

Splenectomy and Systemic Immunity: Balancing Therapeutic Benefit and Infection Risk

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DESCRIPTION

Splenectomy, the surgical removal of the spleen, represents a critical therapeutic intervention in specific hematologic, traumatic, and immunologic conditions, yet it simultaneously introduces profound alterations in systemic immunity that require long-term vigilance and management. Traditionally performed to treat hereditary spherocytosis, immune thrombocytopenia, refractory splenic sequestration crises, portal hypertension-related hypersplenism, and traumatic splenic rupture, splenectomy alleviates pathological hemolysis, improves cytopenias, and prevents fatal internal hemorrhage.

The spleen, however, is not simply a hematologic filter but a highly specialized immunologic organ that modulates innate and adaptive defenses through macrophage-mediated clearance, antigen presentation, immunoglobulin synthesis, complement activation, and microbial surveillance. Its red pulp removes senescent erythrocytes and circulating debris, while the white pulp orchestrates antibody production—particularly IgM—and supports opsonization of encapsulated bacterial pathogens. Consequently, splenectomy disrupts this multi-layered immune architecture, creating a state of functional vulnerability wherein patients face elevated lifelong risk of severe bacterial, parasitic, and opportunistic infections.

The hallmark immunologic consequence of splenectomy is diminished host defense against encapsulated organisms such as *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Neisseria meningitidis*. Because encapsulated bacteria evade phagocytosis without effective opsonization, asplenic individuals experience compromised clearance and accelerated microbial proliferation, contributing to a dangerous clinical entity known as Overwhelming Post-Splenectomy Infection (OPSI). Characterized by rapid onset sepsis, disseminated intravascular coagulation, and shock, OPSI carries high mortality, sometimes unfolding within hours of symptom onset.

Beyond bacterial susceptibility, splenectomy contributes to increased parasitemia in malaria and babesiosis, altered viral immune responsiveness, impaired antibody affinity maturation, and dysregulated cytokine signaling. Thrombotic risk also rises

significantly due to postsurgical thrombocytosis, endothelial activation, and changes in blood flow dynamics, potentially leading to deep venous thrombosis, pulmonary embolism, or portal vein thrombosis. Thus, the therapeutic benefit achieved by splenic removal must be counterweighted by enduring systemic immunologic compromise.

Advances in immunologic profiling have highlighted that not all patients exhibit the same degree of vulnerability post-splenectomy; some demonstrate preserved marginal-zone B-cell function, while others show profound deficits in early antibody responses. This understanding has encouraged the use of adjunctive strategies such as monitoring serologic response durability, assessing complement activity, and utilizing rapid-diagnostics pathways that enable early detection of bloodstream pathogens. The emerging emphasis is on dynamic surveillance—allowing clinicians to adjust preventive measures over time—while ensuring that patients remain engaged partners in recognizing infectious risks and seeking prompt medical assessment.

Patient education has become a defining element of long-term care, encouraging immediate medical evaluation for fever, careful travel precautions, mosquito avoidance in endemic regions, the use of medical alert identification, and recognition of early sepsis symptoms. Concurrently, hematologic monitoring helps track thrombotic risk, with antiplatelet or anticoagulant therapy considered in high-risk settings. Surgical alternatives such as partial splenectomy and spleen-preserving trauma protocols now offer options to retain residual immune function, minimizing long-term sequelae while achieving therapeutic benefit.

CONCLUSION

In contemporary hematologic and infectious-disease practice, splenectomy is understood not simply as a procedural solution, but as the beginning of a chronic immunologic condition requiring lifelong partnership between patient and clinician. The goal is not only to resolve the primary indication for surgery, but to anticipate and mitigate downstream vulnerability through structured prevention, guided surveillance, and informed patient autonomy. As research advances, deeper characterization of splenic immunobiology, immunoglobulin dynamics, and host-

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pathogen interactions will refine tailored strategies that improve protection and quality of life for asplenic individuals. In essence, splenectomy exemplifies the medical challenge of balancing therapeutic gain against persistent immunologic cost,

demanding long-term stewardship that extends far beyond the operating room.