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KRIYAKALA IN RELATION WITH INFLAMMATION

Dr. Vibhooti Chandrakar*

Associate Professor, Department of Samhita Sanskrit and Siddhanta, Patanjali Bhartiya Ayurvigyan Evam Anusandhan Sanstha, Haridwar, Uttarakhand.

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***Corresponding Author**
Dr. Vibhooti Chandrakar
Associate Professor,
Department of Samhita Sanskrit and Siddhanta,
Patanjali Bhartiya Ayurvigyan Evam Anusandhan Sanstha,
Haridwar, Uttarakhand.

ABSTRACT

Concept of development of the disease in current science shares similar school of thoughts as scientifically elaborated in *Kriyakala*. *Kriyakala* means the recognition of the *Avastha* or the stage of the process of disease and the resort to appropriate measures to correct the same. Inflammation is the first response of the body in many diseases. The knowledge of the stages of inflammation by *Kriyakala* helps in the recognition of the disease process even in its very early inceptive stage so that equally early steps may be taken to arrest the further development. In this article genuine effort has been made to show the relation between stages of inflammation and *Kriyakala*.

KEYWORDS: Kriyakala, Inflammation, Sanchaya, Prakopa, Prasara, Sthanasamshraya, Vyaktavastha.

INTRODUCTION

Concept of development of the disease in current science shares similar school of thoughts as scientifically elaborated in *Kriyakala*. Inflammation is the first response of the body in many diseases. The knowledge of the stages of inflammation by *Kriyakala* helps in the recognition of the disease process even in its very early inceptive stage so that equally early steps may be taken to arrest the further development.

INFLAMMATION

"The local response of living mammalian tissues to injury due to any agent" It is a body defense reaction in order to eliminate or limit the spread of injurious agent as well as to remove the consequent necrosed cells and tissues.

Kriyakala: The term *Kriyakala* means the time of action.

Kala, in this context, signifies the *Avastha* or stage of the process of disease.

Kriya, refers to the resort to measures- *Aushadha*, *Ahara* and *Charya*- with a view to eliminate and correct the *Doshik* disturbances.

Kriyakala, therefore means the recognition of the *Avastha* or the stage of the process of disease and the resort to appropriate measures to correct the same.

Types: *Kriyakala* is of four types- *Sanchayawastha, Prakopawastha, Prasarawastha, Sthanasamshrayawastha, Vyaktawastha and Bhedawastha*.[1]

Sanchayawastha and incubation period

Sanchayavastha is the first stage of *Kriyakala*, in this stage the *Doshas* will get accumulated in its own sites.[2]

Similarly during the incubation period of inflammation morbid matter, poisons, microorganisms and other excitants of inflammation gather and concentrate in certain parts and organs of the body. Hence, it can be considered as *Sanchayavastha*.

***Prakopa-Prasarawastha* and vascular and cellular events**

Prakopa: It is the second stage of *Kriyakala*, in this stage the deposited *Doshas* further begin to excite the system and hence cause aggravation.[3] Dalhana has given simile that, when ghee yet start boiling it starts to melt and move inside the vessel, it can taken as Prakopa.

Prasaravastha: It is the third stage of *Kriyakala*, in this stage excessive aggravated *Doshas* will start moving from their own site.

When more heat is applied to ghee it starts come out from vessel .It can be taken as *Prasarawastha*.

Also, Overflowing of Doshas from their respective seats to other places takes place like fermented materials comes out after keeping mixture of yeast, water and flour in a vessel for overnight.[4]

Vascular events in inflammation: Alteration in the microvasculature (arterioles, capillaries and venules) is the earliest response to tissue injury. These alterations include- heamodynamic changes and changes in vascular permeability.[5]

a) Haemodynamic changes: irrespective of the type of injury immediate vascular response are transient vasoconstriction of arterioles. Next follows persistent progressive vasodilatation. Vasodilatation results in increased blood volume in microvasculature bed of the area. Progressive vasodilatation, in turn may elevate the local hydrostatic pressure resulting in transudation of fluid into the extracellular space.[6]

Slowing or stasis of microcirculation follows which causes increased concentration of red cells, and thus raised blood viscosity resulting leucocytic margination or peripheral orientation of leucocyte along the vascular endothelium. The leucocytes stick to the vascular endothelium briefly, and then move and migrate through the gap between the endothelial cells into the extra vascular space.[7]

b) Altered vascular permeability: In and around the inflamed tissue, there is accumulation of oedema fluid in the interstitial compartment which comes from blood plasma by its escape through the endothelial wall of peripheral vascular bed. In the initial stage, the escape of fluid is due to vasodilatation and consequent elevation in hydrostatic pressure. This is trasudate in nature. But subsequently, the characteristic inflammatory oedema, exudates, appears by increased vascular permeability of microcirculation.[8]

Mechanism of increased vascular permeability[9]

i) Contraction of endothelial cells. The endothelial cell develops temporary gaps between them due to their contraction resulting in vascular leakiness. It is mediated by the release of histamine, bradykinin and other chemical mediators.

ii) Retraction of endothelial cells- there is structural reorganization of the cytoskeleton of endothelial cells that causes reversible retraction at the intercellular junctions. This changes too effects venules and is mediated by cytokines such as interleukin1 (IL-1) and tumor necrosis factor.

iii) Direct injury to endothelial cells- causes cell necrosis and appearance of physical gaps at the site of detached endothelial cells. Process of thrombosis is initiated at the site of damaged endothelial cells. The change affects all levels of microvasculature (venules, capillaries and arterioles).

iv) Endothelial injury mediated by leucocytes- adherence of leucocytes to the endothelium at the site of inflammation may result in activation of leucocytes.

The activated leucocytes release proteolytic enzymes and toxic oxygen species which may cause endothelial injury and increased vascular leakiness.

v) Leakiness in neovascularization. In addition the newly formed capillaries under the influence of vascular endothelial growth factor (VEGF) during the process of repair and in tumors are excessively leaky.

Cellular events in inflammation[10]

The cellular phase of inflammation consist of 2 processes

a) Exudation of leucocytes- the escape of leucocytes from the lumen of microvasculature to the interstitial tissue is the most important features of inflammatory response.. In acute inflammation polymorphonuclear neutrophils (PNMs) comprise the first line of body defense, followed later by monocytes and macrophages.

b) Phagocytosis- it is the process of engulfment of solid particulate material by the cells. The cells performing this function re called phagocytosis.

During *Prakopawastha* there will be aggravation of *Doshas* and during *Prasarawatha* there will be movement of *Doshas* from its own place to other.

Similarly in inflammation during vascular events, due to vasodilatation there will be increased hydrostatic pressure resulting in transudation of fluid into the extracellular space.

During cellular events there will be escape of leucocytes from the lumen of microvasculature to the interstitial tissue (exudation of leucocytes) Also, During Prasarawastha, Doshas spread sometimes alone or in various combination in two, all or with blood.[11]

In inflammation also blood is the medium for the spread or dissemination of morbific factors. So, vascular and cellular events can be considered as Prakopa and Prasarawastha. Doshas having been eliminated in the stage of accumulation do not attain successive stages. They become stronger as they proceed further.[12]

If treatment is given during *Prasaravstha* then *Doshapaka* will takes place and progression to further *Kriyakala* can be arrested. If treatment is not given then *Dhatupaka* will taken place and it will reaches into *Sthanasamshrayawastha*. Similarly in inflammation, if phagocytes are successful in killing, then bacteria etc. will be killed and digested, if it fails in killing then living bacteria liberate to cause further damage.

Sthanasamshrayawastha: *Doshas* thus aggravated and spread to different parts of the body and then produce respective disorders. When localized in *Udara*- they produce *Gulma, Vidradhi, Udara, Agnisanga, Anaha, Visuchika.*

Basti- Prameha, Ashmari, Mutraghata, Mutradosha etc.[13]
In inflammation, if the agent which is responsible for inflammation goes to
Appendix- appendicitis
Stomach- gastritis
Colon- colitis
Gallbladder- cholecystitis
Liver- hepatitis will occur.

In *Ayurveda*, *Acharyas* have explained very unique way of treatment i.e. *Pachana Chikitsa*. It is considered as complication in some conditions i.e. *Bhagna* and treatment in some conditions i.e. *Vranashotha*.

Immediately after *Sthanasamshra*, once physician will come to know that the condition is not reversible then he can go for *Pachana Chikitsa*.

For this, the drug should be collected and combined with *Dadhi, Takra, Sura, Shukta, Dhanyamla* and adding ghee and salt *Utkarika* should be prepared with them. It should be applied hot on the part and covered with leaves of *Eranda*. When swelling tends to suppurate wholesome diet should also be given. Then physician should remove the pus.

This is not complication. In this way physician can decrease the time period of *Kriyakala*.

Vyaktavashta
During this *Avastha* there will be clear manifestation of symptoms in disease.
E.g. *Shopha, Arbuda, Granthi* etc.[14]

In **inflammation** also specific symptoms are seen depending on different diseases.

Bhedavastha
It is the sixth period of treatment when they burst and become ulcers and in case of *Jwara, Atisara* etc. attain chronicity. If not treated properly they become incurable.[15]

In **inflammation** also, if treatment is not given in this stage leads to chronicity.

E.g. Gastritis- Left untreated; it may lead to stomach ulcers and stomach bleeding. Some forms of chronic gastritis may increase risk of stomach cancer, especially if the patient has extensive thinning of the stomach lining and changes in the lining's cells.

Appendicitis- if appendicitis bursts, it releases the infection into abdomen, which can lead to a condition called peritonitis. This is when the inner lining of abdomen (the peritoneum) becomes inflamed. This lining is very sensitive to infection.

CONCLUSION

• *Acharya's* perception about the disease process was equally efficient as that of current science.

• Acquiring this knowledge will help to combat the disease in its primitive stages which in turn will result in lowering the mortality rate.

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