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Contemporary Study On Ama

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Ama is responsible for the production of various diseases. In the same way free-radicals & auto-antibodies are also found to be the root cause of many disorders. The role of these factors is all ready established fact in certain auto-immune disorders. Ex: Rheumatoid arthritis. Here is the compilation of both the aspects.

Keywords: Ama, auto-antibodies, auto-immunity, free-radicals, Amavata.

Introduction:

Ama is considered to be one of the important aspects for the development of disease process in the body. It plays a major role in the particular disorders like Amavata, Pandu etc. It is formed due to improperly metabolized bye product of food particles either at Jatharagni level (macro level), Bhoothagni level (micro level) or at Dhatwagni level. So Ama can give rise to disease. It spreads in the body rapidly. Its action is like that of toxic substance. That why it has been described as Amavisha^{1,2}. Apakwa anna rasa is produced in the Amashaya (Jatharagni level). This type of Anna rasa get absorbed inside the body & produce various Annavaha srotogata vikara's. The Ama which produced at Dhatwagni level is absorbed & spread to other dhatu's, ex-Madhumeha.

As Ama is foreign substance to the human system, a strong immune response takes place. At the same time the antibodies will be formed by the immunological cells of the body. Then antigen (Ama) – antibody interaction settles in. At this level failure of power of differentiating between the body's own material (self) & foreign material (non-self) occurred. Thus auto-antibodies (Ama) are freely circulated in the patient's serum. It can affect or destroy any part of the body. When it is affecting the heart, the Hrudroga (Rheumatic Heart Disease) is seen, when it is affecting the joints, Amavata (Rheumatoid Arthritis) is seen. Thus we can correlate the Amaja vikara's with that of Auto-immune disorders.

In contemporary science also, formation of free radicals in the body brings the disease. Here also we can compare the free radicals with that of Ama. So any substance which is remained stable without any Paaka or remained as residual or as bye product without attaining any finality in the metabolism can be considered as Ama such as Pyruvic acid, lactic acid, ketones etc. Let us consider these aspects in detail.

Etymology: Ama is aamyate ishat paschayate paak:/

Which means not complete digestion of the food ie, undigested state of the food.

Auto-immunity is having two words in it; auto means self & immunity means the body's ability to resist infection⁶.

Definition: *Ushmano alpabalatwena dhaatumaadhyam apaachitam*/

Dushtamaamashayagatam rasamaamam prachakshate //

Ama means undigested state ie,paakarahita,apakwa¹. After the formation of Ama in the body, it enters into circulation as a fine particle (free radicals) & wherever it settles in the body parts, it gives so many complications². The body unable to identify its own products (self from non-self) it is then go on attacking the healthy tissues in the body, then it is called as auto-immunity.

Nidana/Etiology: The important etiological factors include *apathya ahara sevana*, *viruddhahara sevana*, *vega udeeran*, *manovyathaa*, *ajeerna & agnimandya avasthaa* causes Ama related disorders².

The causative factors in general for auto-immune diseases are genetic factors (psoriasis, rheumatoid arthritis etc), hormonal factors, microbiological factors (SLE, phempigus vulgaris, infective skin manifestations etc) & environmental factors include smoke, dust^{6,7.8} etc

Samprapti/Pathogenesis: after indulging above said nidanas, agni gets vitiated & due to agnimandya apakwa anna rasa (Ama rasa) is formed. This Ama rasa obstructs the srotas (srotoavarodha). Thereafter dosha-doos hya sammorchana begins. Formed Ama dosha is called Ama visha(toxaemia) which circulates all over the body^{1,3,5}.

After both types of immune responses (natural & adaptive) to the causative agent, formation of excessive production of auto-antibodies takes place in the system. These enters into the circulation & immune complexes are deposited in organs/tissue. Then inflammation & damage to the bodily tissue begins in. With advancement of time it affects & alters the function directly. The factors affecting pathogenicity includes size of the antigen, smaller one (free radicals, virus) & bigger one includes parasites; nature of the antigen (whether it is plant or animal origin); class of antibodies (including sub-classes) & compliment fixing ability^{7,8}.

Diagnostic criteria: on the presence of Ama in the body, we can correlate the parameters to understand the changes in stool & urine for certain extent ie, urine shows slimy, foul smelling, high specific gravity & it is of varying colours; stool is also having shades of various colours, foul smelling & sinks in water.^{3,4,5}

In contemporary, based on certain laboratory investigations which include detection of specific auto-antibodies, Agglutination reaction tests, Indirect Immunofluorescence test & ELISA test used to detect various auto-immune disorders^{7,8}. Ex- Amavata (RA), infective skin manifestations (Twachagata vikaras) & IDDM

Treatment modality: The treatment modality in Ayurveda includes nidana parivarjana, shodhana chikitsa & shaman chikitsa.

Brief summarization on Similarities of Amaja vikara's with Auto-immune disorders

Sl.no	Ama	Auto-immunity

1.	Etymology	Amyate ishat paschayate paakaha Ama = paaka rahita, apakwam, ishath paaka	Auto = self; Immunity = the body's ability to resist infection.
2	Definition	If rasadhatu is inadequately digested due to weakness of agni (Jatharagni) & remains accumulated in the Amashaya ,it is known as Ama	When the immune system fails to recognize the 'self' from 'non-self' & goes on attacking the body's own tissues, then it is called as Auto-immunity
3	Nidana / Etiology	Apathya ahara sevana, viruddhara sevana, vega udeerana, mano vyatha; any etiologies leading to agnimandyta	Genetic factors Hormonal factors Microbiological factors Environmental factors
		Any aetiologies leading to Agnimandyata Formation of apak wa ahara rasa Formation of ama rasa	Immune responses
		Dos ha-dooshya sammurchana Sama dos ha Sroto d ushti— sroto sanga	Natural Adaptive 1. Produce Ig Antigenantibody by B- lympocytes interactions 2. cellular immunity mediated by T- lymphocytes Formation of auto-antibodies (normal physiological process)
4	Pathogenesis	Sroto a varodha (mala vr udd hi)	Excessive production of auto-antibodies

		Dosha – tridosha (vata pradhana) Dooshya's – Rasadhi dhatu's & mala's Sthana samshraya – Based on srotodushti Saama avastha = Sthana samshraya Saama roga = vyaktavastha	Enter into the circulation circulating immune complexes are formed Deposition of immune complexes on the organ/ tissue
			Inflammation & damage to the body tissue Affect function directly
5	Ama swaroopa	Colour – Aneka varna Structure – Dravatwa Odour – Durgandha Guna's – Dravatwa Guru Snigdha Picchila Tantumaya Avipakwa Asamyuktha Dourgandhya Abhishyanda Sroto avarodha	Factors affecting the pathoge necity 1.Size of the antigen 2.Nature of the antigen 3.Class of antibodies 4.Compliment fixing ability
		Formed at the various level of Agni – Jatharagni (Sthoola ama rasa)	

		Bhoothagni (Free-radicals) Dhatwagni (Auto-immune disorders) (These two are Sookshma ama rasa)	
6	Diagnostic criteria	Urine - slimy, foul smelling, varying colours, high specific gravity Stools - shades of various colours, foul smelling, sinks in water	 Presence of specific auto-antibodies (Useful markers of disease activity) Based on Agglutination reaction test (Used to detect rheumatoid factors) Indirect immuno-fluorescence test. (Used to detect organ specific & tissue specific antibodies) ELISA (Used to detect the wide range of proteins & auto- antibodies)
7	Exa mples	Amavata Pandu	Rheumatoid arthritis Insulin dependent diabetes Thyroiditis Multiple sclerosis
8	Treatment modality	 Nidana parivarjana Tadarhtakari chikitsa Apatarpana 	Prevention of Auto-immune disorders 1. T-cells & B-cells are eliminated if they are self reactive (because T & B – cells randomly recombine the genes for their receptors that will react with self antigen) 2. Preventing the self reactive T-cells A. Lack of the co-stimulatory molecules required for T-cell activation B. Lack of antigen presentation C. A negative feedback system prevents overstimulation of an immune response D. Response to self antigen can also be regulated

	by a population of T-cells producing suppressive
	cytokines

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