TEACHING EDITORIAL

Sepsis in Children Following Splenectomy

For several years there has been a growing realization in the medical community that splenectomy in children, for whatever reason, may be followed by fulminating sepsis and rapid death. These overwhelming infections may occur within days of surgery or be delayed up to 15 years, although sepsis usually occurs within 2–3 years following splenectomy. The postsplenectomy infections are characterized by an abrupt onset, florid bacteremia, high incidence of disseminated intravascular coagulation, and death within hours of the onset of symptoms in as many as 80% of septic cases (*1*). This condition usually occurs in children less than 4 yr old, but may happen in young adults who have had a splenectomy when they were over 18 yr old. The greatest risk for postsplenectomy sepsis, however, is in the first 2 years of life, and accordingly postponement of the splenectomy until age 5, if clinically possible, has been suggested (*2*). Children with underlying diseases requiring splenectomy generally have a higher mortality rate than those in whom splenectomy is necessitated by trauma, although figures may vary from series to series. Claret et al. (*2*) report a 36% mortality rate from infection in children who had a previous reticuloendothelial disorder requiring splenectomy, compared with 6.4% in those who did not. Hancock et al. (*3*) report fatal bacteremia in three out of 17 children with Hodgkin's disease who had splenectomy performed at a staging laparotomy. Belfanz et al. (*1*) report a 66% mortality rate in 12 septic children who had splenectomy for traumatic splenic rupture. *Diplococcus pneumoniae* is the most frequent organism causing sepsis (in about 45% of cases), although a variety of other infective agents have been implicated, including *Streptococcus* (up to 15% of cases), *Hemophilus influenzae, Niesseria meningitidis, Escherichia coli, Pseudomonas aeruginosa, Staphylococcus aureus, herpes zoster virus,* and even the protozoan disease, babesiosis. Penicillin prophylaxis is now advocated for all splenectomized children, regardless of the reason for splenectomy (*1,2,4,*), for periods varying from 18 mo (*2*) to 3 yr (*4*).

The primary cause of sepsis following splenectomy has been attributed to a reduction in IgM, resulting in a high susceptibility to infection. An antigenic stimulus is believed to evoke initially a macroglobulin response (IgM), to be followed later by IgA and IgG responses. Claret et al. (*2*) demonstrated significantly higher IgA levels, and correspondingly lower IgM levels, in children who had splenectomies, compared with nonsplenectomized control groups. Hancock et al. (*3*) reported a significant rise in neutrophil count following splenectomy, but only a marginal rise in lymphocytes. In their patients neutrophil phagocytic function was normal or enhanced, and there was no significant change following splenectomy. This loss of humoral response to infection has been proposed as one of several possible explanations of sepsis in children with sickle cell anemia who end up with functional asplenia (*5*).

In the light of such information, many surgeons are not doing complete splenectomies in children with ruptured spleens, but instead are performing either partial splenic resections or are suturing the laceration (*1*). Several groups are also questioning the wisdom of routine splenectomy at the time of staging laparotomy for Hodgkin's disease (*3,6*).

Spleen scanning is the simplest means of determining splenic integrity. Subcapsular hematomas, lacerations, and fractures are seen as variably sized splenic defects or actual separation of splenic components. In the past, children with such scintigraphic defects often had splenectomies. As Fischer and his associates point out in this issue of *The Journal of Nuclear Medicine,* there is an overall 0.25–0.58% mortality rate from sepsis in the postsplenectomy patient group. In those children who have had a splenectomy and are unfortunate enough to become septic, the mortality rate may approach 80%, especially if an underlying disease necessitated the surgery rather than abdominal trauma.

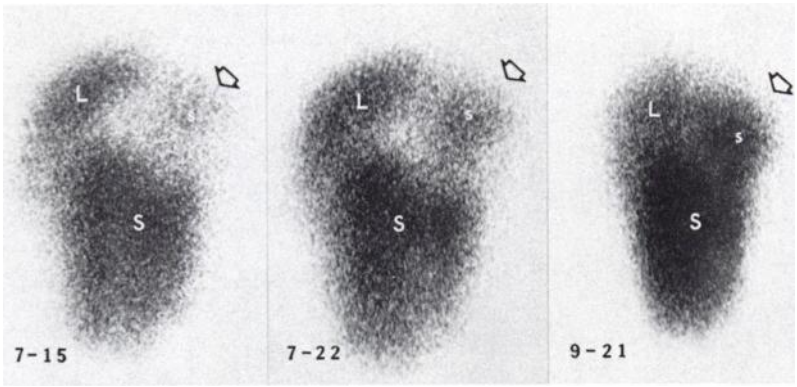

FIG. 1. Serial left lateral scintigraphic views (dates indicated) of a 12-year-old girl with spleen fractured by trauma. Tracer was Tc-99m sulfur colloid. Upper pole of spleen (arrow, small S) was separated from main splenic body (large S) by hematoma that was shown by ultrasonography. Surgery was not performed, and by 2 mo much of the splenic defect had resolved. (L = left lobe of liver.)

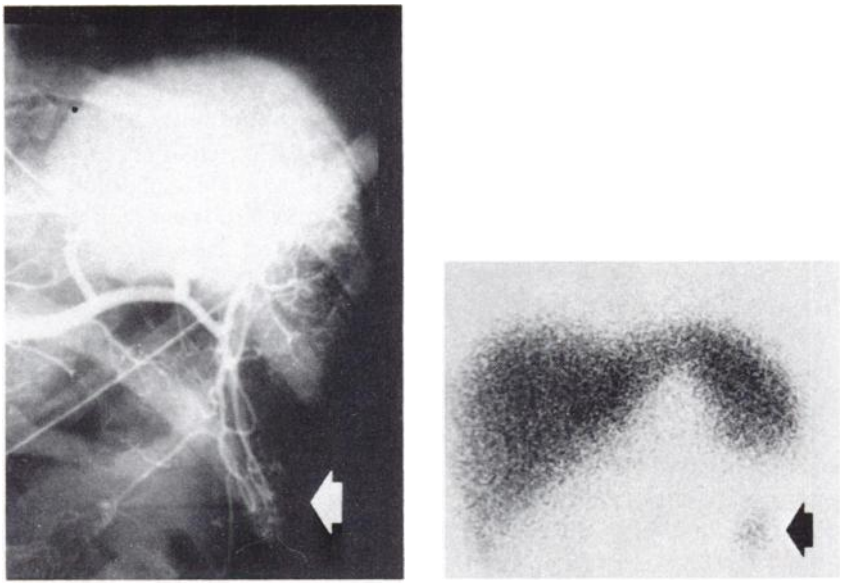

FIG. 2. (A) Pre-operative angiogram (courtesy of Dr. John Miller) of a 15-year-old girl who had gross intra-abdominal hemorrhage following trauma. Upper, main portion of spleen is separated from intact lower pole tip (arrow) by large avascular region. (B) Tc-99m sulfur colloid scintigraphy following surgery and resection of necrotic hemorrhagic mid-splenic portion. Persistent viability of upper and lower (arrow) portions is shown. The two splenic fragments were not sutured together, so the separation does not necessarily indicate recurrent hematoma.

Fischer and his associates have contributed several important pieces of information. First, they have informed the nuclear medicine community of the problems associated with splenectomy in children, since it is obvious that the decision to remove a spleen surgically has far greater implications than has been suspected. Second, their serial scans have shown residual splenic "defects" in nonoperated children persisting for up to 12 mo in patients who were asymptomatic. Eight of these patients had a gradual reduction in the size of the scan abnormality, whereas in two others the defect was unchanged after the 2-mo scan. Three of 13 patients showed disappearance of the splenic defects by 3 mo. The authors speculate that the persistent defects most likely represent scar formation within the spleen, which sounds reasonable. This information will encourage physicians to "wait and watch" rather than to operate on the basis of a persistent scintigraphic defect.

We also have been performing serial scans in patients with splenic trauma who have not had surgery (Fig. 1). As time passes, the splenic defects gradually lessen and the patients are symptom free. If surgery is necessary because of significant intra-abdominal hemorrhage, partial splenectomy may be adequate, with as much splenic tissue as possible left behind (Fig. 2). A splenic pseudocyst or enlarging hematoma must be suspected if serial scanning (using the multiple-projection technique advocated by the authors) shows a defect that is increasing in size. However, as pointed out, a stable sized defect is not necessarily a grave prognostic sign, and the nuclear medicine physician must carry this information to his surgical colleagues.

The question yet to be answered is how much splenic tissue can be removed and still leave enough behind to provide adequate defense against infection. Balfanz et al. (*1*) reported an 8-year-old child who had a splenectomy for trauma and later died of sepsis. At autopsy, however, a small accessory spleen was present. Obviously there must be a minimal amount of splenic tissue necessary to guard against

sepsis. Once this is determined, surgeons will have a better guideline in circumstances when splenectomy (a partial one, preferably) is unavoidable.

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