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Sudden Death and Sepsis After Splenectomy

There have been a number of case reports dealing with pneumococcal infections after splenectomy [*1,2*]. Early reports emphasized that fatal infections occurred in patients with a basic underlying serious disease process that involved primarily the reticuloendothelial system such as in diffuse cancer or lymphoma, histiocytosis, sickle cell anemia, and extensive hemosiderosis [*3*]. It was generally believed that splenectomy done for trauma imparted no greater risk to infection than a simple appendectomy [*4*]. Since that time, follow-up studies have shown that asplenic patients are at risk from overwhelming sepsis regardless of the reason for the removal of the spleen [*5*].

It is the purpose of this report to state the occurrence of pneumococcal sepsis in the asplenic patient as a cause of sudden death and to present the autopsy findings on two deaths that we investigated in Seattle, Washington. In both cases we were not given a clinical workup for clues before the autopsy diagnosis. Both deaths came under our jurisdiction as sudden deaths in healthy, young adults who died following a short, nonspecific, flu-like illness. Both deaths were viewed as either drug-related or the result of criminal violence. One death occurred at a time when the swine influenza mania was at its peak.

Case 1

The first death investigated was that of a 19-year-old black female student attending the University of Washington who had been in good health all of her life. The only significant history was that of a ruptured spleen following an automobile accident approximately three years before her death. An uneventful splenectomy was performed and she had remained in good health. On the evening of her death, she had told her father that she did not feel well and complained of nausea and other nonspecific symptoms. When she was last seen alive she was vomiting in the bathroom of the dormitory where she lived. Twelve hours later the victim's parents became concerned, called the resident manager, and requested that he check on their daughter's welfare. When the manager opened the door he found the victim sprawled across the bed in her nightgown. This death was viewed as suspicious and the father was convinced that she had been murdered.

Autopsy examination demonstrated a large abdominal wall scar. The spleen was absent but multiple splenic deposits were present in the omentum and in the pelvic serosa (Fig. 1). Multiple serosal petechial hemorrhages were present along with bilateral adrenal hemorrhages and hemorrhages in other organs. The lungs were free of pneumonia and no focus of infection was found. Autolysis was disproportionate with the time-of-death interval. Bacterial stains of splenic tissue and adrenal glands demonstrated abundant pneumococcal organisms.

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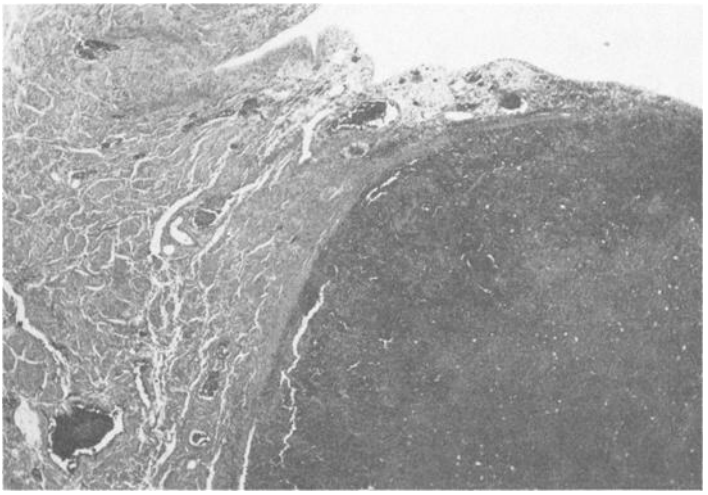

FIG. 1—*Photomicrograph of splenic implants on serosal surfaces (hematoxylin and eosin stain).*

Case 2

The second death was that of a 21-year-old white male cook who four years before his death had sustained a ruptured spleen in a traffic accident. This was treated by splenectomy with an uneventful recovery. Since that time his general health had been good. On the day of his death and while at work, he complained of chest and abdominal pains and began vomiting. He returned home and his symptoms worsened over the next 10 h. He was transported to the hospital where he survived 30 min and died. Clinically he was febrile (39.6°C or 103.2°F) and had spasticity of the face and neck muscles and intense cyanosis of the face.

Autopsy demonstrated a large abdominal wall scar and absence of a spleen. Autolysis of tissues was disproportionate for the postmortem interval. Splenic implants throughout the serosa of the peritoneal cavity were noted. Hemorrhagic adrenals and hemorrhagic pulmonary edema were the only other significant findings. Blood culture was positive for *Streptococcus pneumoniae* and bacterial stains of organs demonstrated many clusters of pneumococcal bacteria (Fig. 2). Pneumonia was absent and no focus of infection was uncovered.

Discussion

Infection after splenectomy is characterized by an abrupt onset with the patient becoming severely ill in a short time. The sudden onset and rapidity of the illness can suggest a food, drug, or chemical poisoning. *Streptococcus pneumoniae* is the bacterial agent most frequently responsible although other encapsulated bacteria are occasionally encountered. The portal of entry is apparently the nasopharynx, similar to sepsis of meningococcemia. There is an unusual exuberance of bacterial growth, and the bacteremia can be so intense that organisms have been seen in peripheral blood with an estimated concentration of 10^6/ml of blood [*6*].

Although the gross autopsy findings in our two cases can be considered nonspecific, there were observations worthy of comment. These include the degree of autolysis in all organs out of proportion with the postmortem interval. Both victims showed considerable

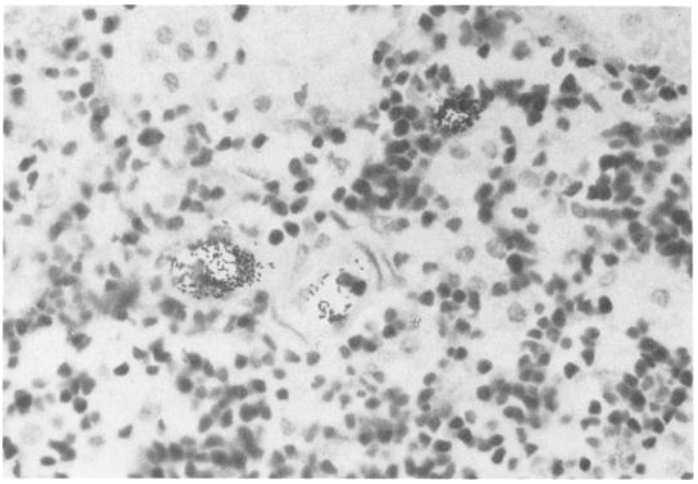

FIG. 2—*Photomicrographs of adrenal gland with pneumococcal bacteria in sinuses and vessels (Gram stain).*

autolytic change of body tissues as compared to tissue ordinarily seen in deaths from traumatic or noninfectious disease. The febrile nature of sepsis and the disseminated bacteremia appear to be the responsible mechanism for this accelerated autolysis. In one death, diffuse hemorrhages and petechiae of the serosa were present, and hemorrhages were so exaggerated in organs such as the larynx and epiglottis that an asphyxial death was suggested. However, the diffuse distribution of hemorrhage included the serosa of both the thorax and abdomen. Hemorrhages were also present in the adrenal glands. Although disseminated intravascular coagulation appears to account for such hemorrhages and has been infrequently reported [*7*], we were not able to find fibrin thrombi to make a definitive diagnosis. Both victims showed splenic implants of various sizes, some of which were hemorrhagic. The splenic tissue present showed its usual microscopic lymphoid appearance although no clearly defined blood supply could be demonstrated. The features present were those of architecturally recognizable splenic pulp without a definable central blood supply.

Sepsis after splenectomy was initially reported in children who had undergone splenectomy for hematologic disorders [*4*]. The first reports of sepsis were in patients less than four years of age, and sepsis occurred within two to three years of splenectomy. An underlying immunological defect was implicated as a cause for the sepsis. Later, with the ensuing use of splenectomy for diagnostic management of lymphoma, sepsis was seen to occur with increased frequency in this group of patients. Again the basic lymphoid disease process was cited as the reason for the increased incidence of sepsis. However, recent reports have shown sepsis occurring in the asplenic trauma victim at a rate 60-fold greater [*8*] than expected. Hence, all asplenic individuals are at risk for sepsis at any age and at any interval after the removal of the spleen.

Animal experiments and observations in humans indicate that at least two factors are important in the splenic ability to protect against infection: the volume of splenic tissue and the presence of normal blood supply. In animals the implantation of splenic tissue in asplenic rats does not confer the same protection against infection as normal animals have, but it does provide a degree of protection as compared to asplenic animals [*9*]. The two cases in this report showed residual deposits of splenic tissue, microscopically normal,

but clearly not enough tissue was present to provide protection from bacteremia. It may be an oversimplification to view the major protective role of the spleen simply as a mechanical filter, but certainly that appears to be a major function. How much splenic tissue and what relationships to the systemic circulation are necessary to provide protection remain undiscovered.

The spleen is the site of the production of antibodies to intravenously injected foreign particulates in the absence of preexisting antibodies. In the presence of antibodies from prior exposure to similar antigens, the function of other parts of the reticuloendothelial system obscures the contribution of the spleen [*10*]. The spleen may be the site of production of a leukophilic immunoglobulin that increases the phagocytic activity of leukocytes. Likewise, complement abnormalities produced by splenectomy suggest that a deficiency of complement activation may account for impaired host defense. Complement promotes an early nonspecific mechanism of defense against infection by opsonization and clearance of bacteria [*11*]. In subjects whose spleens have been removed complement activation abnormalities exist and may contribute to impaired host defense.

The protection that the spleen provides in host defense—both as a mechanical filter and as an immunological organ promoting antibody protection—has caused surgeons to reevaluate their surgical approach to the spleen in trauma and hematologic disorders. The clinical risk of the patient after splenectomy is well recognized, and pathologists investigating sudden death should view the scar on the abdomen as perhaps the first clue to the diagnosis of the fatal condition.

Summary

This paper has reported two deaths occurring in young adults who had undergone splenectomy for trauma several years before developing fatal pneumococcal sepsis. Tissues at autopsy demonstrated a disproportionate autolysis for the postmortem interval. One victim also showed diffuse serosal hemorrhages, presumably as a result of disseminated intravascular coagulation. Both showed evidence of residual splenic implants but such implants clearly did not provide protection against sepsis. The mechanisms whereby the spleen protects from sepsis appear to be that of a mechanical filter and an immunological organ producing antibody or antibody-like substance. How much splenic tissue and what relationships to the systemic circulation are necessary to provide protection remain undiscovered.

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