond differently to the same environmental exposure.This explains why some individuals have a fairly low risk of developing a disease as a result of an environmental insult, while others are much more vulnerable. The AtmaAsatmyata is related to genetic predisposition to that allergen.

Asatmyata puts adverse effects on SatmyajaBhava of the body leading to suppression or overactivity of immunity leading to hypersensitivity reactions. SatmyaSampat is one of the BalaVruddhikaraBhava and indulgence of " Satmya" is responsible for Bala. SatmyaSampat is hereditoryas well as acquired by persons Ahara and Vihara.

AsathmyaNidanas are one of the Nidanas taken as exposure to exogenous or endogenous allergens. Allergic bronchial asthma is a growing clinical and public health problem in world-wide, is likely determined by multiple environment and genetic factors. This can be grasped under the umbrella term "Asathmya". Ayurveda literature stresses about AsathmyajaNidanas like Raja, Dhuma, Pragvata,SheethaSthana,Megha, AnilaSevana[5]as precipitating factors of TamakaSwasa. Hence it is the need of the hour to find out the cause for this disease and to avoid exposure to that cause because "Nidana Parivarjanameva Chikitsa". The treatment principles include avoiding adaptation toAtmaAsathmyafactors, treatment withShamanaChikitsa, suppressing Atma Satmya Viruddha response, treatment with Shodhana Chikitsa, preventing theAtmaSathmyaVirudharesponse andRasayanaChikitsareconditioning the body. So the present study is planned to evaluate the role of AsathmyajaNidana inTamakaSwasa.

OBJECTIVES

The objectives of the study were to evaluate and analyse the AsathmyajaNidana of TamakaSwasa and to study the concept of allergy (Asathmya) in Ayurveda

SELECTION OF PATIENTS:

A minimum of 100 patients who were suffering from TamakaSwasa between age group of 16-60 years with the help of special case proforma with the details of history taking , physical signs and symptoms as mentioned in our classics and allied science were selected. Patients were analysed and selected accordingly who fulfils the diagnostic and inclusion criteria. Individual Nidana indulged by the

patient were noted in the case sheetalong with other criteria's specially mentioned in assessment criteria.

STUDY DESIGN:

It was a clinical observational study of minimum of 100 patients suffering fromTamakaSwasa

STATISTICAL ANALYSIS OF THE RESULT:

The data obtained during study was entered and tabulated using the statistical parameter Pearson Chi-Square test.

INCLUSION CRITERIA:

Patient aged between 16-60 yrs.

Patient who were having cardinal symptoms of TamakaSwasa(allergic bronchial asthma).

EXCLUSION CRITERIA:

1. Patients with Pulmonary TB, Cardiac Asthma and Tropical Eosinophilia.
2. Patients with COPD like Chronic bronchitis ,Emphysema and Bronchiectasis.
3. Patients with other systemic disorder.

OBSERVATION AND RESULTS:

TABLE NO : 48 Showing relation between AsathmyajaNidana and IgE

ASATHMYAJA NIDANA	**IgE (P value)**
RAJO SEVANA	**0.001**
DHUMA SEVANA	**0.197**
PRAGVATA	**0.016**
ANILA SEVANA	**0.072**
SHEETHA STHANA	**0.298**
MEGHA	**0.397**

TABLE NO : 48 Showing relation between Objective parameters and IgE

OBJECTIVE PARAMETERS	IgE (P value)
AEC	0.074
PEFR	0.001

DISCUSSION:

1) AGE:

In this study higher incidence of TamakaSwasa is seen in people between age group 16-30 years. It's because in this study higher incidence of patients are diagnosed with extrinsic asthma (atopic asthma) which occurs more often in children and younger adults.

2) SEX:

64% of patients were females, in this study. It may be because asthma prevalence is greater in females than males after puberty. As children, boys have increased prevalence of asthma compared to girls. This change in prevalence around puberty suggests sex hormones and other factors alter the pathway important in asthma pathogenesis.After puberty in females testosterone decreases with increase in estrogen. This female hormone estrogen is a potent activator and alsoknown to drive the lung inflammation.

3) RELIGION:

There is no relation between the disease and the religion. Since the study was conducted in area where Hindu population is high, higher incidence was seen in Hindu religion people.

4) SOCIO-ECONOMIC STATUS:

Higher incidence of the disease is seen in middle class people (70%) followed by rich people. This could be due to urbanization(presence of residence in high polluted areas) and industrialization.

5) OCCUPATION:

It was observed that majority of the patients were house wives(20%),

students (21%), office work (17%) and others like drivers, farmers etc. This is because these employees are having more chance of exposure to allergens like dust, polluted air, house dust, mites, pollens, grain dust etc. It was observed that they have severe symptoms with higher IgE level compared to other patients. The ranges were 2500 IU/ml ,3150 IU/ml,4100 IU/ml etc where the normal range is 1-150 IU/ml. It is also observed that dust is highly irritant and Asatmya to most of the patients.

6) HABITAT:

Study reveals that higher incidence of TamakaSwasa is seen in patients who are residing in urban area (60%). This is because of increased pollution in urban areas. The hectic lifestyle and polluted environment triggers the disease.

7) MARITAL STATUS:

Higher incidence of Tamakaswasa is seen among married people ie) 61%. This is because of selection of age criteria.

8) DIET:

Higher incidence is seen in people with mixed diet ie) 62%. Since this study was conducted near coastal area, consuming matsya(3-6 times) per week is common. And it is observed that symptoms worsen for some patients when they consume shell fish.

9) PRAKRUTI:

Higher incidence of Tamakaswasa is seen in Vatakapha (57%) and Pitta kapha (29%) Prakruti persons. Thus majority of patients were Kaphapradhanaprakrithi prone to Tamakaswasa, which is also a Kaphapradhanaroga.

10) SATMYA:

75% of the patients are having Avarasatmya. Avarasatmya may be birth or acquired. Thus Avarasatmyaia a precipitating factor for Tamakaswasa.It is becauseAvaraSatmyajaBhava persons

develop more hypersensitivity reactions. Aperson with AvaraSatmyajaBhava is more prone to diseases, especially to "AsathmyajaRogas". Asathmya is one among such unique concepts. There are different factors in body contributing towards Asathmya. AsathmyaNidanas are modulated by variety of factors such as Prakruti,Desha,Kala,Vaya,Rtu and Vyadhi. Asathmya may be termed as AtmaSathmyaVirudhaNimittajaVyadhi. Allergens can be included under Asathmya.Hypersensitivity can be included in Asathmya as effect. Allergic Bronchial Asthma is one such manifestation. AsathmyaSevana (eg: Rajo, Dhuma) disturb SahajaSatmyataof doshas. If SahajaSatmyata of the Doshas is lost it lead to the formation of hypersensitivity reactions. The person with "AvaraSatmyajaBhava" is not able to make the foreign substance "Satmeekarana" thus producing allergic manifestations.

11) AHARA SHAKTI:

45% of the patients have Madhyamaaharashakti. 24% of patients have Avaraaharashakti. This is because Jatarangimandya plays an important role in pathology of Tamakaswasa.

12) VYAYAMA SHAKTI:

45% were having Avaravyayamashakti, 40% were having Madhyamavyayamashakti. This indicates that manifestation of Tamakaswasa is a Pranavahasrotodushtivikara and Vyayama is one among the Pranavahasrotodushtihetu.

13) FAMILY HISTORY:

60% of the patients have family history of bronchial asthma and 40% have no such history. This is because extrinsic asthma affects children and young adults and have an inheritance allergic pre- disposition. These patients have AvaraSatmyajaBhaava by birth,

and are more prone to diseases due to AtmaSatmyaViruddhaVyadhi like TamakaSwasa.

VYANJAKA HETUS:

DHUMA:

50% of the TamakaSwasa patients have Dhumaas the triggering factor.Dhumais Ruksha,Sukshmawhich leads to PranavahaSrotoDushti and VataPrakopa that contribute towards Samprapti of TamakaSwasa. Smoke is a lethal cocktail of inhales and toxins. When a person inhales smoke irritating substance settle down in the moist lining of the airways, make them swollen, narrow filled with sticky mucus. Smoke cause lungs to make more mucus than normal triggering asthmatic attack. Normally cilia (tiny-hair like structures) in the airway sweeps away dust and mucus out of the airways. But tobacco smoke damages cilia allowing mucous to accumulate in the airways.

Passive smoking also trigger the symptoms quickly and make asthma worse causing permanent damage to the lungs. Smoke from open fires, wood, burning stove, burning fossil fuel irritates the airways and trigger asthma.

The emittent of respiratory irritants such as sulphur - dioxide also trigger immediate asthma symptoms.

ANILA SEVANA:

Hot and humid air combines with pollutants to create ground -level ozone. This kind of ozone can be a strong asthma trigger. The " ozone pollution" is a major part of "smog" the brownish yellow haze often seen hanging over cities on the horizon. It can irritate the lungs and cause breathing problems. In the earth's lower atmosphere near the ground level (Ground level ozone)is formed. When pollutants emitted by cars, power plants, industrial boilers, refineries, chemical plants and other sources react chemically in the

presence of sunlight. Ground-level ozone is the harmful and the most major air pollutant.

In urban areas on hot sunny days ozone reaches to unhealthy levels and reach high levels during cold weather. In rural areas also ground level ozone is transported by the wind.

People suffering from lung diseases like asthma are at more risk due to ozone pollution.AnilaSevana are Pitta Prakopa causing inflammation in the airways contribute to the process of the disease TamakaSwasa and 36% of patients in the study get triggered by AnilaSevana.

SHEETHA STHANA:

67% of TamakaSwasa patients develop symptoms in Sheethasthana. Tamakaswasa is mostly precipitated or aggravated in winter and SharatRtu due to cold climate and concentration of air-borne pollens. Cold air can have direct irritant effect on inflamed airways. Cold air induce vaso-constriction in bronchial mucosa narrowing the airways . Cold dry air stagnated in ground level create suspended pollutants in the air which trigger response in asthmatics.In ShishiraRtu (Sanchaya) and VasanthaRtu (Prakopa) of Kapha take place which adds to the Nidanaleading to the manifestation of the disease.

MEGHA:

30% of the TamakaSwasa patients get triggered by Megha. In classicsDurdina is considered as Durdina-Mega Chadita Dinaie) Cloudy days which precipitate the attack of TamakaSwasa.

The bio-statistical observations and discussion on Vyanjakahetus which showed significant p value are

RAJO SEVANA:

P value was very highly significant in relation betweenRajosevanaand IgE[ie) p< 0.001]. RajoSevana act as a triggering factor for 85% of patients in

the study hence proving majority patients suffer from extrinsic asthma. Raja is one of the prime factors which vitiate the PranavahaSrotas. Rajo is considered as "Dhuli". Dhuli indicate minute particles which are visible or may not visible to our naked eyes.Raja enters through mouth and nose develops diseases such as Shwasa. Dust particles has various classification according to size,shape and different origin. The dust particle hampers the breathing pattern when it is excessively inhaled. Dust particles add friction to the airflow during breathing.Particles larges than 30-50um in size tend to not to be inhaled through the nose whereas particles size (5-10um) enter through the naso-pharynx conducting the airways.

Dust is the most prevalent aero-allergen. Dust mites are microscopic tiny insects like pests. They feed on dead human skin cells found in dust.The harmful allergen they create comes from fecal pellets and body fragments. They builds up around our homes in carpets,soft toys,bedding,cushion,curtain and furniture.

Humidity is the most important factor determining the growth of dust mites. In low humidity they cannot survive. They cling to particles that are too heavy to remain in the air for long(humid).And in our study most of the patients suffering extrinsic asthma are nearby coastal areas.The coastal regions are with higher humidity and warmer temperature have higher household dust mite levels than inland areas with a drier climate and more extreme temperatures.

According to researchers the lung epithelial cells release free radicals known as RONS(Reactive oxygen and nitrogen species) on their own when exposed directly to dust mite proteins.

When person with asthma inhales an allergen immune cells flood the chest and release molecules known as cytokines that cause inflammation and constriction of the smooth muscle in the lungs. Researchers showthat house dust mite exposure causes inflammation, reactive oxygen species, DNA double- strand breaks ,damage to protein and apoptosis.

House dust mite (HDM) specific IgE are more consistently associated with asthma developments.

Allergic broncho-pulmonary aspergillosis (ABPA) is a condition characterised by an exaggerated response to the immune system to the fungus Aspergillus. Aspergillusspores(molds) are small (2-3 μm) in diameter can penetrate deep into the respiratory system to the alveolar level.The reaction between IgE with Aspergillus antigens results in mast cell degranulation with bronchoconstriction.

Pollens is the most common trigger for many people who have asthma.The pollen when breathed in, body produces histamine and other chemicals."Thunder pollen" are pollen that are smashed by storms that go deeper into lungs. The high levels of pollen are usually seen in air on warm, sunny days.

The flakes of dried skin called dander develop asthma leads to worsen asthma symptoms.These proteins are very small particles that are carried through the air, come in contact with nose or mouth gets directly inhaled into the lungs. Some people are allergic to cockroach droppings and remains called frass which also lead to asthma attacks.

PRAGVATA:

P value was significant in relation betweenPragvataand IgE[ie)

p=0.016].59% of TamakaSwasa patients have Pragvata as a triggering factor. Pragvata is a DushtaVatahaving AbhishyandhiGuna . Patients allergic to pollen get attack during the change of season. Whenever the quality of air differs in different season by change in temperature or humidity precipitates asthmatic attack. High pollen count were associated with easterly and westerly winds blowing in from areas containing rivers and streams. Sudden change in climate release allergens that make asthma worse in people whose asthma is allergy- related.

DISCUSSION ON OBJECTIVE PARAMETERS:

AEC (ABSOLUTE EOSINOPHIL COUNT):

53% of patients have (Grade 0) AEC and 47% have (Grade 1) AEC. The role of IL-5/Eosinophil pathway considered as a consequence of the whole process.Eosinophilic inflammation of the airways characterizes disease severity in subsets of individuals with severe asthma and there is direct relationship between eosinophil count and frequency of asthma exacerbations.Eosinophil differentiation,activation and survival mainly depends upon the effects of IL-5. Function of eosinophils relate to the induction of bronchial wall damage. Studies prove that in types of asthma with high eosinophilia are frequently non-allergic types.

PEFR (PEAK EXPIRATORY FLOW RATE):

39% of the patients have PEFR (Grade 1), 23% have (Grade 2),and 38% have (Grade 3).The primary functional disturbance in asthma is bronchial narrowing or obstruction.This may be due to spasm of the bronchial muscles,swelling of the bronchial walls or obstruction of the lumen by mucus. Bronchial obstruction makes the flow of air in and out of lungs more difficult. In particular, the time

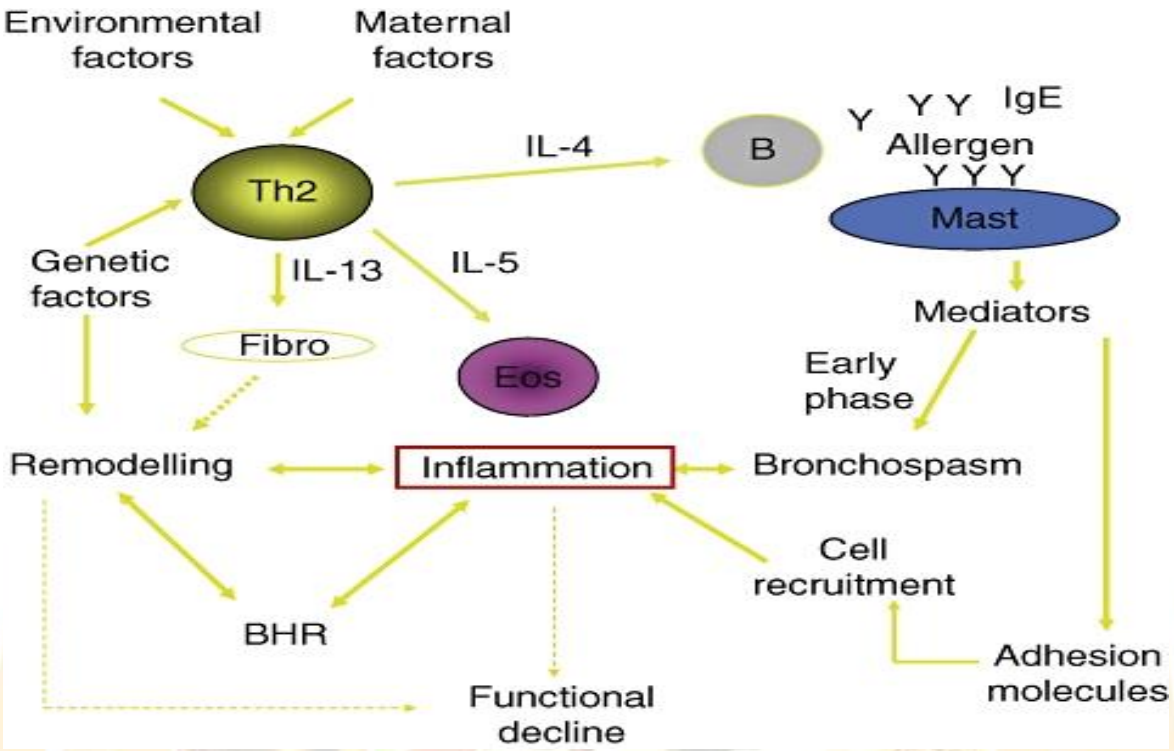
required to expel the inspired air is increased. If expiratory forces are insufficient to expel all of the inspired tidal volume then air will be trapped in the lungs and pulmonary ventilation will be impaired. Peak expiratory flow rate is reduced in asthma and may be simply measured by a specially designed peak flow meter.

IgE (Immunoglobulin E):

The IgE mediated reaction that occurs in Allergic Asthma is a well-known pathogenic model. In the allergic subject, produces an increased amount of IgE towards inhalatory proteins.IgE reaction triggers a complex network of molecular and cellular events which results in mucosal inflammation. The allergic phenotype is characterized by a particular T lymphocyte that in turn favours inflammation process.IgE levels are increased in patients affected by atopic conditions. IgE provide critical link between antigen recognition role of the adaptive immune system and effector functions ofmast cells and basophils at mucosal and cutaneous sites of environmental exposure. The biological role of IgE is complex and related to its ability to influence the functioning of several immune and structural cells involved in the pathogenesis of chronic allergic inflammations. The functions of IgE place in center of pathogenic mechanisms of allergic inflammatory process.Once the allergen is bound to two contagious molecules of IgE mast cell is activated and immediately releases the substances stored in their granules. Among these mediators histamine is the most important via H_1 receptors causing early manifestations so- called "early phase".These manifestations increase mucus secretion, vaso-dilatation, stimulation of nerve ends and broncho spasm. In

addition to the release of mediators, activated mast cells also start to synthesize other inflammatory mediators and cytokines (IL-4 and IL-5) and leukotrienes.The allergic subjects possess a special subset of T lymphocyte known as T helper type 2. Thus T lymphocyte favors inflammatory process and these Th_2 cells secrete high amount of IL-4, IL-5 and IL-13. T- cell immune and inflammatory pathways play an important role in allergy and asthma symptomatly. CD4+ T cells regulate most of the immune responses to foreign proteins by secreting cytokines such as interleukins (IL) and interferons (IFN) categorized into phenotypes. Eg: Th_0,Th_1,Th_2) on the basis of their products. The Th_2class initiates the immediate allergic response by releasing interleukins primarily IL-4 and IL-5 which induce IgE production. The activation of Th_2 and B lymphocyte underlies the process of sensitization to allergens in genetically pre-disposed individuals. Interleukins released by activated Th_2 cells enhance IgE antibodies. These antibodies in turn, attach to other resident cells that possess specific receptors for IgE. The establishment of IgE- bearing cells in the nasal or bronchial mucosa during the sensitization phase activate during subsequent allergen exposure.

Figure No: 34 Showing Pathogenic Mechanism of IgE in Allergic Bronchial Asthma



OBSERVED RESULT IN THE PRESENT STUDY:

In the present study of 100 volunteers suffering from TamakaSwasa the following result were obtained. Thus there is very highly significance of RajoSevana(dust,mites,pollenetc) with objective parameter IgE. There is also significance of Pragvata with IgE due to release of pollens during climate change in the environment.Thus proves that most of the patients affected belong to extrinsic bronchial asthma. Other factors like Dhuma, AnilaSevana, SheethaSthana and Meghaare insignificant with IgE.

THUS ALTERNATE HYPOTHESIS (H_1) IS ACCEPTED AND NULL HYPOTHESIS (H_0) IS REJECTED.ie) There is relation between AsthmayajaNidana and TamakaSwasa.

CONCLUSION

The scientific study starts with identification of the problem, observation of phenomena ,to test the

hypothesis and finally the development of conclusion, that confirms ,rejects or modifies the hypothesis. TamakaSwasa is one of the Pranavaha roto Dusti Janya Vyadhi. Vataand Kapha Doshas are considered to be the chief Doshas involved in the pathogenesis of TamakaSwasa. VataDosha obstructed by KaphaDosha afflicting PranavahaSrotas producing TamakaSwasa. Food and activities in general which is compatible is called Satmya. Food and activity made compatible by habituation is called as Oka Satmya. Incompatible food/activity is called as Asathmya. Incompatibility specific to an individual is called as AtmaSatmya or Idiosyncrasy or Allergy. AtmaSatmya is " Pratipurusha Satmya" which is specific to the person.VyanjakaHetus are stimulating, precipitating or aggravating cause. It is due to the VyanjakaHetu that aggravation of symptoms occur in an already generated disease or these cause the precipitation of the Samprapti of disease. In the present study from the list of AsatmyajaNidanas the Vyanjaka Hetus are best related to the pre-disposing factor of Allergic Bronchial Asthma. AsathmyajaNidanas can be found out by history taking. The effect of AsathmyajaNidana could beAshukaarior Kaalantara. Among them "RajoSevana"is incidently predominant with elevated IgE levels in majority of the patients with ($p<0.001$). Hence it can be concluded that the higher incidence of TamakaSwasa was seen in patients who are exposed to " Raja"(ie) House dust, mite, pollen, mould, fungi, animal dander etc) and hence most of the patients are affected by the Extrinsic Asthma (atopic asthma).

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