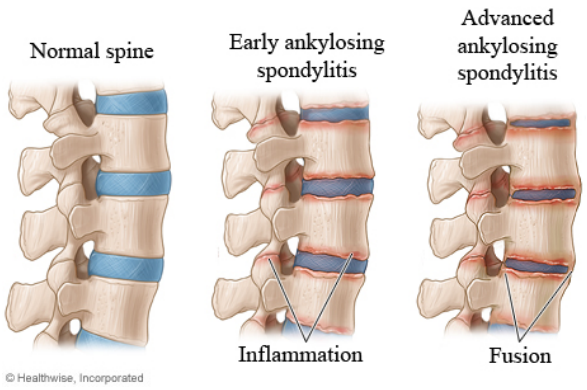
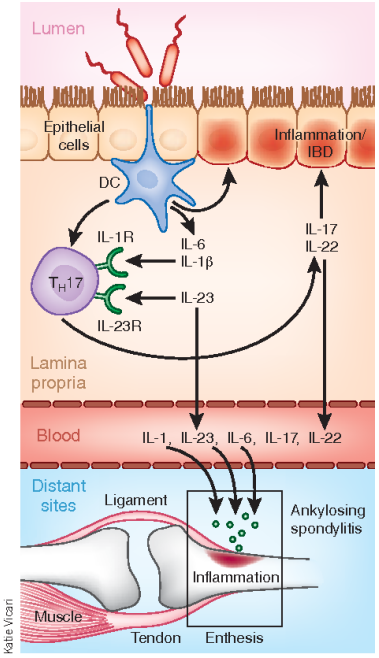
Ankylosing spondylitis (AS) is a rare type of arthritis that causes pain and stiffness in mainly the spine. AS can also affect other areas of the body such as the shoulders, hips, ribs, heels, and small joints of the hands and feet. The soft tissues, joints, tendons, and ligaments are the main sites of inflammation seen in AS. The main symptom of AS is inflammatory spinal pain; with time, some patients develop fibrosis and calcification, resulting in the loss of flexibility and the fusion of the spine.



As an autoimmune disease, AS develops through complex interactions between genetic background and environmental factors. Most individuals who have AS also have a gene that produces a genetic marker, a protein called HLA-B27. This marker is found in more than 95 percent of people in the Caucasian population with AS.[1] It is important to note, however, that one does not have to be HLA-B27 positive to have AS. Also, a majority of people with this marker never develop AS.

Scientists suspect that other genes, along with a triggering environmental factor such as a bacterial infection, for example, are needed to activate AS in susceptible people. The differences observed in immune cells and cytokines in AS suggest the role of immunological effects in AS pathogenesis.[3] HLA-B27 transgenic rats failed to develop features of AS in a germ-free environment, which changed when commensal bacteria were introduced into the germ-free models, suggesting possible interactions between HLA-B27 and the microbiome.[2] One classic hypothesis has been that AS may start when the defenses of the intestines break down and certain bacteria pass into the bloodstream, triggering changes in the immune response.[3]



The coexistence of AS and intestinal inflammation has been known for some time. Around 70% of patients with AS develop subclinical gut inflammation.[4] This altered composition of microorganisms in the gut leads to more bacteria that play a role in inflammation and AS progression. A study done on patients with AS found an increase in the abundance of Lachnospiraceae, Ruminococcaceae, and Prevotellaceae.[5] Altered gut bacteria produce high levels of inflammatory cytokines that when enter into the blood stream can travel to distant sites and attack the soft tissue of the body leading to the progression of AS.

The severity of AS varies greatly from person to person, and not everyone will experience the most serious complications or have spinal fusion. Some may experience only intermittent back pain and discomfort, while others may experience severe pain and stiffness over multiple areas of the body for long periods of time. AS can be debilitating and, in some cases, lead to disability.

Almost all cases of AS are characterized by acute, painful flare ups which are followed by temporary periods of remission when symptoms subside. The hallmark feature of ankylosing spondylitis is the involvement of the sacroiliac (SI) joints during the progression of the disease. The SI joints are located at the base of the spine, where the spine joins the pelvis. The pain typically becomes persistent (chronic) and is felt on both sides, usually lasting for at least three months. Over the course of months or years, the stiffness and pain can spread up the spine and into the neck. Pain and tenderness spreading to the ribs, shoulder blades, hips, thighs, and heels is possible as well.

Wellness Recommendation

The wellness recommendation for AS includes WHITEE Patches, LC Balancer, Brown, and Java. The WHITEE Patch, placed directly over the affected area, helps to decrease inflammation, increase local blood flow in order to enhance nutrient supply, and cellular activity for spine and disc regeneration. Increasing the nutritional supply to the site of degeneration will accelerate the healing mechanism necessary for recovery. Herbs in the WHITEE Patch also help to break down nodules and stasis in those who have more advanced AS in which fibrosis and calcifications are present. The WHITEE Patch also enhances the lymphatic circulation to remove metabolic waste from necrotic tissue such as lactic acid. LC Balancer functions to open the smallest blood vessels to enhance whole body microcirculation and accelerate healing. The enhanced microcirculation also improves nutrient absorption from the digestive tract to assist in healing.

To address the autoimmune factors of AS, Java and Brown are recommended to alternate every two weeks throughout the protocol. Java, a spleen Damp formula, helps to improve lymphatic function. Lymphatic system dysfunction is strongly linked to autoimmune conditions such as AS. Improving lymphatic circulation helps to clear toxins that could be a trigger in AS pathogenesis. Java also helps calm down the immune over reaction and reduce the autoantibody levels. Brown, a liver Yin formula, helps to improve liver function and structure to reduce inflammation and calm down the hyperactive T cells and the resulting autoimmunity.

To address the gut inflammation and infection which can lead to the progression of AS related autoimmunity, the wellness recommendation includes Silver, Probiosis, SJ, Spring Capsule, and Formula B. Silver and Probiosis help remove infection and inflammation from the GI tract. Silver helps to remove the excess gram-negative bacteria such as Prevotellaceae, while Probioisis helps to address the gram-positive bacterial overgrowth of Lachnospiraceae and Ruminococcaceae. Spring Capsule warms the middle jiao (upper GI tract) to improve blood flow to the stomach. SJ repairs degenerated lining in the upper GI tract to restore stomach acidity and improve digestion by nurturing the stomach Yin. Formula B improves the stomach emptying process and increases intestinal contraction by enhancing the Stomach Qi. Patients can experience symptom improvement with reduce pain and stiffness and enhanced flexibility and movement in 1 month. 3 months of the protocol is recommended for significant improvement.

References:

1. Reveille, J. D. The genetic basis of spondyloarthritis. Ann. Rheum. Dis. 70, i44–i50 (2011).
2. Taurog, J. D. et al. The germfree state prevents development of gut and joint inflammatory disease in HLA-B27 transgenic rats. J. Exp. Med. 180, 2359–2364 (1994).
3. Zhu, W., He, X., Cheng, K. et al. Ankylosing spondylitis: etiology, pathogenesis, and treatments. Bone Res 7, 22 (2019). https://doi.org/10.1038/s41413-019-0057-8
4. Thomas GP, Brown MA. Genetics and genomics of ankylosing spondylitis. Immunol Rev. 2010;233(1):162-180. doi:10.1111/j.0105-2896.2009.00852.x
5. Costello ME, Ciccia F, Willner D, et al. Brief Report: Intestinal Dysbiosis in Ankylosing Spondylitis. Arthritis Rheumatol. 2015;67(3):686-691. doi:10.1002/art.38967