PERSPECTIVE

Bacterial infections in lupus

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ABSTRACT

In individuals with systemic lupus erythematosus (lupus), bacterial infections of the lungs, skin, circulation, and other organs are prevalent, and they are often more severe and invasive than similar infections in healthy people. Using both human patient samples and animal models of lupus, a number of research have looked at changes in bacterial abundance in lupus patients, infection rates, and the effect of certain bacterial species on disease development. The purpose of this review is to summarize human and mouse studies that describe changes in the bacterial microbiome in lupus, the role of a leaky gut in stimulating inflammation, the identification of specific bacterial species associated with lupus, and the potential roles of common bacterial infections in promoting lupus progression. The data was gathered by searching the PubMed database for papers about bacterial infections in lupus and alterations in the microbiome linked with the disease. The bacterial microbiome of lupus patients differs significantly from that of control people, and in lupus-prone mice differs significantly from that of control mice, according to the research evaluated. Furthermore, there is evidence that lupus patients and lupus-prone mice have a leaky gut. Live bacteria or bacterial components may enter the blood through a leaky gut, causing inflammation. Invasive bacterial infections are more prevalent in lupus patients, and they are typically more severe. *Staphylococcus aureus, Salmonella enterica, Escherichia coli, Streptococcus pneumoniae*, and mycobacteria are among the microorganisms that cause illnesses. These bacterial infections can cause enhanced immunological activation and inflammation, which can stimulate autoreactive lymphocyte activation and aggravate lupus symptoms.

Key Words: *Lupus; Infection*

INTRODUCTION

SLE (also known as lupus) is a multisystem autoimmune disease marked by significant immunological dysfunction. Self-reactive T and B cells persist in lupus patients and enhance systemic immunological activation as a result of the loss of central and/or peripheral immune tolerance mechanisms. The fundamental causes of lupus are unknown, however genetic and environmental risk factors are suspected. As a result of these genetic and environmental variables, the immunological milieu is drastically altered, with fewer regulatory T cells, more effector T cells, and higher B cell activation. Autoantibodies are produced against a variety of self-antigens, including double-stranded DNA, ribonucleoproteins, connective tissues, and immunoglobulins, as a result of abnormal autoreactive B cell activation in response to self-antigen and the provision of T cell help by autoreactive T cells. Immune complexes generated in tissues can facilitate antibody-dependent immune activation mechanisms, such as complement activation, resulting in inflammation and organ damage. Furthermore, persistent stimulation of innate and adaptive immune cells alters the cytokine milieu, with numerous cytokines being identified at elevated levels. Lupus prevalence varies greatly by geographic location, ethnicity, and sex, with women and people of African origin having the greatest incidence. In comparison to males, women get the condition at a 9:1 ratio. SLE causes inflammation and damage to a range of organs, including the kidneys, lungs, cardiovascular system, and brain, all of which contribute to SLE patients' death. Lupus Nephritis (LN), a kind of glomerulonephritis, is a prevalent symptom of SLE that affects 50-75 (%) of individuals. It can progress to chronic kidney disease and significantly decreased kidney function.

Neuropsychiatric lupus, which is caused by persistent immunological activation in the brain, has been linked to cognitive impairment and an increased risk of stroke. Patients with SLE have been demonstrated to have higher incidences of cardiovascular illness, including atherosclerosis, myocardial infarction, and pericarditis. SLE patients frequently die as a result of these physical symptoms and the subsequent end-stage organ failures. Infection, on the other hand, has been found as a major cause of hospitalization and mortality in studies across time. For example, 27% of 1000 European SLE patients followed prospectively for 5 years had infections, and 29 percent of those who died during the research period perished from infection. The percentage of patients who die from infection has grown as therapy for autoimmune disease has improved, resulting in reduced mortality rates owing to organ damage caused by SLE. For example, according to a Chinese research, from 1986 to 1995, around 25% of SLE patient fatalities were caused by infections, but from 2006 to 2012, almost 50% of deaths were caused by infections. Bacterial, viral, and fungal pathogens can all cause infections in SLE patients. We initially focus on descriptions of the microbiome in lupus patients and mice models of lupus in this review, with an emphasis on mechanisms through which alterations in the microbiome may impact lupus development. Pathogenic bacteria can penetrate tissues and cause illnesses if the microbiome is disrupted. In the second section of the review, we describe the incidence of various bacterial infections in lupus patients, as well as their relationship to morbidity and death.

The large intestine is home to the majority of bacteria that colonize humans. These bacteria normally stay in the gut, but disruption to the lining of the intestine can cause bacteria or their products (such as LPS) to seep into the bloodstream. Bacteria that escape the gut can colonize other organs such as the liver and lymph nodes in the mesenteric region. 'Leaky gut syndrome' refers to the leaking of bacteria or their products from the intestine, which has been linked to a number of human autoimmune disorders, including rheumatoid arthritis, multiple sclerosis, and Type I diabetes.

In the gastrointestinal tract, as well as the skin, eyes, nose, and genitourinary systems, mice and humans have a varied and prolific microbiota. Bacterial species present in these tissues have the ability to both promote and control inflammation (by producing immune activating chemicals like TLR ligands) (by promoting the development of regulatory immune cells). Short chain fatty acids generated from microbes, such as butyrate, has been found to enhance the formation of regulatory T cells. Microbial dysbiosis, or an imbalance in the bacterial microbiota, is frequent in immunological diseases and can result in the proliferation of pathogenic and/or opportunistic microorganisms. It's crucial to remember that the makeup of the gut microbiome varies depending on the age and gender of people examined, therefore microbiome research must account for this. Furthermore, the human and murine gut microbiotas differ significantly, therefore caution should be exercised when interpreting rodent research and their applicability to human lupus.

Enterococcus gallinarum is a bacterium that has been associated to lupus pathogenesis in humans. In mice, *E. gallinarum* has been associated to leaky gut syndrome. Human SLE patients have a comparable leakiness. Biopsies of

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liver tissue from lupus patients revealed the presence of *E. gallinarum*, whereas biopsies of liver tissue from normal donors did not reveal the presence of *E. gallinarum*, though other Enterococcus species were found in four of six normal liver donors using PCR with primers specific to *E. gallinarum* or all Enterococcus species. When *E. gallinarum* was co-cultured with primary hepatocytes, type I interferon was produced, a cytokine that plays a key role in lupus pathogenesis. As a result of the poor intestinal barrier, *E. gallinarum* may be able to exit the gut and colonize the liver, where it can cause inflammation and trigger the release of immune-stimulating cytokines. Ruminococcus members were shown to be overrepresented in SLE patients in two investigations. One of these investigations found a link between the presence of Ruminococcus gnavus and poorer illness in lupus patients, as measured by high SLEDAI scores. Anti-dsDNA antibodies cross react with antigens present in the *R. gnavus* strain RG2, suggesting that the *R. gnavus* may drive anti-DNA autoantibody formation. *R. gnavus* has also been discovered to be abundant in people with Crohn's Disease, an inflammatory bowel disease. This shows that *R. gnavus* may have a special function in inducing inflammatory immunological responses. *R. gnavus*, on the other hand, was shown to be lower in the gut microbiome of lupus patients with active illness compared to those in remission in a separate study. Streptococcus bacteria, particularly *S. anginosus*, were found to be more abundant in lupus patients, especially those with active illness. Streptococcus and *Streptococcus anginosus* levels were similarly favorably connected with higher SLEDAI values and negatively correlated with complement C3 levels. The research discussed above reveal that the bacterial microbiome of lupus patients differs from that of control participants, and these investigations have identified certain particular bacteria species that may play a pathogenic role. However, it is still unclear whether the variations discovered are important for lupus development or are the result of lupus-related organ damage or variances in environmental variables. The involvement of numerous bacterial species in the course of lupus can be addressed mechanistically using mouse research.

CONCLUSION

In this review, we collected existing data on microbial colonization and infection in systemic lupus erythematosus, including characteristics of the bacterial microbiome and bacterial species that are commonly found in lupus patients. The gut microbiota of lupus patients differs from that of control persons, according to several studies. Similarly, in lupus, the oral and cutaneous microbiome appears to be distinct, indicating a widespread dysbiosis. Infections produced by *S. aureus, S. enterica, E. coli,* and *S. pneumoniae*, which are common in lupus patients, are also prevalent in the general population. Lupus patients, on the other hand, are more vulnerable to these pathogens' severe and invasive infections. According to evidence from animal models, bacterial infection with a variety of bacteria can cause enhanced immune activation and accelerate autoimmune development. The increased occurrence of bacterial infections in lupus patients is likely due to both intrinsic immune system weaknesses and immunosuppressive medications. As a result, owing to immune deficiencies induced by immunological abnormalities or immunosuppressive medications, SLE patients may become infected with bacteria, and the bacterial infection drives additional immune activation, exacerbating the autoimmune symptoms. More research is needed to better understand the changes in bacterial colonization and infection in lupus patients, as well as how they affect disease progression.