WORLD JOURNAL OF PHARMACEUTICAL RESEARCH
Coden USA: WJPRAP Impact Factor 8.63
Volume 15, Issue 13, 1783-1819. Review Article ISSN 2277–7105

A CRITICAL REVIEW ON ANKYLOSING SPONDYLITIS IN THE LIGHT OF TRIKA-KATI-PRISHTA GRAHA – LITERARY RESEARCH BASED CONCEPTUAL STUDY

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Article Received on 05 June 2026,
Article Revised on 25 June 2026,
Article Published on 01 July 2026,

https://doi.org/10.5281/zenodo.21155483

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How to cite this Article: Dr. Chandana K.*[1], Dr. A. S. Prashanth[2], Dr. MD. Yaseen Hullur[3]. (2026). A Critical Review on Ankylosing Spondylitis In The Light Of Trika-Kati-Prishta Graha – Literary Research Based Conceptual Study. World Journal of Pharmaceutical Research, 15(13), 1783-1819.

ABSTRACT

Ankylosing Spondylitis (AS) is a chronic inflammatory autoimmune disorder predominantly affecting the axial skeleton, characterized by progressive pain, stiffness, and structural deformity. Despite advancements in pharmacotherapy, current management remains largely symptomatic with significant long-term limitations. Ayurveda does not describe AS as a distinct clinical entity; however, its symptomatology closely resembles Trika-Kati-Prishta Graha, a subtype of Vata Vyadhi associated with Ama. This review critically evaluates the pathogenesis, clinical features, and progression of AS through Ayurvedic principles, emphasizing the roles of Mandagni, Ama formation, and Tridoshic imbalance. Classical references are systematically analyzed and correlated with contemporary biomedical understanding. The study highlights that AS can be interpreted as a Sama Vata disorder involving Asthi and Majja Dhatu. This integrative approach provides a deeper understanding of disease mechanisms and suggests potential for holistic and root-cause-based management strategies in Ayurveda.

KEYWORD: sankylosing Spondylitis, Trika graha, Kati Graha, Prishta Graha, Samavataja Vyadhi, Ayurveda.

INTRODUCTION

Ambulation is a fundamental function in human life, largely dependent on the flexibility and integrity of the axial skeleton. Ankylosing Spondylitis (AS) is a chronic inflammatory autoimmune disorder primarily affecting the sacroiliac joints and spine, leading to persistent pain, stiffness, progressive restriction of movements, and postural deformities. With increasing sedentary lifestyle patterns, the condition often remains underdiagnosed until symptoms significantly impair daily activities and quality of life.
AS typically manifests in early adulthood and is more prevalent in males. It involves complex interactions between genetic and environmental factors, with HLA-B27 and immune-mediated inflammatory pathways playing a crucial role. Progressive inflammation may lead to fibrosis, calcification, and eventual spinal fusion, resulting in the classical “bamboo spine” appearance.[1]

Although Ankylosing Spondylitis is not described as a distinct entity in Ayurveda, its clinical features closely resemble conditions such as Katigraha, Trika–Kati–Prishta Graha, and Samavata, broadly classified under Vataja vikara. Pain (shula) is the principal manifestation of vitiated Vata, while stiffness (stambha) and heaviness (gaurava) indicate the involvement of Kapha. Inflammatory features suggest the association of Pitta, reflecting a tridoshic involvement with predominance of Vata–Kapha.

From an Ayurvedic perspective, AS can be understood as an Amapradhana vyadhi, originating from agnimandya. Impaired digestive fire leads to the formation of ama, which, in association with vitiated Vata, localizes in sandhi, asthi, and majja dhatu, particularly in the trika region. This results in clinical manifestations such as pain, stiffness, and restricted spinal movements, resembling Trika - Kati–Prishta Graha. As the disease progresses, the pathology extends along the axial skeleton due to the chala guna of Vata, producing a characteristic pattern of involvement.

Classical Ayurvedic principles emphasize that when ama is present, its elimination and digestion form the cornerstone of treatment.

As stated,

Sariranugate same rase langhana pachanam |[2]

Amashayagate suddhir vamanair virecanaih ||

indicating that depana, pachana, and appropriate shodana therapies are essential depending on the stage and location of pathology. Thus, understanding Ankylosing Spondylitis through the lens of Ayurveda provides a comprehensive framework that integrates dosha, dushya, agni, and ama in its pathogenesis. This forms the basis for adopting a treatment approach aimed at ama pachana, srotoshodhana, and vata shamana, thereby addressing both the root cause and clinical manifestations of the disease.

ETYMOLOGY

The term Sandhi Graha is composed of two words - Sandhi and Graha. Sandhi refers to the junction or union of body structures, particularly the articulations of bones (asthi samyoga sthana). Graha denotes seizing, holding, or obstruction. In the clinical context, it signifies restriction or impairment of joint movement. Thus, Sandhi Graha can be understood as a condition characterized by stiffness and restricted mobility of joints.

Although Sandhi Graha is not described as an independent disease in classical Ayurvedic texts, conditions such as Kati Graha, Trika Graha, and Prishta Graha are mentioned under Vatavyadhi, where restriction of movement is a predominant feature.[3,4]

The term Trika – Kati - Prishta Graha specifically refers to the involvement of the sacral (Trika), lumbar (Kati), and dorsal (Prista) regions. Trika corresponds to the sacral region at the base of the vertebral column, Kati denotes the lumbar region, and Prishta refers to the back. Graha indicates restricted movement of these regions due to pathological involvement.

Nidana of Trika - Kati - Prishta Graha

The causative factors of Trika - Kati - Prishta Graha are not described independently in classical texts; hence, the general nidana of Vatavyadhi is considered. These are broadly classified into Aharaja, Viharaja, Manasika, and Anya nidana.[5,6,7,8,9,10,11]

Aharaja nidana includes consumption of ruksha (dry), sheeta (cold), laghu (light), and kashaya - tikta– katu rasa predominant foods, along with inadequate or irregular diet. Such dietary habits lead to Vata prakopa and contribute to agnimandya and ama formation.

Viharaja nidana comprises excessive physical exertion (ativyayama), night awakening (ratrijagarana), fasting or excessive langhana, suppression of natural urges (vegadharana), and improper daily routines. These factors disturb Vata and impair normal physiological functions.

Manasika nidana includes psychological factors such as chinta (stress), shoka (grief), krodha (anger), and bhaya (fear), which indirectly vitiate doshas, especially Vata.

Anya nidana involves factors like trauma (abhighata), dhatukshaya, chronic debilitating conditions, excessive blood loss, and most importantly ama, which plays a key role in the pathogenesis.

Thus, these factors collectively lead to Vata vitiation associated with ama, resulting in localization of pathology in the Trika – Kati - Prishta region and manifestation of restricted movements and pain.

Poorvarupa

Poorvarupa refers to the early, premonitory symptoms that appear during the sthana samshraya stage of shatkriyakala, when dosha–dushya sammurcchana begins. These features are usually avyakta (mild or indistinct) and indicate the initial stage of disease development, offering an important window for early diagnosis and intervention.

In Vatavyadhi, purvarupa are described as subtle and less manifested symptoms due to mild causative factors and incomplete expression of pathology. In conditions like Trika Kati–Prihsta Graha, these may include vague low back pain, mild stiffness, discomfort, slight restriction of movements, and difficulty in walking. From a modern perspective, these correlate with early symptoms of Ankylosing Spondylitis such as mild backache, muscle stiffness, and transient joint discomfort, which precede full clinical manifestation.

RUPA OF TRIKA KATI PRISHTAGRAHA

In a broad sense, the affliction of *Pratara* i.e., *Prishta Vamsha Sandhi,* by *Ama* and *Vata* constitutes the central pathological event in *Trika–Kati–Prishtagraha.* A *Sandhi* is formed by the junction of two or more bony ends and is supported by *Snayu Bandhana* (ligaments) and *Sleshaka Kapha*. Furthermore, the *Sandhi* is considered the origin (*Srotomula*) of *Majjavaha Srotas*.[12] From this, it may be inferred that *Asthi* and *Majja* are invariably affected in *Trika - Kati – Prishta graha*.

Additionally, the vitiated *Vata* exists in *Samavastha* (associated with *Ama*). Hence, the pathognomonic features of *Asthigata Vata***,** *Majjagata Vata*, and *SamaVata* may be correlated with *Trika–Kati–Prishtagraha*. The similarity between the characteristic features of these conditions and those of Ankylosing Spondylitis further supports this correlation.

A. Cardinal Signs and Symptoms

Each of the symptoms is being discussed here to understand its nature as well as to understand the *Samprapti* of this disease, being described elsewhere.

1) ***Stabdhata*:** At the initial stage, patients experience stiffness in the affected joints while performing bending movements, which may be considered *Stabdhata*[12] In *Trika–Kati–Prishtagraha*, this stiffness is attributed to the affliction of the joints by *SamaVata.*[14] when vitiated *Vata* settles in a *Sandhi* the function of *Sandhi* is lost. *Sushruta* has mentioned this immobility of the joint by saying *"Hanti Sandhe"*[15] Commenting on this *Dalhana* clarifies it as *"Akunchana Prasaranayoho Abhava"* i.e. loss of flexion and extension.

According to modern medical literature, patients with Ankylosing Spondylitis, particularly those with active sacroiliitis, almost invariably experience low back stiffness. The exact basis of this symptom remains unclear; however, it may be related to reflex muscle spasm. Initially, the stiffness is localized to the lower back and is most severe during the early morning hours. It is temporarily relieved with movement but tends to worsen again toward the evening. Periods of rest aggravate the symptom, and patients often report awakening at night with stiffness of such severity that they must get out of bed and move around before returning to sleep.[16]

According to modern science, the Ankylosing can be of two types:

I. Extracapsular stiffness of a joint due to induration or ossification of the surrounding tissues i.e. due to the involvement of muscles, ligaments, tendnns etc.
II. Intracapsular Stiffness of a joint due to the presence of bony or fibrous adhesions between the articular surfaces of the joint.[17]

2) *Sandhishula:* Severe and continuous pain in the affected joints, at times disturbing sleep, is one of the cardinal symptoms of *Trika–Kati–Prishtagraha*, as this condition primarily involves the *Asthi* and *Majja Dhatus* affected by *Vata Dosha*.[18] The characteristic features of

the *SamaVata* condition are also observed. These include aggravation of pain following the use of unctuous substances, during cold and cloudy weather, and during the morning and night hours.[51]

Pain in the buttocks, hips, sacral, or lumbar regions may be precipitated by twisting, jolting, lifting, coughing, or sneezing; however, it may also occur spontaneously. As the disease progresses, the pain tends to become constant and dull, producing persistent discomfort that is often described as a deep ache, a tired feeling, or soreness. The discomfort is typically more pronounced during the early morning hours. Patients may awaken around three or four o'clock in the morning and remain uncomfortable unless they take a hot bath or get up and move about (Munjal YP et al., 2019)

3) *Sparsha-asahyushnatata* – Although *Sparsha-asahyata* is not described separately in the *Ayurvedic* classics as a distinct symptom of *SamaVata*, the term *Sandhishula* broadly encompasses both subjective pain and, in some cases, objective tenderness. However, tenderness is not invariably present and may or may not accompany the experience of pain.
4) *Sandhi Shotha* - Swelling about the joint is the result of *Srotorodha* in the joint caused by the accumulation of *Ama* particularly *SamaVata.*[18]

According to modern texts; pain, tenderness, and swelling at the points of insertion of major tendons is very common. The site where a ligament or tendon inserts into bone is the enthesis. Inflammation at this sites is termed enthesitis. The disease process is referred to as enthesopathy. Bony erosions and new born formation follow at these sites most notably at the spinous processes, greater trochanters, pelvic bones and heels.[19]

The cardinal symptom *Trika-Kati-Prishta sandhigraha* is described here according to the stage of involvement.

TRIKAGRAHA

Trikagraha is one of the *Nanatmaja VataVyadhi. Bhavaprakasha* has mentioned that the vitiated *Vata* settles in the *Trikasandhi* and thus produces *Shula.*[20] *Trikasandhi*, the junction of *Prishtavansha asthi* and *Sphiga asthi*, correlates with the sacroiliac joint; therefore, *Shula* and *Graha* at this site signify primary sacroiliac joint involvement in the disease.

Trikagraha is also found as a symptom in the conditions like *Kapha pittadhika Tritiyaka Jvara*[21], *Sahaja Arsha*[22], general symptoms of *Madatyaya.*[23,24] *Vankshana Vidradhi.*[25] *Madhyama Rogamarga*[26], *Tritiyaka Jvara*[27], *Pakvashayakruddha Vata*[28] and *Tritiyaka Jvara.*[29]

KATIGRAHA

Katigraha is classified under the *Nanatmaja* disorders of *Vata*[30] Very little explanation is available in the classics regarding this disorder. *Kati* is also among the seats of *Vata. Bhavaprakasha* have mentioned that vitiated *Vata* alone or with the association of *Ama* lodge in the *Kati* and produces *Shula* and *Katigraha*. It is clear that the cardinal symptoms of this disease are *Graha* and *shula.* This condition is such that it caeses the lumbar spine movements like flexion, extension, rotation and bi-lateral movements thus hampering the day-to-day activities While explaining *Katigraha, Acharya Shodala* told that *ruja* as one of the prime symptom. Pain is confined to the *Katipradesha* or the Lumbosacral and sacroiliac region only. A comparable description of this condition is available in *Vangasena Samhita, VataVyadhi Adhyaya.*

Katigraha is also mentioned as a symptom under the following conditions a *Vrikkaja Vidradhi*[31], *Kshata Kshina Purvarupa.*[32] *general symptoms of Ajirna.*[33] *Vata Jvara.*[34], *kshataja Kasa,*[35] *Pakvasaya Kruddha Vata*[36],*Vankshana Vidradhi*[37], *Kshata Kshina*[38], *Vankshana Vidradh.*[39]

PRISHTA GRAHA

Prishtagraha is described as a general symptom of *VataVyadhi.*[40] However, a detailed description of its clinical features is not provided, and the commentators have also not elaborated further on its symptomatology. *Prishtagraha* is exhibited as a symptom in other conditions also. They are *Vrikkaja Vidradh*[41] *VataKapha dhika Tritiyaka Jvara,*[42] *Kshata Kshina Purvarupa.*[43] *Sahaja Arsha.*[44] *General symptoms of Ajirna,*[45] *General symptoms of VataVyadhi.*[46] *Vankshana Vidradhi.*[47] *Kshataja Kasa,*[48] *Kshata Kshina*[49] *Tritiyaka Jvara*[50], *and Vankshana Vidradhi.*[51]

UPASHAYA – ANUPASHAYA

Such of the medicine, diets and regimens which brings about long lasting happiness either by acting directly against the cause of the disease and or the disease itself or by producing such

effects indirectly are called *Upashaya*.[52] It provides diagnostic aid for disease which are otherwise difficult for diagnosis.

In the disease *Trika-Kati-Prishtagraha, Ruksha Sveda, Langhana, Ushna Kala* are considered to be the *Upashaya* while *sneha, Meghodayakala* and *Pratah Kala* are the *Anupashaya* in this disease. Though *Vata* is vitiated in this disease due to the association of *Ama; Ruksha Sveda* alleviates the signs and symptoms of *Trika Kati Prishtagraha*. On the contrary, *Sneha* increases *Ama* and thus makes the condition worse.Due to the presence of *Mandagni, Langhana* is beneficial in *Trika-Kati-Prishtagraha*, while santarpana may further spread up the formation of *Ama.* Cold season, cloudy weather, and early morning hours cause natural constriction of the body channels (*Srotas*). During these times, the predominance of *Sheeta Guna* (cold quality) increases the harmful effects of both *Vata* and *Ama*. Hence, these factors aggravate the symptoms and are considered as *Anupashaya*.

In contrast, application of mild heat and exposure to warm or hot climate, Such *yoga asana's* like *Bhujangasana, Shalabhasana, Halasana, Uttanapadasana* help relieve stiffness and pain, therefore are regarded as *Upashaya* in *Trika- Kati -Prishtagraha*.

SAMPRAPTI

Understanding the process of disease manifestation from the time of *nidana sevana* till the *Dosha-dushya samucchrana* is very necessary to plan the treatment, ie. *"Samprapti Vighatanameva Chikitsa"* As cited earlier, *Trikagraha, Katigraha* and *Prishtagraha* are the *SamaVataja Vyadhi*. *Bhavaprakasha* has described the pathogenesis of *Triksashula* according to which the vitiated *Vata Dosha* settles in *Trikasandhi* and produces *Trikashula.*[53] While prescribing the use of *Trayodashanga Guggulu* in *Trikashula, Bhavaprakasha* mentions *Trikagraha* as the first amongst the many indications for this drug. This naturally lends to the assumption that the author recognises the close relationship between *Trikagraha* and *Trikashula* which is also found practically in the patients.

As cited earlier, *Trikasandhi* is formed by the union of *Prishtavamsha Asthi* and *Sphiga Asthi*.[88] *Bhavaprakasha* and *Vangasena* have mentioned that vitiated *Vata* alone or in assotiation with *Ama* lodges in *Kati* and produces *Katishula* and *Katigr.*[54]

Madhava Nidana has elaborated the *samprapti of AmaVata* as follows. In state of pre-existing *Mandagni,* if the person is exposed to its etiological factors, then *Ama* is formed. The vitiated

Vata brings this morbid *Ama* to *Shleshma Sthana*, where it becomes more virulent. Then again while circulating through *Dhamanis* it further vitiates all the three *Doshas i.e Vata, Pitta and Kapha* as well as further gets vitiated itself and becomes heavy and vicious manifesting different colours. Due to these qualities it causes *Srotoabhishyandana* and *Srotorodha*. These changes taking place in the Srotas leads to the manifestation of symptoms like *Daurbalya*, *Hridgaurava* etc. when it involves *Trika* or *sandhi* it causes Stabdhata and then the condition of *AmaVata* comes into manifestation.[56]

Trika-Kati-Prishtagraha (Ankylosing Spondylitis) and *AmaVata* (rheumatoid arthritis) are considered as separate disease entities now a days. Even though there are certain features which are almost identical in both the diseases. The main causative factors of both the diseases is *Ama* and *Vata.* The involvement of *Pitta* and *Kapha Doshas* are also significant in both the diseases. *Dushyas* involved in both the diseases are *Asthi*, *majja a*nd *Rasa*. The *Srotas Asthimajjavaha* and *Rasavaha* are involved in both the diseases and in later stages of the disease, pathological changes occur in *Snayu*. In this regard *Charaka* quotes when aggravated, one and the same *Dosha* may cause manifold diseases depending upon the various etiological factors and the sites of manifestation.[57] So a physician should try to comprehend the nature of the disease, the site of its manifestation and etiological factors and should then initiate the treatment.[57]

In pathogenesis, *Khavaigunya* in the *srotas* is the most important factor determining the site of onset of illness. *Charaka* quotes that while being circulated if this *Doshas* accumulate at any place in the body, owing to the morbidity of the circulating passages it causes pathological changes there.[58] If *Khavaigunya* (morbidity) exists in the *Kora Sandhi* (synovial joints of radial extremities) thus offering a nidus, *Dosha Dushya Sammurchhana* may result in this place causing *AmaVata.* On the other hand if *Khavaigunya* exists in the *Trika* (sacroiliac joint) and *Pratara Sandhi* (spinal joints) offering a nidus for *Dosha Dushya Sammurchana*, manifestation of symptoms may take place at that point.

The manifestation of the symptom when *Trika* and *Pratara Sandhi* is involved varies depending upon the exact site, the precise subdivision of *Doshas* and the stage of involvement. Though separate *Samprapti* is not been described for *Trika-Kati- Prishta graha, VataVyadhi samprapti* of *Dhatukshayajanya VataVyadhi* and *Margavarodhajanya VataVyadhi* can be considered here.

Dhatukshayajanya Samprapti

Continued ingestion of *Ruksha, sheeta,* and *Laghu guna pradhana ahara,* along with improper *viharas* such as *Prajagara* (night awakening), *Vishamshana* (irregular eating), *Atyashana* (overeating), and *Vegadharana* (suppression of natural urges), leads to progressive *Dhatu kshaya* and *Vata prakopa*.

Due to the predominance of *Vata guṇas* like *Ruksha* and *Khara*, there occurs *Kapha kshaya* both gunatah (qualitative depletion) and karmatah (functional depletion). As *Kapha* is responsible for *sandhi-bandhana, sneha,* and *dhatu poshana,* its reduction results in loss of lubrication, stability, and nourishment of tissues.

Consequently, degenerative changes set in, leading to the formation of poor-quality tissues. This predominantly affects *Mamsa, Meda, Asthi,* and *Majja dhatus*, causing their progressive depletion and structural weakness.

Reduction of *Kapha* in Sandhis leads to *Sandhi-bandhana Shaithilya* (laxity and instability of joints). Due to the *Ashraya-Ashrayi sambandha* between Asthi and *Vata*, *Asthi dhatu kshaya* results in *Khavaigunya* (structural vulnerability) in the joints. Continued exposure to *nidanas* provokes *Vata prakopa*, which then circulates throughout the body via the *srotas*.

During this stage, the aggravated *Vata* undergoes *Sthana-samshraya* at the site of *Khavaigunya*, particularly the *Trika pradesha*. The localized *Vata* along with diminished *Kapha* produces clinical features such as *stambha* (stiffness) and *Shula* (pain), resulting in *Trika graha*.

Subsequently, owing to the *Chala guna* of *Vata*, the pathology progressively spreads to the *Kati* and *Prishta pradesha*, leading to pain, stiffness, and gradual restriction of spinal movements. This characteristic axial involvement is described in *Ayurveda* as *Trika - Kati - Prishta graha.*

Margavarodhajanya Samprapti

Katigraha can be manifested due to *Shuddha Vata* or by *SAma Vata*. This *SAma Vata* implies the *Margavarodhajanya* type of *Vikruti*. The causative factors like *Adhyaashana, VishAmashana* and other *Amakara Nidanas* first vitiates *Agni* leading to *mandagni*. Both *Jataragni* and *Dhatwagni* are dearranged leading to formation of *Ama* causing *srotavarodha* in the *Adhishtana*. Due to such *Srotavarodha* in the *Gati* of *Vata* produces *Lakshanas* like,

Ruk, Sthambha etc In the process of disease manifestation a vast number of factors invariably takes part and accordingly contribute for the pathogenesis. These are collectively called as *Samprapti Ghataka*. For the proper understanding of any disease, consideration of these factors is quite essential.

The following *Samprapti Ghataka* are being discussed in detail in relation to *Trika-Kati-Prishta graha.*

Table No. 1 Showing *Samprapti Ghataka.*

Dosha	*Vata*	*Prana, Udana, SAmana, Vyana & Apana Vata*
	Pitta	*Pachaka Pitta*
	Kapha	*Bhodaka, kledaka and shleshaka Kapha*
Dushya	*Dhatu*	*Rasa, Mamsa, Asthi and Majja*
	Upadhatu	*Sandhi and Snayu*
	Mala	*Mutra and Purisha*
Agni	*Jataragni, Dhatvagni*	
Ama	*Jataragnijanya and Dhatvagnijanya Ama*	
Srotas	*Rsavaha, Mamsavaha, Asthuvaha and Majjavaha*	
Srotodushti	*Sanga*	
Udbavasthana	*Amapakvashaya*	
Sancharasthana	*Sarva sharira*	
Vyaktasthana	*Trika, Kati and Prishta*	
Vyadhiswabhava	*Chirakari*	
RogAmarga	*Abhyantara and MadhyAma RogAmarga*	

Chikitsa

The management of *Trika -Kati–Prishta Graha*, correlated with Ankylosing Spondylitis, is based on the principles of *Vatavyadhi* and *Amavata chikitsa*. The primary objective is *samprapti vighatana*, which includes correction of *agnimandya*, *ama pachana*, pacification of aggravated *vāta*, and restoration of normal joint function.

Nidana Parivarjana forms the cornerstone of management. Avoidance of *vata* provoking factors such as *ruksha*, *sheeta ahara*, irregular food habits, excessive exertion, *ratrijāagarana*, and psychological stress is essential to prevent further progression of the disease.

Snehana (internal and external oleation) is indicated to counteract the *ruksha guna* of *vata*, promote tissue nourishment, and facilitate mobilization of vitiated *doshas*. *Eranda Taila*, owing to its *vatahara* and *anulomana* properties, is particularly beneficial in such conditions.

Swedana (sudation therapy) plays a significant role in relieving *stambha* and *gourava*, thereby reducing pain and improving flexibility. Procedures such as *Valuka Sweda* and *Patrapinda Sweda* enhance circulation and help in alleviating musculoskeletal stiffness.

Among **Shodhana therapies**, *Mridu Virechana*—preferably with *Eranda Taila*—is effective in expelling morbid *doshas* and relieving *srotorodha*. **Basti chikitsa**, being the prime therapy for *vata*, plays a pivotal role by regulating *vata*, nourishing *asthi - majja dhatu*, and improving spinal mobility.

Following purification, **Shamana chikitsa** is administered to pacify residual *doshas*. Formulations such as *Yogaraja Guggulu*, *Simhanada Guggulu*, and *Rasnadi Kashaya* are widely used for their *vatahara*, *shothahara*, and *vedanasthapana* properties.

Supportive measures including **Yogasana** (e.g., *Bhujangasana*, *shalabhasana*, *Makarasana*) and **Marma cikitsa** further aid in improving flexibility, reducing stiffness, and enhancing functional capacity.

Thus, a comprehensive approach integrating *nidana parivarjana*, *shodhana*, *shamana*, and rehabilitative measures provides an effective strategy in the management of *Trika – Kati - Prishta Graha*.

UPADRAVA

Upadrava (complication) occurs as a sequelae to the main disease. It may be in the nature of either a major or a minor ailment, The sequelae is so called because it is secondary to the disease. It is more troublesome than the main disease itself, because it appears in the later stages of disease when the body is already weakened.[59]

The manifestations resulting from the affliction of *Snayu* (ligaments and tendinous structures) may be included within the symptom complex of *Trika – Kati - Prshta Graha*, as the junction of *Snayu* and *Kandara* is functionally included under the term *Sandhi*. Since *Snayu* binds the bony ends and forms an integral component of joint structure, its involvement is commonly evident in this condition.

This classical view is supported by modern understanding, which demonstrates that inflammation in Ankylosing Spondylitis primarily occurs at the enthesis, including the intervertebral ligaments and surrounding structures. These inflammatory changes are

followed by structural alterations such as fibrosis, calcification, and new bone formation, ultimately leading to ankylosis of the spine.

The complication seen in Ankylosing Spondylitis can be treated synonymous to *Upadravas* of *Trika-Kati-Prishta Graha*. As cited carlier, *Trika-Kati-Prishtagraha* is considered as *VataVyadhi*. Hence *Upadrava* of *Trika-Kati-Prishtagraha* can be deduced as *Upadravas* seen *Charaka* has described certain conditions in the chapter dealing with *VataVyadhi Chikitsa,* according to *Chakrapani* which should be understood as *Upadravas* of this disease *Sushruta* has devoted a full chapter for Upadravas occuring in different diseases where *Vata Vyadhi* is also included.[60] Among these *Upadravas* of *VataVyadhi - Bhagna, Kubjata, Sankocha, Pangulya, Sandhichyuti and Supti* are of importance in the disease *Trika-Kati-Prishtagraha*. The *Upadravas* of *VataVyadhi* given by *Charaka and Sushruta* are enumerated herein.

1. ***Bhagna*****:** *Sushruta* has enumerated this complication under *VataVyadhi*. The spine of patients with Ankylosing Spondylitis is extremely vulnerable to trauma and seeingly insignificant strain or trauma may result in a fracture. The cervical spine is involved most frequently.

2. ***Kubjata*****:** Is one among the *Nanatmaja* disorders of *Vata* and also enlisted under the general symptoms of *VataVyadhi.* It is well recognised that with progressive long standing of this disease the dorsal spine nay develop a kyphotic configuration. Entire cervical dorsal spine may be aligned in a single kyphotic curve.

3. ***Pangulya*****:** The vitiated *Vata* alone or in association with *Ama* lodges in the *Kati* and produces *Katigraha.* When it lodges in both the *Sakthi* it leads to *Pangulya.*[61] *Pangulya* is also among the Nanatmaja disorder of *Vata* and the general symptoms of *Vata*. Paraplegia in the patients of Ankylosing Spondylitis is usually secondary to dislocation at the atlanto- axial joint or fracture of the brittle spine.

4. ***Sandhichyuti*****:** Among the effects produced on the body by the action of vitiated *Vata, Bhramsha* is also mentioned.[61] *Chakrapani's* commentary assumes this to be *Sandhichyuti,* which is one of the *upadravas* of *VataVyadhi*. Spontaneous atlanto axial dislocation in an uncommon and usually unrecognised complication.

5. ***Sankocha:*** Abnormal fixed state of any limb in flexion or inability to extend the limb is termed as *Sankocha.*[62] This is caused due to the affliction of *snayu* and *Kandara* by vitiated

Vata. It is seen that patients with a significant cervico-dorsal kyphotic curve develop compensatory flexion at the hips and knees in order to maintain their peripheral vision.

6. ***Supti*:** Sushruta has enumerated *Supta Tvacham* under the complication of *Vata Vyadhi*[63] *Charaka* quotes that *supti* is a characteristic feature of affliction of *Snayu*. Due to involvement of the lumbo-sacral nerve root, loss of sensation occurs in the saddle area, posterior thighs and lateral aspects of the feet.

7. **Miscellaneous:** Modern texts describe some of the neurological disorders as a complication of Ankylosing Spondylitis. The Cauda Equina syndrome is a rare complication appearing usually in patients with long standing and apparently inactive disease. The costovertebral joints are involved and the resulting fixity of the thorax predisposes to pulmonry complications. In short, *Bhagna, Kubjata, Pangulya, Sandhichyuti Sankocha, supti* and other such conditions can reckoned to be the *Upadravas* of *Trika-Kati-Prishtagraha*. Affliction of *Snayu* causes *Kubjata, Sankocha* and *Supti*. The manifestation of these *Upadravas* indicate poor prognosis of the disease.

SADHYA - SADHYATA

The presence of virulent *Ama* with the dominance of *Vata*, involvement of *Madhyama Roga Marga* and perpetual affliction of the whole spine illustrating the severity and lingering nature of the disease. However, the variance in the causative agents, severity of the Doshic involvement as well as affliction of *Dushya* and difference in idiosyncratic bodily resistance for the disease contributes to the Variation in the severity of manifestation of the disease and thereby responsible for good or poor prognosis.

Accordingly, in *Trika–Kati–Prishtagraha*, the presence of fewer etiological factors (*Nidana*), mild manifestation of symptoms, involvement of a limited number of joints, and absence of *Upadravas* indicate a favourable prognosis (*Sadhya*).

Conversely, association with other *Doshas* (*Anubandha*), extensive involvement of multiple spinal joints, severe symptomatology, and the presence of complications (*Upadravas*) suggest a difficult prognosis (*Krcchra Sadhya*) or even incurability (*Asadhya*).

These principles are in accordance with the general prognostic guidelines described for *VataVyadhi* in Charaka Samhita, where chronicity, multisystem involvement, and development of complication are considered as markers of poor therapeutic response.

According to modern medicine, early in the course, one cannot predict the ultimate prognosis for an individual patient. However, patients with involvement of peripheral weight bearing joints appear to suffer greater functional impairment than those with spinal disease alone. With adequate management, functional capacity declines slowly with advancing age. Mortality due to inherent features of the disorder is quite low. Death results primarily from uncommon sequelae, such as aortic insufficiency and secondary amyloidosis (Munjal et al., 2019).

PATHYA – APATHYA

Pathya (Dietary regulation) holds equal importance to therapeutic measures in the management of disease. *Ayurvedic* classics clearly emphasize that the success or failure of treatment largely depends upon strict adherence to *Pathya* and avoidance of *Apathya*. Without appropriate dietary and lifestyle discipline, even well-planned medications may not produce the desired results.

In the classical texts, the condition *Trika – Kati - Prishta graha* is not described as a separate disease entity; however, its symptomatology and pathogenesis are explained in brief under *Vata Vyadhi* and related disorders. Clinically, this condition closely resembles *AmaVata*. In this context, *Ama* and aggravated *Vata* are considered the principal causative factors. Therefore, the *Pathya* and *Apathya* prescribed for *AmaVata* can be suitably advised for Substances possessing *Katu* and *Tikta rasa, Ushna* and *Tikshna guna,* and having *Vatahara* and *Ama-pachana* properties are regarded as wholesome. Such diets help in digestion of *Ama,* reduction of *Kapha,* and normalization of aggravated *Vata,* thereby reducing pain, stiffness, and inflammation.

The Pathya's mentioned in the classics may be classified as follows:
(*Yoga Ratnakara*)

1. Monocotyledons: *Raktashali, Yava*
2. Dicotyledons (pulses/millets): *Kulattha, Kodrava, Shyamaka*
3. Vegetables: *Ardraka, Lasuna* with *Takra, Patola, Punarnava, Shigru*
4. Non-vegetarian preparations: *Jangala mamsa rasa*
5. Liquids: *Ushnodaka* (boiled warm water).

Apathya

Just as *Pathya* plays an important role in disease management, avoidance of *Apathya* is equally essential. Substances possessing *Madhura* and *Amla rasa*, along with *Guru, Picchila, Sheeta*, and *Abhishyandi guna*, are considered unwholesome in *Trika - Kati - Prishta graha.* These factors aggravate *Vata* and promote the formation and accumulation of *Ama*, thereby worsening stiffness, inflammation, heaviness, and restriction of joint movements. Hence, such diets and regimens are contraindicated.

The *Apathya ahara-vihara* described in *Yoga Ratnakara* may be classified as follows:

A) Ahara (dietary factors)

1. Dicotyledons: *Masha*
2. Vegetables: *Upodika*
3. Non-vegetarian items: Matsya
4. Liquids: Unclean or impure water
5. Miscellaneous: *Guda*, unwholesome or heavy diet, *Vishamashana* (irregular eating habits)
6. Cow products: *Dadhi* and *Kshira*

B) Vihara (lifestyle factors)

1. Exposure to easterly cold wind
2. Night wakefulness (*ratri jagarana*)
3. Suppression of natural urges (*vegadharana*)

Review on Ankylosing Spondylitis

Ankylosing spondylitis is a composite term derived from two words: *ankylosing* and *spondylitis*. The term *ankylosing* originates from the Greek word *ankylos*, meaning hooked, crooked, fusion, or immobility, indicating stiffness or fixation of a joint.[64] According to standard medical classification, ankylosis may be extracapsular, due to involvement of surrounding soft tissues, or intracapsular, due to fibrous or bony adhesions within the joint.

The term *spondylitis* is derived from the Greek word *spondylos*, meaning vertebra, and denotes inflammation of the vertebrae. Collectively, ankylosing spondylitis refers to a pathological condition characterized by inflammation of the vertebrae leading to progressive rigidity and eventual fusion of the vertebral column.[65]

Ankylosing spondylitis is defined as a chronic inflammatory disease primarily affecting the axial skeleton, particularly the sacroiliac joints and spine.[65,66,67] The disease typically begins at the sacroiliac joints and gradually ascends, resulting in ossification of spinal ligaments and adjacent soft tissues, ultimately causing restriction and loss of spinal mobility[66,67,68] In advanced stages, peripheral joints may also become involved

1. Unknown Specific Cause and Hereditary Predisposition

In modern medicine also the specific causes of Ankylosing Spondylitis are not known. Predisposition for the disease appears to be influenced by heredity. Recurrent reports of disease involvement among twins, siblings, parent–offspring pairs, and multiple affected individuals within the same family suggest a significant familial aggregation and genetic susceptibility. Stecher and colleagues studied that disease was found to be 30 times more prevalent among the relatives of spondylitic patients than among the relatives of non-spondylitic controls. In this way, *Adibala Pravrtta* and *Janmabala Pravrtta* may have been playing a major part as *Utpadaka hetu* here in the production of this disease.[69,70]

2. Precipitating and Contributory Factors (Multifactorial Triggers)

Various precipitating factors, including trauma (*Abhighata*), excessive physical exposure or strain, different types of infections, psychological stress (*Manasika Hetu*), and preceding debility or tissue depletion (*Dhatu Kshaya*), have been considered contributory in the manifestation of the disease. A study conducted by Philip Boland and Alfred observed that in nearly 50% of cases, no definite immediate precipitating or predisposing factor could be identified, while in the remaining cases, no consistent etiological factor was demonstrable. These observations suggest the multifactorial nature of the disease, which may be better understood through the *Ayurvedic* concepts of *Nidana, Dosha–Dhatu Vaishamya*, and individual susceptibility (*Vyadhi Kshamatva*).[71]

3. Early Infectious Theories

Earlier hypotheses proposed an infectious origin for the disease, with suggestions ranging from gonococcal to non-gonococcal genitourinary infections as initiating factors. However, current evidence does not support a direct infectious etiology. Instead, the disease—now well recognized as a form of axial spondyloarthritis—is understood to be a chronic immune-mediated inflammatory disorder with a strong genetic predisposition, particularly associated with the HLA-B27 allele. While infections are no longer considered primary causative agents, certain microbial triggers, especially from the gut and genitourinary tract, are believed

to play a role in initiating or modulating the immune response through mechanisms such as molecular mimicry and dysbiosis. This has shifted the perspective from a purely infectious cause to a complex interaction between host genetics, immune dysregulation, and environmental factors, including the microbiome.

4. Constitutional and Systemic Inflammatory Features

Patients with Ankylosing Spondylitis may present with general constitutional manifestations such as elevated erythrocyte sedimentation rate (ESR), low-grade fever, fatigue, weight loss, and anemia of chronic disease. These systemic features reflect the presence of chronic inflammation affecting the axial skeleton and sacroiliac joints. Earlier investigators proposed an infectious etiology because of these inflammatory manifestations; however, no specific causative organism has been identified. Current research indicates that Ankylosing Spondylitis is a chronic immune-mediated inflammatory disorder strongly associated with genetic susceptibility, particularly the HLA-B27 antigen, along with environmental and immunological factors.[69]

5. Other Proposed Theories with Limited Evidence

Other theories of etiology such as metabolic, allergic and endocrine have been advanced with little or no evidence to support them. Recent research strongly supports the role of genetic predisposition in the etiology of Ankylosing Spondylitis. Advances in the study of the Human leukocyte antigen system (HLA) have conclusively demonstrated that hereditary factors significantly contribute to disease pathogenesis. HLA molecules, analogous in function to the ABO blood group antigens, are cell-surface glycoproteins present on most nucleated cells, The genetic blueprint for these antigens is located on a cluster of closely linked genes within the major histocompatibility complex on chromosome 6. Each individual possesses a unique HLA profile determined by inherited alleles. Earlier identification methods relied on serological typing using anti-HLA antibodies obtained from multiparous women or multiply transfused individuals, based on complement-mediated cytotoxic reactions of lymphocytes. However, contemporary techniques such as molecular typing and polymerase chain reaction (PCR)-based assays now provide more precise characterization.

To date, numerous HLA antigens have been identified, primarily encoded by the class I gene loci : HLA-A, HLA-B, and HLA-C. Among these, the HLA-B27 antigen shows a particularly strong association with Ankylosing Spondylitis, underscoring the central importance of genetic and immunological mechanisms in the development of the disease.

8. HLA Typing and Immune-Mediated Mechanisms

Following the establishment of the practical utility of Human leukocyte antigen system (HLA) typing, researchers began systematically evaluating the HLA profiles of patients with various diseases of previously unknown etiology. Particular attention was directed toward disorders suspected to have immune-mediated or autoimmune mechanisms. Experimental studies in animal models demonstrated that genes regulating the magnitude of humoral and cellular immune responses are closely linked to the HLA gene complex located on chromosome 6. This observation suggested that individuals with a predisposition to immune or autoimmune disorders might be identifiable through their specific HLA patterns.

Among the many conditions investigated, the most significant and consistent association was observed in Ankylosing spondylitis, where a strong correlation with the HLA-B27 antigen allele was demonstrated. This finding was initially unexpected, as Ankylosing Spondylitis had not traditionally been classified as a classical autoimmune disease based on conventional serological markers such as complement levels, immunoglobulin titers, rheumatoid factor, or antinuclear antibodies. Contemporary understanding, however, recognizes the condition as an immune-mediated inflammatory disorder rather than a typical antibody-mediated autoimmune disease.

9. Gut–Skin–Joint Axis and Associated Disorders

Furthermore, inflammatory spinal manifestations such as sacroiliitis and spondylitis are frequently observed in association with conditions like ulcerative colitis, Crohn's disease (regional enteritis), and psoriatic arthritis. It has been proposed that disruption of intestinal or cutaneous barrier integrity in these disorders facilitates microbial translocation and immune activation, thereby contributing to spinal inflammation and disease pathogenesis.

Epidemiology of Ankylosing Spondylitis

1. Prevalence in Asia[72]

The prevalence of AS in Asia varies by region and ethnic group, generally falling within 0.1% to 1.4% of the population. Population-based estimates suggest mean prevalence of about 16.7 per 10,000 people in Asia (≈0.17%).

2. Prevalence in India[72]

Epidemiological studies from India estimate AS prevalence at approximately 7–9 per 10,000 persons (≈0.07–0.09%).Other reports indicate a similar range of 0.1% to 0.5% in Indian

populations. Hospital-based data demonstrate that a substantial proportion of Indian AS patients test positive for the HLA-B27 antigen.

3**. Sex and Age Distribution**[73]

AS commonly begins in young adults, most often in the second to fourth decades of life. Male predominance remains consistent in Indian studies, with male-to-female ratios reported around 2.7:1 in recent samples.

4. HLA-B27 Association in India

The HLA-B27 antigen - a major genetic risk factor—is found in a high proportion of AS patients, though the exact frequency varies by region and subtype. HLA-B27 frequency among Indian spondyloarthritis (SpA) patients ranges widely (≈30–94%). In the general Indian population, HLA-B27 prevalence is much lower (~1–6%).[74] Subtype profiling in South Indian (Tamil) AS patients shows predominant HLA-B27 subtypes such as HLA **B27:04** and HLA B*27:05*, reflecting geographic and ethnic variation in genetic risk.[73]

5. Ethnic and Geographic Variation

AS prevalence closely correlates with the frequency of HLA-B27 in the population; regions with higher HLA-B27 positivity generally show higher AS prevalence. Studies indicate that the spectrum of HLA-B27 subtypes associated with AS in Indians differs from that in Western populations, underscoring ethnic influences on disease expression.

6. Overall Etiological Concept

Although the exact etiology of Ankylosing Spondylitis has not been fully elucidated, current evidence suggests that the disease arises from a genetically determined predisposition interacting with one or more unidentified environmental or precipitating factors.

Cardinal Signs and Symptoms

Sacroiliac Joint Involvement

With involvement limited to these two joints, the symptoms primarily include aching, stiffness, and pain localized to the lower back. Tenderness over one or both sacroiliac joints is elicited on percussion or deep palpation in approximately 50% of patients. As ankylosis develops, both tenderness and pain tend to subside. Orthopedic maneuvers that induce movement of the sacroiliac joints may reproduce localized pain. Mild paravertebral muscle spasm without restriction of lumbar mobility is commonly observed; however, pronounced

muscle spasm usually suggests involvement of the lumbar apophyseal joints. (Munjal YP et al., 2019).

Sacroiliac involvement tests.[75]

To know sacroiliac irritability following methods can be adopted

1) Pressing down firmly on the Anterior Superior Iliac Spine and forcing the pelvis backward when patient is lying in a supine position.

2) With the hip and knee flexed to 90° while maintaining support to the lumbar lordosis, a posteriorly directed thrust applied through the knee reproduces pain.

3) The most sensitive tests : compressing the iliac bones towards each other, when patient is lying in a supine position, then positive pain will be produced in the region of the involved sacroiliac joint.

4) Pressing firmly downwards on the sacrum with the patient lying prone.

5) Fixing one shoulder, with the patient lying supine and flexing the ipsilateral thigh cause pain.

6) Hyper extending the one hip while fully flexing the other causes pain in the sacroiliac joint - Gaenslen's sign.

7) Active S.L.R. test: The patient is asked to lift the thigh up with the knee extended. This will cause pain at the pathological sacroiliac joint due to the rotational strain imposed on this joint.

Lumbar Involvement

This segment is rarely involved in isolation, as the disease almost invariably extends upward from the sacro-iliac joints. Pain and stiffness remain the predominant symptoms; however, they have limited localizing value. The following clinical signs suggest involvement of the lumbar spine

1. Limitation of lumbar spine movements
2. Paravertebral muscle spasm
3. Straightening (loss of normal lumbar lordosis) of the lumbar spine
4. Deep tenderness on palpation over the lumbar vertebrae
5. Pain aggravated by forced flexion and extension of the lumbar spine
6. Muscle atrophy, particularly in the lower lumbar region, which, when associated with loss of lumbar lordosis, produces an “ironed-out” appearance of the lower back (Munjal YP et al., 2019).

Lumbar Spine Motion tests

The modified Schober test**,** described by Macrae and Wright, is considered one of the most reliable methods for quantifying the loss of lumbar spinal mobility Lumbar anterior flexion is assessed with the patient standing erect. A skin mark is placed over the spinous process of the fifth lumbar vertebra (L5). Two additional marks are then made: one 10 cm above and another 5 cm below the initial mark. The patient is instructed to bend forward maximally, and the distance between the upper and lower marks is re-measured. Normally, the original 15 cm distance should increase by at least 5 cm during full flexion in individuals younger than 50 years. A lesser increase indicates restriction of lumbar mobility. Serial measurements in the same patient are useful for monitoring disease progression. In cases of spinal ankylosis, no significant separation of the skin marks is observed (Harrison et al., 2022).

Thoracic Involvement

When the thoracic segment is affected the same localizing signs are present as are outlined for the lumbar segment, but at a higher spinal level. In addition the following special features usually develop

1. Thoracic girdle pain.
2. Chest pain on deep inspiration.
3. Diminished chest expansion.
4. Flattening of the anterior chest (expiratory position).
5. Thoraco-lumbar kyphosis.

Thoracic Involvement test

Involvement of the costo-vertebral joints produces restriction of inspiratory capacity which may be objectively measured by calculating the difference of chest circumference during inspiration and expiration. This is usually measured at two levels, one at the level of nipples and the other at the level of xiphoid process. A value below one centimeter is very suggestive of thoracic cage immobility.

General Symptoms

The vitiation of *Rasa Dhatu* by aggravated *Vata* in association with *Ama* produces both localized and constitutional manifestations. These include *Aruchi* (loss of appetite), *Jvara* (fever), *Panduta* (pallor), and *Nadi-drutatva* (tachycardia). Due to *Srotorodha* of the *Mutravaha Srotas* caused by *Ama* and *Vata*, the patient may also experience dysuria. As the condition is categorized under *Asthi-Majjagata Vata*, progressive *Mamsa-kshaya* (muscle

wasting) is commonly observed in patients presenting with *Trika–Kati–Prishtagraha*, as mentioned in *Charaka Samhita*

From a modern clinical perspective, Ankylosing Spondylitis commonly presents with systemic manifestations such as low-grade fever, anorexia, weight loss, fatigue, muscle atrophy, and recurrent episodes of iritis. Less frequent findings include anaemia, lymphadenopathy, splenomegaly, vasculitis, secondary amyloidosis, and cardiovascular involvement (Munjal YP et al., 2019).

They can be classified as

1. **Neuroligical involvement**: Neurological involvement may occasionally accompany the disease, most commonly due to nerve root compression, radiculitis, or sciatica. Other contributing factors include vasculitis, atlanto-axial subluxation, and cauda equina lesions, resulting in varying degrees of sensory and motor impairment.

2**. Extra Spinal Musculoskeletal Disease**: Transient or chronic involvement of peripheral joints occurs in 50 % of cases especially in the hips and knees but is not common in small joints of the hands and foot. Peripheral arthritis occasionally monoarticular, may become chronic in about one fourth of spondylitis cases.

3. **Extra Musculo Skeletal Disease**

a) Uveitis : Acute Anterior Uveitis is observed in 20 to 30% of patients and may be recurrent.

b) Cardiovascular Diseases (Spondylitic heart disease): Aortic valve incompetence is present in 3% of patients and may result in severe Aortic regurgitation. Conduction abnormalities including varying degree of heart block and left bundle branch block. The conduction defects are more apt to appear in patients with aortic valve incompetence. Some patients require implantation of pacemaker.

c) Pulmonary Disease: Bilateral upper lobe fibrosis is a recognised late manifestation of Ankylosing Spondylitis and may mimic tuberculosis.

4. **Constitutional Symptoms**: Constitutional symptoms are generally mild at the onset and tend to remain subtle throughout the course of the disease. However, in a small proportion of patients with severe disease activity, features such as fatigue, anaemia, low-grade fever, and weight loss may be observed, reflecting systemic involvement.

Pathology of Synchondroses and Enthesis in Ankylosing Spondylitis

Based on his observations of the manubriosternal joint and symphysis pubis, Cruickshank, proposed a sequential concept to explain the pathology of synchondroses in ankylosing spondylitis. In the initial stage, an inflammatory osteitis develops in the subchondral bone, characterized by infiltration of chronic inflammatory cells. Radiographically, this early phase appears as osteopenia. Subsequently, the articular cartilage and adjacent bone are destroyed by osteoclastic activity and the area becomes infiltrated with granulation tissue. These histopathological changes correspond to erosions and widening of the cartilage space on imaging.

During the healing phase, reossification and bridging of the cartilage space occur, producing sclerotic bone and eventually leading to ankylosis. In later stages, partial resorption of the newly formed bone may occur, and radiographs may show bone of near normal density replacing the cartilage space.

Postmortem studies of the intervertebral disc joints demonstrated that ossification is the most common change. In early or limited lesions, ossification is confined to a strip along the anterior or posterior surfaces of the discs, although both surfaces may be involved. These bony proliferations originate near the vertebral border and may extend across the disc space. Such changes produce the radiographic appearance of syndesmophytes. When multiple contiguous discs are affected, particularly in the dorsolumbar region, the characteristic bamboo spine deformity develops. Ossification may also involve the anterior vertebral body adjacent to the disc. The same sequence of osteitis, osseous destruction, granulation tissue infiltration, and reossification likely explains these peripheral disc and vertebral body changes.

Further studies have demonstrated that the localization of lesions in Ankylosing Spondylitis is closely related to inflammation at the enthesis, the site of attachment between ligaments or tendons and bone. This process, termed enthesitis, is now considered a hallmark feature of spondyloarthritis. Inflammatory changes have been observed at sites such as the iliac crest, greater trochanter, and patella. The enthesis represents a transition zone from fibrous tissue to cartilage, calcified cartilage, and bone, and is characterized by high metabolic activity, making it particularly susceptible to inflammatory processes.[76]

Only the outer layers of the fibrocartilaginous discs are vascularized, and their junction with the vertebra functions similarly to an enthesis. Therefore, lesions in Ankylosing Spondylitis tend to localize at these fibro-osseous interfaces. Healing occurs with deposition of new bone in the outer layers of the annulus, leading to syndesmophyte formation. These syndesmophytes may enlarge due to persistent inflammatory activity at their tips. Ossification of the anterior longitudinal ligament and the capsules of apophyseal joints may also occur. Synovitis is usually minimal, although proliferative synovitis similar to that seen in rheumatoid arthritis has been reported in some cases.

Current understanding suggests that sacroiliac joint involvement in Ankylosing Spondylitis begins with inflammation at the enthesis and subchondral bone, followed by structural changes including cartilage d*ama*ge, erosion, and subsequent new bone formation leading to ankylosis. Progressive ossification and fusion of the joint occur as a result of chronic inflammation and repair mechanisms. Peripheral joints may also be involved and can exhibit synovitis, with features such as inflammatory cell infiltration and structural d*ama*ge. However, unlike rheumatoid arthritis, the predominant pathology in Ankylosing Spondylitis is entheseal inflammation and new bone formation rather than purely synovial proliferation.[77]

In the later stage of *Trika-Kati-Prishta graha*, total loss of movement is commonly seen due to fibrous or bony ankylosis. In the symptoms of *Pravriddha AmaVata*, *Jadyata* is mentioned, term refers to the loss of function.[78] From this it can be congenctured that the fibrous or bony ankylosed joints seen in the patients of *Trika-Kati-Prishta graha* falls within the purview of *Jadyata* where complete restriction of the joint movement is inferred.

Sandhi jadyata (joint stiffness and immobility) occurs due to *Sandhi slishtata* produced by abnormally increased *Kapha* within the joints. The excessive *Kapha*, owing to its *Snigdha, Manda,* and *Sthira gunas*, causes increased compactness, heaviness, and reduced mobility of the *Sandhis*, thereby restricting normal joint movements.

Dalhana, in his commentary on *Sushruta Samhita*.[79] describes this condition as *Adhyasthi-slishtata***,** wherein abnormal bony deposition or excessive hard tissue proliferation occurs around the joints. This leads to adhesions, fusion, and consequent immobili.

Principal features differentiating Ankylosing Spondylitis from Rheumatoid Arthritis[80]

1. Ankylosing Spondylitis shows a higher prevalence in males, whereas Rheumatoid Arthritis is more common in females.
2. Ankylosing Spondylitis predominantly involves the axial skeleton, especially the sacroiliac joints and spine, while Rheumatoid Arthritis mainly affects peripheral joints.
3. Peripheral joint involvement is less frequent and usually asymmetric in Ankylosing Spondylitis, whereas it is symmetrical in Rheumatoid Arthritis.
4. Bilateral sacroiliitis is a characteristic radiological feature of Ankylosing Spondylitis, which is absent in Rheumatoid Arthritis.
5. Inflammatory back stiffness is more prominent than pain in Ankylosing Spondylitis, especially in early morning hours.
6. Subcutaneous nodules are typically seen in Rheumatoid Arthritis but are absent in Ankylosing Spondylitis.
7. Extra-articular manifestations such as recurrent uveitis are more common in Ankylosing Spondylitis.
8. Ankylosing Spondylitis is associated with HLA-B27 positivity, whereas Rheumatoid Arthritis is associated with rheumatoid factor and anti-CCP antibodies.
9. Rheumatoid factor is usually negative in Ankylosing Spondylitis.
10. Ankylosing Spondylitis shows new bone formation (syndesmophytes), whereas Rheumatoid Arthritis leads to joint erosion.

Clinical laboratory studies

In Ankylosing spondylitis, routine laboratory investigations are generally of limited value for diagnosis. C-reactive protein (CRP) is considered a more sensitive and dependable marker of active inflammation compared to ESR. CRP levels rise rapidly with inflammatory activity and correlate better with disease progression and response to treatment. Therefore, CRP serves as a more useful investigation for diagnosing and monitoring disease activity.

Mild elevations of creatinine phosphokinase (CPK), serum glutamic oxaloacetic transaminase (SGOT/GOT), and alkaline phosphatase may be observed in some patients. Rheumatoid factor is typically negative, which helps differentiate Ankylosing Spondylitis from rheumatoid arthritis. Synovial fluid obtained from peripheral joints usually shows Class II inflammatory characteristics, including poor viscosity, poor mucin clot formation, and increased white blood cells, resembling the findings seen in rheumatoid arthritis.

Detection of the HLA-B27 antigen strongly supports the diagnosis of Ankylosing Spondylitis in clinically suspected cases, particularly in the early stages before definite radiographic changes appear. However, its diagnostic utility is limited, as not all patients with Ankylosing Spondylitis are HLA-B27 positive, and the antigen may also be present in healthy individuals. Therefore, HLA-B27 testing alone cannot be used as a definitive diagnostic tool and should always be interpreted in conjunction with clinical features and imaging findings.

Radiological Findings

The most characteristic radiological feature of Ankylosing Spondylitis is bilateral sacroiliitis. In the early stages, it appears as widening and irregularity of the sacroiliac joint space, followed by subchondral sclerosis, erosions, and eventual ankylosis.

On lateral radiographs of the lumbar spine, erosions are observed at the anterior margins of vertebral bodies near the intervertebral disc spaces, leading to squaring of the vertebrae and loss of normal lumbar lordosis. As the disease progresses, ossification of the anterior longitudinal ligament and fusion of the apophyseal joints occur.

New bone formation at vertebral margins results in the development of syndesmophytes, which may bridge adjacent vertebrae. In advanced stages, this leads to the classical “bamboo spine” appearance.

Additional findings may include involvement of the hip joints with joint space narrowing, erosions, and ankylosis, as well as changes in the symphysis pubis. Extra-articular manifestations such as apical pulmonary fibrosis and aortic involvement may also be observed.(Sieper et al., 2017)

Peripheral joint involvement is also observed in Ankylosing Spondylitis, with the hips being the most commonly affected peripheral joints, involved in approximately 30–50% of cases, while the shoulders and temporomandibular joints are less frequently involved. These joints may demonstrate radiographic changes such as joint space narrowing, erosions, and eventual ankylosis. Although some features may resemble those seen in rheumatoid arthritis, the pattern of involvement and underlying pathology differ, with Ankylosing Spondylitis predominantly characterized by enthesitis and new bone formation.

Imaging in Early Detection of Sacroiliitis

Several imaging modalities have been utilized for the early detection of sacroiliac joint involvement in Ankylosing Spondylitis. Although radionuclide bone scanning can detect increased osteoblastic activity at sites of inflammation and may identify early changes before they are visible on plain radiographs, its specificity is limited. The uptake of radiotracers such as technetium-99m reflects increased metabolic activity associated with inflammation and bone remodeling.

However, magnetic resonance imaging (MRI) is currently considered superior to both conventional radiography and radionuclide scanning for early diagnosis, as it can directly demonstrate active inflammation in the sacroiliac joints, including bone marrow edema and soft tissue changes. In advanced stages, diagnosis becomes more straightforward due to characteristic clinical and radiographic features, including marked spinal rigidity and the classical “bamboo spine” deformity.

BASIC SUPPORTIVE MEASURES

In perspective of modern medicine, in the absence of a curative agent, effective management depends on early dignosis, patient co-operation and various therapeutic objectives. Management also includes basic supportive measures which are described as follows:

1. Education: The first step to educate the patient and family members on the nature of the disease and their roles in treatment. Well motivated individuals who are taught to understand and follow a long range programme of management always do better than those whose interests and instructions are superficial and who looked only for attainment of immediate comfort. with proper counselling, continuous encouragement and a careful individualised comprehensive programme of management, most patient will continue to lead productive lives.

2. Postural Training: Postural training plays a vital role in the management of ankylosing spondylitis. Patients should be instructed to maintain an erect posture while standing and walking, avoid stooping, and adopt proper squatting techniques. Sitting should be done on a hard, straight-backed chair to support spinal alignment. Sleeping in a supine position on a firm mattress without pillows under the head or knees helps prevent flexion deformities. The use of appropriate braces may assist in maintaining posture and reducing muscle spasm.

Regular rest intervals during the day are also beneficial in relieving fatigue and preventing postural drooping.

3. Exercise and Sports: Exercise is fundamental for prevention of deformity. Emphasis must be placed on strengthening muscle groups that oppose the direction of potential deformities (extensors rather than flexors). Exercises should be performed vigorously and deliberately unless the inflammation is acute; then they should be gentle and assisted. The patient should be taught, trunk stretching exercises both in erect and supine positions, calf and hamstring stretching exercises and others as indicated. Deep breathing exercise in which the patient regularly stretches the chest, simultaneous upward and backward movement of the shoulders may prevent chest restriction. Exercise programs are individually prescribed according to age, strength, degree of involvement and ability to cooperate. Warm tub or shower, paraffin or other, may help in relief of joint pain, stiffness and may be useful before exercising. Sports and recreational activities planned on the basis of kinesiological analysis can significantly support the general therapeutic objectives in ankylosing spondylitis. Such activities help maintain joint mobility, improve muscle strength and flexibility, enhance posture, and prevent stiffness and deformity. With qualification, archery, swimming and bait casting may be helpful, while bowling, golfing and surf-casting may prove harmful.

4. Rest: Complete bed rest should be avoided and the patient should be encouraged to exercise daily even when fever and fatigue are present. Daily exercise, performed by the patient at home, must be planned individually and a proper balance sought between rest and activity.

5. The Psychosocial and Rehabilitative needs: The psycho-social and rehahilitative needs of the patient deserve the most thoughtful attention. Psychologic support (as in any chronic disease) may be further enhanced by a healthy relationship between patient, his physician and staff. Patient may feel the effects of their disease by a reduced earning potential, unless they become adjusted to work which does not demand heavy lifting, undue exposure or fatigue or prolonged sitting or standing. Subjects who are self employed or who work at jobs that permit them to perform at their own pace adapt more readily to their disease.

6. Pharmacological Management: Pharmacological therapy plays an important role in controlling symptoms and slowing disease progression. Non-steroidal anti-inflammatory drugs (NSAIDs) such as Indomethacin, Naproxen, and Diclofenac are considered first-line

agents for reducing pain and stiffness. Analgesics like Paracetamol may be used for mild pain, while opioids are rarely indicated for short-term use. Disease-modifying anti-rheumatic drugs (DMARDs) such as Sulfasalazine and Methotrexate are useful mainly in peripheral joint involvement, with limited role in axial disease. In patients with active disease not responding to NSAIDs, biologic agents such as TNF-α inhibitors and IL-17 inhibitors are used, which help in reducing inflammation, improving function, and preventing structural damage. Newer agents like JAK inhibitors are used in refractory cases. Corticosteroids have a limited role and are mainly used as local intra-articular injections.

7. Surgical Management: Surgical intervention is considered in advanced cases with severe deformity, significant functional disability, or hip joint involvement. Procedures such as total hip replacement and corrective spinal osteotomy help in improving mobility, relieving pain, and restoring function.

8. Management of Extra-Articular Manifestations: Ankylosing spondylitis may involve other systems, requiring multidisciplinary management. Uveitis is treated with topical or systemic corticosteroids, inflammatory bowel disease requires gastroenterological care, psoriasis needs dermatological management, and cardiac involvement requires appropriate cardiological evaluation.

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