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Krivit W. Overwhelming postsplenectomy infection.
Amer. J. Hematol. 2:193-201, 1977.
[University of Minnesota Hospitals, Minneapolis, MN]

This review paper provided data on the clinical background, pathophysiologic process, and potential means for prevention of overwhelming postsplenectomy infection. It provided tables that indicated incidence of IgM deficiency in a postsplenectomy state. [The *SCI*® indicates that this paper has been cited in over 100 publications, making it the most-cited paper ever published in this journal.]

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The rationale for writing the review article of pathophysiology of overwhelming postsplenectomy infection (OPSI) was that scientific colleagues continued to be "doubting Thomases" about the validity and frequency of OPSI. My colleagues in practice required more concise and precise data about the incidence, frequency, and severity of OPSI.

My own personal clinical observations of a 50 percent mortality rate and severe morbidity consequent to splenectomy were quite dramatic. Because of this, I maintained a continued interest in and lectured about OPSI. I received information and personal communications about incontrovertible cases following abdominal trauma. The anecdotal nature of these cases was well recognized by me, but my clinical colleagues still remained unconvinced because the incidence of OPSI was only 1 to 2 percent in some series.

The observation and documentation that the spleen served as an excellent site of bacterial clearance was well known. That removal of the spleen placed the individual at risk because of lack of bacterial clearance was apparently not a sufficient pathophysiologic process to convince my colleagues. What they required was an immunologic understanding of the pathophysiology. Why did OPSI occur?

Mondorf[1] had shown that the IgM levels were statistically quite low in a series of patients who had had their spleens removed because of trauma. This was the first real evidence of an immunologic defect subsequent to splenectomy. I then reviewed all the printed series. I observed that in the splenectomy groups the IgM was always lower than appropriate controls done at that institution.

The compilation of data became more and more intriguing. Serial observations of opsonic activity by Giebink and colleagues indicated that splenectomy for trauma impaired seroconversion of pneumococcal vaccine.[2] This data gave greater weight to the belief that the postsplenectomy state was an abnormal immunologic state.

Trauma to the spleen secondary to automobile and motorcycle accidents had been considered a sine qua non for doing a simple splenectomy in past years. Surgeons faced with a potentially damaged spleen would immediately consider and complete a splenectomy. However, subsequent to finding immunologic deficit in patients who have had splenectomy because of trauma, surgeons began to utilize newer techniques. Appropriate care now includes an attempt at reconstructive surgical repair to maintain an intact hilus. For maintaining normal immunologic capacity and normal reticuloendothelial clearance, this approach is far superior to simple splenectomy.

Much less effective is the use of "splenosis." This consists of dumping spleen tissue into the abdominal cavity and allowing regrowth of splenic nodules. There is only a slight degree of improvement of immunologic defect following "splenosis" over that subsequent to total splenectomy.

Surgical repair with retention and preservation of splenic hilus has also been compared to autotransplant implantation of a portion of splenic tissue in an abdominal cul-de-sac. The data would indicate that the former (repaired spleen) has a better capacity for the immunologic and reticuloendothelial clearance than the latter (implanted spleen).

Now, the dictum of removal of a spleen whenever there is damage or trauma to it is no longer tenable. Repair of the spleen is best for preservation of function, autotransplant is next best, and "splenosis" is only slightly better than total splenectomy.[3]

Finally, in a recent letter to the editor, impairment of leukocytes and macrophages following splenectomy was suggested.[4] This observation needs to be confirmed in other laboratories.

1. **Mondorf W, Lennert K A & Kollmar M.** Immunological globulin synthesis in the human spleen. (Lennert K & Harms D, eds.) *Die Milz: Struktur. Funktion. Pathologie. Klinik. Therapie.* New York: Springer-Verlag, 1970. p. 162-5.
2. **Giebink G S, Le T C & Schiffman G.** Decline of serum antibody in splenectomized children after vaccination with pneumococcal capsular polysaccharides. *J. Pediatrics* 105:576-82, 1984.
3. **Smith C & Giebink S.** Personal communication.
4. **Simon M, Jr., Djawari D & Hohenberger W.** Impairment of polymorphonuclear leukocyte and macrophage functions in splenectomized patients. (Letter to the editor.) *N. Engl. J. Med.* 313:1092, 1985.

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