ORIGINAL RESEARCH PAPER

Ayurveda

CRITICAL ANALYSIS OF AYURVEDIC PERSPECTIVE OF ULCERATIVE COLITIS IN RELATION WITH VYADHIKSHAMATWA AND AGNI

KEY WORDS: Ulcerative colitis, grahani, Vyadhikshamatwa

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ABSTRACT

Ulcerative colitis is an idiopathic, chronic inflammatory disorder of the colonic mucosa that commonly involves the rectum and may extend in a proximal and continuous fashion to involve other parts of the colon. The disease typically affects individuals in the second and third decade of life with hallmark clinical symptoms of bloody diarrhoea and rectal urgency with tenesmus. The clinical course is marked by exacerbations and remissions, which may occur spontaneously or in response to treatment changes. However, even with medical therapy, up to 15% of patients will require surgery to treat UC or disease complications of dysplasia. In Ayurveda the pathogenesis and clinical manifestations of chronic UC can be correlated with disease entities such as ***atisara*** and ***grahani*** *where* agnidusti plays a major role in the manifestation of disease. Since agni is inherently related with vyadhikshamatwa, the altered immune response in UC can be correlated with agnivaishamya. The persistent agnivaishamya in UC leads to chronic inflammatory state of colon. So ayurvedic management should focus in mere correction of kayaagni.

INTRODUCTION

Dr Samuel Wilks is credited with being the first to describe UC in 1859 when he wrote on idiopathic colitis and recognized it as distinct from the then more common bacillary dysentery. It is an idiopathic, chronic inflammatory disorder of the colonic mucosa that commonly involves the rectum and may extend in a proximal and continuous fashion to involve other parts of the colon. The disease typically affects individuals in the second and third decade of life with hallmark clinical symptoms of bloody diarrhoea and rectal urgency with tenesmus. The clinical course is marked by exacerbations and remissions, which may occur spontaneously or in response to treatment changes. However, even with medical therapy, up to 15% of patients will require surgery to treat UC or disease complications of dysplasia. The morbidity associated with ulcerative colitis patients leads to poor quality of life associated with enormous mental health problems such as depression. So the aim of treatment is to reduce disease burden through appropriate selection of medications based on severity of disease and thereby induce rapid remission. Currently, the impaired mucosal healing is the challenge faced by the modern physicians while dealing ulcerative colitis patients which further leads to persistence of chronic inflammatory state and thereby causes colonic dysplasia.

Beeja dusti is inevitable in the pathogenesis of autoimmune diseases, and it will be further triggered by ahara- vihara janya dosha dusti. In *Astanga hrdaya, it is mentioned that vishama-ajirna-seeta-sankeerna bhojana and ruksa- guru anna leads to samana and apana vayu vaigunya respectively*. In the present scenario; due to changing dietary patterns and life styles the above mentioned nidanas plays a major role in the manifestation of autoimmune disease like ulcerative colitis. The present management strategies of UC fails to induce mucosal healing which in turn causes future relapses. So many patients approaches ayurvedic system of medicine in search of alternate therapeutic options to maintain clinical remission and to reduce steroid dependent states.

INCIDENCE AND PREVALENCE

The overall estimated IBD population in India in 2010 came out to be 1.4 million, which was the second highest number after the USA (with 1.64 million). Data obtained from two studies conducted in Northern states of India is as follows. A study of 21,971 persons in Haryana in 1984 recorded a prevalence rate of 42.8 per 100,000 and the second study was conducted in Ludhiana, Punjab where a population of 51,910 was screened through a cluster random sampling method and 23 patients were diagnosed with definite UC. The prevalence of UC was 44.3 per 100,000 and the crude incidence rate calculated after a second visit one year later was 6.02 per 100,000.

ETIOLOGY AND PATHOGENESIS

The etiology of UC is currently unknown but is likely multifactorial. The currently held paradigm involves a complex interaction of three elements.

1. Genetic susceptibility
2. Host immunity
3. Environmental factors

- ***Dysregulation of the enteric immune response*** in genetically predisposed persons leads to the development of acute and chronic inflammation and the pathologic feature of mucosal damage.
- The specific inciting antigens for the inflammatory process have yet to be identified, but several sources have been suggested including:
    1. Pathogenic and commensal microorganisms
    2. Metabolic by products of these agents
    3. Normal epithelial structures
- *Under physiologic conditions, homeostasis normally exists between the commensal microbiota, epithelial cells that line the interior of the intestines and immune cells within the tissues.*
- Specific environmental (Eg: smoking, antibiotics, entero-pathogens) and genetic factors in a susceptible host, cumulatively and interactively disrupt homeostasis and culminates in a ***chronic state of dysregulated inflammation***.
- Currently considered as inappropriate immune response to the endogenous commensal microbiota within the intestines, with or without some component of autoimmunity.

DEFECTIVE IMMUNE REGULATION IN IBD

- ***Mucosal immune system is normally unreactive to luminal contents due to oral (mucosal) tolerance.***
- Oral tolerance may be responsible for the lack of immune responsiveness to dietary antigens and the commensal microbiota in the intestinal lumen.
- In both UC and CD, an inflammatory pathway thus likely emerges from the genetic predisposition that is associated with inappropriate immune and epithelial

sensing and reactivity to commensal bacteria that secrete inflammatory mediators together with inadequate regulatory pathways that lead to activated CD4+ and CD8+ T cells within the epithelium and lamina propria that altogether secrete excessive quantities of inflammatory cytokines relative to anti-inflammatory cytokines.

INFLAMMATORY CASCADE IN IBD

- In IBD, ***the abnormal innate immune sensing of bacteria by parenchymal cells (IEC) and hematopoietic cells*** is once initiated and later the immune inflammatory response is perpetuated by T cell activation.
- Inflammatory cytokines such as IL – 1, IL -6 and TNF have diverse effects on tissues. They promote fibrogenesis, collagen production, activation of tissue metalloproteinases and the production of other inflammatory mediators.
- *These cytokines are normally produced in response to infection but are usually turned off or inhibited at the appropriate time to limit tissue damage.* In IBD their activity is not regulated, resulting in an ***imbalance between the pro-inflammatory and anti-inflammatory mediators***.

CONCEPT OF AGNI vs AUTOIMMUNITY

- Inflammation arising in response to immunological insults that cannot be resolved in days / weeks gives rise to chronic inflammation. The chronic inflammation is the hallmark of autoimmune diseases which may lead to permanent organ damage or impaired vascular function.
- Acharya charaka explains about relation of agni with bala in the context of grahani chikitsa.
- The evolution of autoimmune responses can be explained on the basis of agni; because *impaired agni results in formation of ojas which has viruddha swabhava.*
- In relation with ulcerative colitis, *rakta dushti has a pivotal role in initiating agni vaishamya that is why it shows viruddha swabhava (soumya bhava of bala is inherited with tikshna bhava).*
- In general pathogenesis of a disease, the dosha dushti resulting from aahara vihara nidanas has a direct influence on agni and thereby creating agni dushti at koshta level which may progress to dhatu level in later stages. If the agni is influenced by kapha dosha then it results in aama (mala sanchayam).
- With respect to ulcerative colitis, aama at genetic level should also be considered along with koshtagata aama because koshtagata aama can be easily corrected but genetic level aama should not be corrected completely.
- Also the persistence of vishamagni in autoimmune diseases shows the involvement of aama at genetic level. This agni vaishamya in turn causes dearrangement in sareera bala.
- In case of ulcerative colitis, mala sanchaya occurs in pureeshavaha srotas along with impaired bala results in the manifestation of disease as impaired gut immunity and resultant inflammatory response in the colon.
- Here the *koshtagata aama acts as a triggering event for autoimmune mechanism* and thereby leads to chronic inflammatory state by the activation of proinflammatory mediators and cytokines which can be considered as dhatwagni mandya.
- Hence we can conclude that *normalcy of agni is important for proper functioning of immune system and thereby maintains homeostasis of the body.*

PATHOGENESIS OF CHRONIC ULCERATIVE COLITIS IN THE LIGHT OF VYADHIKSHAMATWA AND AGNI

While considering the pathogenesis of ulcerative colitis; agnimandya acts as triggering factor for the initial manifestation of disease. It can be clearly stated that aahara-vihaarajanya agni vaishamya initiates aama at the kosta level which became gradually progressed to dhatu level and finally leads to ojovyapat. The concept of vyadhikshamatwa is related with kapha, bala, agni and ojas. Acharya charaka explained the term bala as prakrta sleshma, which was inherently related with immunity. ***With respect to ulcerative colitis***, impaired immune tolerance leads to disturbed gut homeostasis and results in gut inflammation. Above mentioned facts clearly shows that, *involvement of rakta dushti and alpa satwa serve as triggering factors in the initiation of autoimmune response*. In case of ulcerative colitis, this event is specifically happens in colon.

ASSESSMENT OF KOSHTA - AGNI - AAMA IN ULCERATIVE COLITIS

- In UC, vishamagni is observed in majority of patients. This can be precisely explained as follows; i.e due to the impairment of agni the bala of patient will be altered because bala denotes the proper functioning of agni. Here the term bala can be interpreted as vyadhikshamatwam i.e immunity.
- In case of autoimmune disorders like ulcerative colitis due to the involvement of rakta dushi there is severe agnivaishamya which in turn causes bala vaishamya *i.e there is admixture of soumya nature of bala with tikshna guna.* This can be further explained on the basis that rakta dushti is considered as kapha pitta dushti because rakta resembles kapha with respect to structure and in functional aspect it resembles pitta. Therefore in rakta dushti agni is influenced by kapha and pitta and there will be an admixture of mandagni and tikshna agni i.e vishamagni. This altered agni is sometimes reflected in the form of impaired immune responses. In ulcerative colitis, this response is limited only to gut.
- ***While analysing kostha in ulcerative colitis patients***; mrdu and krura koshta can be observed. The *vishama swabhava of agni* is reflected in the koshta as mrdu and krura koshta. This can be explained as the *qualitative increase in saratwa, dravatwa and snigdha gunas* will result in mrdu koshta which will be reflected as rakta atisara etc. Increase in laghu, ruksa and kharatwa guna will result in krura koshta and the manifestations will be baddha mala, aanaha and hrt daha etc.
- The formation of ama is greatly influenced by the genetically formed agnibhava. For example, a person practicing apathya in his daily routine, having strong prakruta agnibhava may not develop a pathological state, but person with weak agnibhava may go to a pathological state very fastly even with mild apathya.
- *Persons with morbid genetic agnibhava in prakruti* are very susceptible to genetic and autoimmune diseases. In these individuals, even weak etiological factors will initiates the pathogenesis.

APPLICATION OF BASIC PRINCIPLES OF AYURVEDA IN THE MANAGEMENT OF UC

The relation between agni and vyadhikshamatva is well explained by Acharya Charaka in grahani chikitsa. This concept can be further elucidated into the ayurvedic pathogenesis of auto immune disorders because malfunctioning of immune system is the net result of underlying agnidusti. In jwara chikitsa acharya specifically mentions about the importance of bala samrakshana in treatment aspect. As we know that, in all diseases, the role of treatment is only to enable the body's immune system to fight against foreign bodies. So this principle is also applicable in the management of autoimmune diseases like ulcerative colitis. ***Therefore in nut shell, the prime aim of treatment in autoimmune disorders is to correct agnidushti and also to prevent the nidanas of agnivaishamya because the aama resulting from impaired agni itself acts as a triggering factor for initiation of autoimmune events and further enhances disease progression.***

In order to address the autoimmune mechanism and chronic inflammatory states of colon, treatment paradigm of chronic UC must include :

- Correction of ***ojo vaishamya resulting from prolonged agnidusti.***

- ***Selection of drugs includes those which possess deepana, pachana and grahi properties.***
- The probable mechanism of action of these properties at the level of immune system can be explained as follows. i.e *The grahi property of drugs will help to control excess immune response and the pachana property helps to prevent agnimandya and thus further enhances functions of immune system.*

CONCLUSION

UC is a chronic inflammatory condition where medications are used to induce remission and maintain a steroid free remission. Up to 15% patients may require colectomy due to inability to control the disease. In Ayurveda the pathogenesis and clinical manifestations of chronic UC can be correlated with disease entities such as ***atisara*** and ***grahani*** *where* agnidusti plays a major role in the manifestation of disease. Since agni is inherently related with vyadhikshamatwa, the altered immune response in UC can be correlated with agnivaishamya. The persistent agnivaishamya in UC leads to chronic inflammatory state of colon. So ayurvedic management should focus in mere correction of kayaagni.

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