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Commentary

A Modern Analysis of Immune Alterations Following Surgical Removal of the Spleen

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DESCRIPTION

Surgical removal of the spleen, or splenectomy, remains a important intervention for a range of hematologic, traumatic and immunologic disorders. While often lifesaving, splenectomy significantly reshapes the immune landscape of the body, producing a series of adaptive and maladaptive changes that persist lifelong. The spleen functions as a central immunological organ, facilitating filtration of senescent erythrocytes, clearance of blood-borne pathogens and orchestration of cellular and humoral immune responses.

After splenectomy, the absence of splenic macrophages, marginal zone B cells and reticuloendothelial filtration capacity disrupts normal immunologic equilibrium, predisposing patients to overwhelming infections, altered inflammatory responses and increased susceptibility to encapsulated bacteria such as *Streptococcus pneumoniae*, *Neisseria meningitidis* and *Haemophilus influenzae*. This heightened vulnerability explains why post-splenectomy patients may experience sudden, fulminant episodes of sepsis, a condition that can escalate within hours and carries a substantial risk of mortality if not treated immediately.

The loss of splenic phagocytic function leads to profound alterations in innate immunity. Without splenic macrophages, circulating immune complexes accumulate, microbial clearance becomes delayed and neutrophil activation is dysregulated. Reduced production of opsonizing immunoglobulins and complement components further weakens microbe detection and destruction. Additionally, splenectomy alters leukocyte trafficking, resulting in increased peripheral lymphocyte counts, monocytosis and thrombocytosis. These hematologic changes reflect a compensatory, yet imperfect, systemic response to splenic absence.

From a humoral immunity perspective, the depletion of splenic marginal zone B cells impairs the body's ability to generate rapid IgM-mediated responses against polysaccharide antigens. This defect is especially critical in young patients, whose immune systems depend heavily on splenic maturation pathways. The long-term immune disadvantage following splenectomy has also been linked to impaired vaccine responsiveness, reduced memory cell development and increased inflammatory cytokine expression.

Beyond infection risk, splenectomy induces functional immunologic shifts that influence coagulation, inflammation and autoimmune susceptibility. Elevated circulating platelet levels increase the risk of thrombosis, particularly portal and venous thromboembolism. Additionally, the absence of splenic clearance allows abnormal erythrocytes and platelets to persist in circulation, contributing to microvascular dysfunction and chronic inflammatory stimulation.

Studies have shown that splenectomy alters cytokine profiles, increasing pro-inflammatory mediators such as Interleukin-6 (IL-6) and Tumor Necrosis Factor-alpha (TNF-α), while decreasing anti-inflammatory signaling pathways. These changes may partially explain the increased risk of atherosclerosis and cardiovascular complications observed in long-term splenectomized patients. In autoimmune disorders, the immunologic effects of splenectomy are complex: reduced antigen clearance may exacerbate autoimmune activation in some conditions, while in others, the removal of autoreactive immune cell populations may yield therapeutic benefit. This duality illustrates the intricate immune consequences of splenic loss.

CONCULSION

Modern strategies to manage immune alterations post-splenectomy emphasize proactive preventive care. Vaccination against encapsulated organisms, lifelong infection surveillance, and prompt antibiotic access remain essential components of management. Additionally, thromboprophylaxis and targeted immunologic monitoring are increasingly recommended to counterbalance both infectious and thrombotic risks. Emerging research into splenic tissue transplantation, artificial splenic filtration devices, and immunomodulatory therapy offers new hope for restoring partial immune function in high-risk patients. Overall, the immune alterations that follow splenectomy reflect an intricate interplay between adaptive reconstruction and persistent vulnerability.

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