Elective Subtotal Splenectomy

Indications and Results in 33 Patients

PHILIP C. GUZZETTA, M.D., E. JERRY RULEY, M.D., HENRI F. W. MERRICK, M.D., CAROL VERDERESE, R.N., and NORMAN BARTON, M.D.

Elective subtotal splenectomy was performed in 33 patients (30 children and 3 adults) between 1981 and 1989. Indications for the procedure were (1) prevention of azathioprine-induced neutropenia (n = 20); (2) Type I Gaucher disease (n = 9); and (3) cholesteryl ester storage disease, chronic myelogenous leukemia, thalassemia major, and splenic cyst in one patient each. There were no operative deaths, no reoperations for bleeding, and 30 of 33 (91%) patients had a functioning splenic remnant documented by a postoperative radionuclide spleen scan. One patient developed neutropenia without evidence of viral infection that required temporary cessation of azathioprine and the patient with thalassemia major had only transient improvement in transfusion requirements. All other patients (94%) had control of the underlying condition for which the operation was performed. We conclude that subtotal splenectomy is a safe, effective therapy for a variety of nontraumatic conditions.

MANAGEMENT OF HYPERSPLENISM that is unrelated to primary hepatic disease traditionally has been treated by total splenectomy. The specter of postsplenectomy sepsis, particularly in children,[1-4] has made retention of splenic function desirable whenever possible. Although several series of partial splenectomies for trauma have been reported, this is the first large collection of elective partial splenectomy patients. The intent in almost all these patients was to control hypersplenism. Therefore more than 80% of the spleen was resected, hence the term subtotal rather than partial splenectomy. The techniques we used for subtotal splenectomy (STSx) were based on those developed for the management of traumatic splenic laceration.[5] This review assesses elective subtotal splenctomy in 33 patients during the last 8 years, with an emphasis on the long-term effects on these patients.

Correspondence and reprint requests to Philip C. Guzzetta, M.D., Department of Surgery, Children's Hospital National Medical Center, 111 Michigan Ave., NW, Washington, D.C. 20010.

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From the Departments of Surgery and Nephrology, Children's Hospital National Medical Center, the Department of Child Health and Development, George Washington University, Washington, D.C., and the Developmental and Metabolic Neurology Branch, IRP, NINCDS, NIH, Bethesda, Maryland

Materials and Methods

Thirty-three patients underwent STSx. They can be divided into three groups: (1) patients awaiting renal transplantation who were at risk for developing azathioprine-induced neutropenia; (2) patients with Type I (adult, non-neuronopathic) Gaucher disease; and (3) other diseases requiring splenic resection.

Azathioprine-Induced Neutropenia

The patients determined to be at risk for azathioprine-induced neutropenia were between 9 months and 21 years of age. These 20 patients were awaiting renal transplantation and all had the procedure before our routine use of cyclosporine in 1984. One patient previously had lost a living related donor kidney to rejection when azathioprine was stopped because of refractory neutropenia.

Patients were considered to be at risk to develop neutropenia while on azathioprine if they demonstrated the following signs while on dialysis awaiting renal transplantation: (1) chronic neutropenia, white cell count less than 5000; (2) mild neutropenia, white cell count of 5000 to 6000, with other signs of hypersplenism, *i.e.*, thrombocytopenia or excessive transfusion requirements; or (3) mild neutropenia with an abnormal cortisol stimulation test.[6]

Gaucher Disease

Nine patients had STSx for type I Gaucher disease. Gaucher disease is an autosomal recessive genetic disorder

caused by a deficiency of the enzyme glucocerebrosidase. The result is the accumulation of glucocerebroside predominantly in the reticuloendothelial system and the bone marrow.[7] Massive splenomegaly with hypersplenism is a common complication of type I Gaucher disease (Fig. 1). Type I is the most common of the three types of Gaucher disease. Type I is non-neuronopathic, usually diagnosed in adulthood, and generally the patients enjoy a normal life span. Type I patients diagnosed as children commonly have more severe disease than those diagnosed as adults. Children often suffer growth retardation, poor nutritional status, and frequent bone infarction crises. Six of our nine patients were 20 years old or younger.

Subtotal splenectomy was offered only to those who would have been considered for total splenectomy in the past. Indications for surgery were: (1) severe hypersplenism as evidenced by white cell counts less than 4000, hemoglobin level less than 9 g, or platelet count less than 50,000; or (2) severe symptoms due to the massive splenomegaly, such as orthopnea, or recurrent pain due to splenic infarcts. Recurrent spontaneous epistaxis or gastrointestinal bleeding was a major complaint in seven of the nine patients.

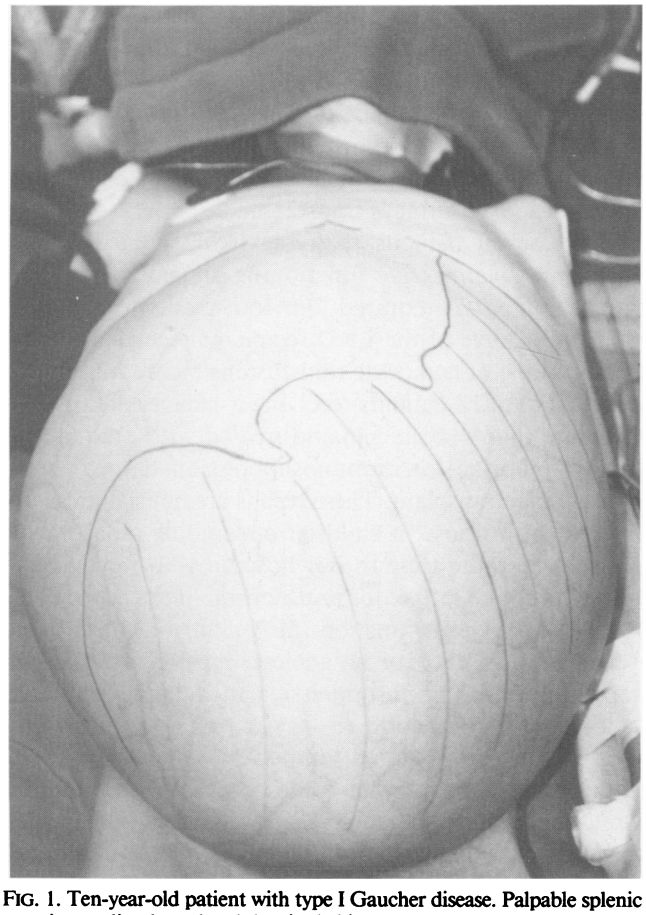

FIG. 1. Ten-year-old patient with type I Gaucher disease. Palpable splenic margins outlined on the abdominal skin.

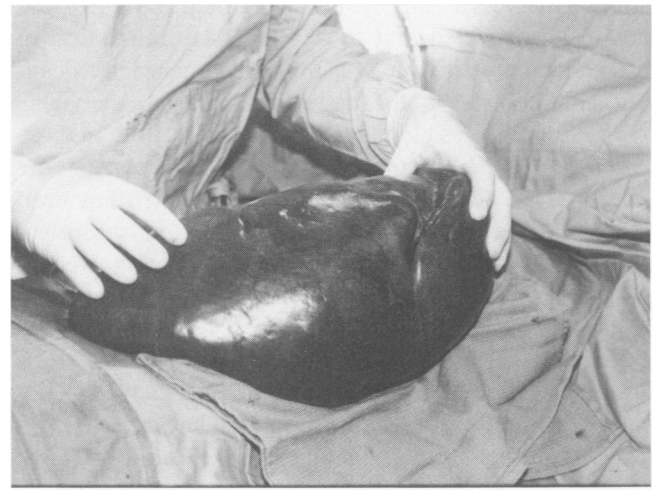

FIG. 2. Twenty-five-year-old patient with type I Gaucher disease. Intraoperative photograph showing mobilization of splenic attachments before ligation of splenic vascular branches.

Other Diseases Requiring Splenic Resection

Four children had STSx for the following diagnoses: (1) cholesteryl ester storage disease resulting in severe hypersplenism and splenic infarcts; (2) chronic myelogenous leukemia with severe splenic infarct pain unresponsive to conventional therapy with chemotherapy or radiation; (3) thalassemia major with increasing transfusion requirements and; (4) post-traumatic splenic cyst with chronic left upper-quadrant pain.

Surgical Technique

All patients received pneumococcal vaccine before surgery and were given perioperative antibiotics. The objective in all patients, with the exception of the one with a splenic cyst, was removal of 80% to 90% of the functioning splenic mass. The size of the splenic remnant retained varied depending on the size of the patient, the size of the spleen, and the underlying disease process. In the patients with massive splenomegaly, which includes all patients with Gaucher disease, the remaining splenic remnant was approximately the size of a man's fist. In this group 90% of each spleen was removed.

The technique for STSx has been described elsewhere.[8] Briefly, the spleen is dissected free from all attachments except the splenic artery and vein and is brought through the incision onto the anterior abdominal wall (Fig. 2). The vessels usually divide into three main branches before entering the splenic parenchyma: a moderate-sized superior branch that supplies 20% to 25% of the spleen, a large central branch that supplies 60% to 70% of the spleen, and a small inferior branch that supplies 10% to 20% of the splenic mass. This vascular arrangement makes it easiest, in most instances, to preserve the inferior portion of the spleen on its vascular pedicle.

Once the vessels supplying the segment to be resected have been dissected and ligated (Fig. 3), transection of the splenic parenchyma is accomplished using electocautery. Digital occlusion of the vessels supplying the viable remnant minimizes blood loss during transection. In those patients with platelet counts less than 50,000, platelets are administered after the segment to be resected has been devascularized and while the spleen is being transected. The dose of platelets is one pack per 5 kg of body weight, with a maximum of 10 platelet packs in patients weighing more than 50 kg.

The raw splenic surface is compressed with mattress sutures of chromic catgut (0 chromic in massive splenomegaly and 00 chromic suture in smaller spleens) on a straight needle. Suture ligation with chromic of large transected branches on the raw surface may be necessary. Microfibrillar collagen (Avitene, Alcon Surgical, Ft. Worth, TX) and a viable omental patch are placed over the raw surface. It is important to fix the splenic remnant to the left diaphragm with two chromic sutures, to prevent torsion, taking care to orient the splenic vessels properly. No drains are used. All patients were maintained on ampicillin, or an alternate antibiotic if they were allergic to penicillins, for several months after surgery. The length of time on prophylactic antibiotics varied according to patient age and diagnosis.

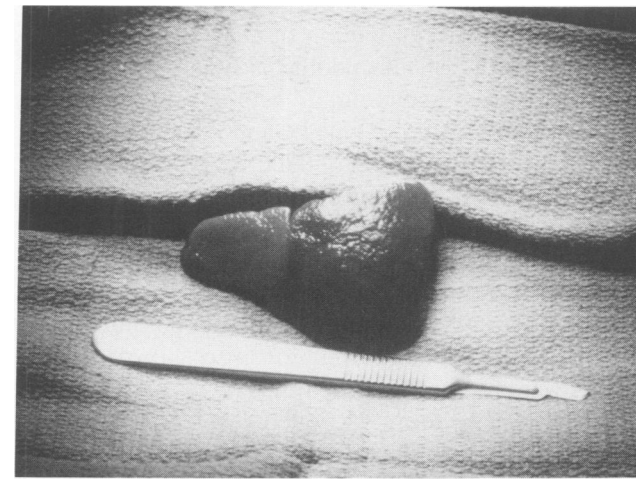

FIG. 3. Nine-month-old patient with renal failure at risk for azathioprine-induced neutropenia. Intraoperative photograph showing line of demarcation between viable (left) and nonviable splenic segments after ligation of the blood supply to the superior 80% of the spleen.

Results

There were no operative deaths and no patients required reoperation for bleeding or for infarction of the splenic remnant.

Azathioprine-Induced Neutropenia (Table 1)

Nineteen of the 20 patients had a functioning splenic remnant documented by a postoperative spleen scan. The one patient without a functioning remnant was operated on early in our experience and the remnant was not sutured to the left diaphragm. A functioning remnant was seen on spleen scan six days after operation but the patient developed left upper-quadrant pain and a left pleural effusion two days later and subsequently no remnant could be detected by scan. It is presumed that the remnant underwent torsion and infarction. Fixation of the splenic remnant to the left diaphragm became a routine part of the procedure after this complication occurred.

Seven patients had the STSx while undergoing another intra-abdominal procedure, five with bilateral nephrectomies, and two with cholecystectomies for cholelithiasis. Thirteen patients had STSx without an associated procedure. There were four complications. One splenic remnant infarct associated with fever and left pleural effusion, as mentioned above, resolved spontaneously. Two small-bowel obstructions occurred in patients having had bilateral nephrectomies at the same time as STSx; both required laparotomy and lysis of adhesions. One patient developed an incisional hernia that was repaired after kidney transplantation.

Nineteen of the 20 patients have undergone renal transplantation. Seventeen (85%) patients are alive and eight (47%) of those alive have functioning kidney allografts. All patients, except one, are more than 4 years beyond the date of their transplant. Six of the eight patients with functioning kidneys had living related donors and two had cadaver kidneys transplanted.

Six of seven patients receiving living related donor (LRD) transplants have functioning allografts, but one patient (patient 2) required four transplants to obtain a good result. Five of the LRD recipients received donor-specific blood or leukocyte transfusions before transplantation. Two of 12 patients receiving a cadaver donor kidney more than 4 years ago and treated with prednisone and azathioprine for immunosuppression have a functioning renal transplant. These results are not significantly different from those in children undergoing renal transplant at the same time in our hospital who were determined to not be at risk for azathioprine-induced neutropenia. The allograft function rate might well have been greater; however three of our adolescent patients purposely stopped their immunosuppressive medication and lost their kidneys to rejection.

Three patients required temporary cessation of their azathioprine after transplantation because of neutropenia (white cell count less than 4000). Two of the patients had documented cytomegalovirus (CMV) infection at the time of neutropenia and after recovery from the CMV illness they were returned to their preillness dose of azathioprine.

TABLE 1. *Subtotal Splenectomy Patients with Azathioprine-Induced Neutropenia*

Patient	Age (Years)	Concurrent Procedure	Viable Remnant	Kidney Donor	Complication	Follow-up		
						Neutropenia	Sepsis	Current Status
1	20	None	Yes	Cadaver	None	No	No	On dialysis; kidney Tx rejected at 1 year
2	9	None	Yes	Cadaver X2 LRD X2	None	No	No	Functioning transplant; on cyclosporine at 1 year. Previous Txs lost to rejection
3	7	Cholecystectomy	Yes	Cadaver	None	No	No	On dialysis; kidney Tx rejected at 3 years
4	11	None	Yes	Cadaver	None	No	No	On dialysis; kidney Tx rejected at 2 years
5	14	None	Yes	Cadaver	None	Yes—with CMV infection	No	On dialysis; kidney Tx rejected at 4 years
6	0.75	None	Yes	Cadaver X2	None	No	No	Died; Primary nonfunction of both Txs, died of renal failure
7	14	None	Yes	Cadaver	None	No	No	Died; Liver failure due to chronic active hepatitis, nonfunction of Tx
8	12	None	Yes	Cadaver	None	No	No	On dialysis; lost graft after 2 years due to medication noncompliance
9	16	None	Yes	None	None	No	No	On dialysis
10	15	None	Yes	LRD	None	No	No	Functioning transplant—5 years
11	13	None	Yes	LRD	None	No	No	On dialysis; lost graft after 4 years due to medication noncompliance
12	13	Bilateral nephrectomy	Yes	Cadaver	Small-bowel obstruction	No	No	On dialysis; lost graft after 2 years due to medication noncompliance
13	12	Bilateral nephrectomy	No	LRD	Infarcted remnant & L pleural effusion	No	No	Functioning transplant—6 years
14	3	Cholecystectomy	Yes	Cadaver	Incisional hernia	No	No	Died; good graft function for 2 years, died of hypertensive encephalopathy
15	12	Bilateral nephrectomy	Yes	Cadaver	None	Yes—with CMV infection	No	On dialysis; good graft function for 2 years, lost graft to chronic rejection
16	11	None	Yes	LRD	None	Yes—2 episodes	No	Functioning transplant—6 years
17	17	Bilateral nephrectomy	Yes	Cadaver	Small-bowel obstruction	No	No	Functioning transplant—4 years
18	16	None	Yes	LRD	None	No	No	Functioning transplant—6 years
19	21	None	Yes	LRD	None	No	No	Functioning transplant—7 years
20	2	Bilateral nephrectomy	Yes	Cadaver X2	None	No	No	Functioning transplant—5 years 1st Tx lost to renal artery stenosis

Tx, transplant.

CMV, cytomegalovirus.

LRD, living related donor.

One patient had two episodes of neutropenia without evidence of CMV infection that required a temporary decrease in the dosage of azathioprine. One patient was transplanted early in our cyclosporine experience and one was never transplanted, thus neither received azathioprine. Of the 18 patients at risk for azathioprine-induced neutropenia, one (6%) developed neutropenia without evidence of viral infection.

Gaucher Disease (Table 2)

Eight of nine patients have a functioning splenic remnant documented by postoperative spleen scan. It is noteworthy that two of the eight patients with splenic function did not show radionuclide uptake within the first postoperative week, although subsequently splenic remnant uptake was demonstrated at 3 and 6 months after surgery. Pitted red cell counts, another measure of splenic function, were performed after operation in eight patients and were in the normal range (less than 1%) in seven patients. The one patient with a nonfunctioning remnant had a pitted red cell count of 0.8% and is likely to have some residual splenic function that is not yet detectable by scan. One patient with a functioning remnant with areas of infarction had a count of 8.6%. All patients had dramatic improvement of hypersplenism and the symptoms related to splenomegaly.

A small pleural effusion developed in four patients and was associated with a temperature elevation of 38.5 C to 39 C. These effusions resolved without thoracentesis in each case and did not delay discharge. One patient developed abdominal wall cellulitis that required IV antibiotic therapy for 1 week and remained hospitalized for 12 days. This was the only patient in this group hospitalized for more than eight days.

In the first year after STSx, four children had bone pain episodes consistent with bone infarct crises. Three of these patients had had similar symptoms before surgery, but the frequency and severity of the episodes increased after the operation. One patient had an episode of bilateral tibial pain after minor trauma, one patient had a single episode of polyarticular arthritis, and another had two episodes of gout after surgery.

The first patient with STSx for Gaucher disease had the procedure 5 years ago. The splenic remnant has approximately doubled in size (Figs. 4A and B), but the patient's hypersplenism remains controlled. In all patients the remnant has increased in size but much less so in the adults than in the children. No patients have shown signs of hepatic dysfunction due to increased glucocerebroside deposition in the liver. Eight of the nine patients, including the one without splenic function, considered themselves much improved after surgery.

These patients have massively enlarged spleens that usually comprise more than 10% of their body weight compared to the normal spleen-to-body weight ratio of 0.3%. Although it is difficult to quantify the relief that these patients experience once the STSx has been done, it is clear that improved muscle mass, appetite, and energy level consistently develop after surgery. In patients with growth potential, two of four had a significant growth spurt within 1 year of STSx.

Other Diseases Requiring Splenic Resection (Table 3)

In the last and less specific category, three of four patients have a functioning splenic remnant. The patient with chronic myelogenous leukemia and multiple splenic infarcts had no evidence of splenic function after surgery and died of recurrent leukemia 1 month after surgery. The patient with cholesteryl ester storage disease had significant improvement of her hypersplenism but continues to suffer from severe liver disease. The patient with thalassemia major had temporary improvement of his transfusion requirements, but within 1 year had returned to his preoperative transfusion needs. The patient with the post-traumatic splenic cyst has a viable splenic remnant and is asymptomatic.

Discussion

It is well known that the spleen has both hematologic and immunologic functions. With the exception of the fetus and certain disease processes, the human spleen does little to generate new blood cells. The primary hematologic function of the spleen is to act as a sophisticated filter to delete from the circulation those erythrocytes, leukocytes, and platelets that are seen as flawed. The hypersplenic state is due to excessive sequestration of the blood cells resulting in cytopenias in the peripheral circulation. Hypersplenism is usually associated with splenomegaly.[9] The immunologic function of the spleen is complex but, on a very basic level, is designed to clear encapsulated bacteria and parasites, generate a cellular response to infection, and initiate antibody formation. The spleen is also very active in the nonself antigen-recognition pathways activated by allograft transplantation.

Splenectomy was commonly used as a nonspecific immunosuppressive technique in patients awaiting renal transplantation in the 1960s and 1970s. Reports of unimproved allograft function and definite increased mortality rate from infection, particularly in children, in those having had splenectomy[10–13] have eliminated the procedure from the pretransplantation preparation of these patients.

Hypersplenism occurs in about 10% of hemodialysis patients.[14] The cause of azathioprine-induced neutropenia after renal transplantation is probably a combination of bone marrow suppression and increased splenic seques-

TABLE 2. *Subtotal Splenectomy Patients with Gaucher Disease*

Patient	Age (Years)	Hgb (gms) Pre	Hgb (gms) Post*	WBC ×10³ Pre	WBC ×10³ Post*	Platelets ×10³ Pre	Platelets ×10³ Post*	Viable Remnant	Complication	Follow-up: Pitted RBC's %†	Follow-up: Bone infarcts	Follow-up: Solenic Remnant	Follow-up: Current Status
21	7	9.1	11.0	3.9	4.8	47	142	Yes	Left pleural effusion	0.2	Yes—multiple	Doubled in size in 4.5 years	Significant problems with bone infarcts, no hypersplenism. Developed nephrotic syndrome; 5 years postop.
22	10	7.0	10.1	3.7	14.1	123	279	No—1 month Yes—3 months	Left pleural effusion	8.6	Yes—multiple	No change in size in 4 years Infarcts present	No hypersplenism, many orthopedic problems; 4 years postop.
23	20	7.8	11.1	1.8	6.6	35	149	Yes	Left pleural effusion	0.2	1 episode of bilateral tibial pain following trauma	33% larger at 6 months, slightly larger at 3 years	Excellent; 4 years postop.
24	48	6.0	12.1	2.7	5.5	29	123	Yes	None	0.3	1 episode of polyarticular arthritis	33% larger at 6 months, no increase at 3 years	Excellent; 4 years postop.
25	12	8.0	10.5	2.1	11.1	41	285	No—1 month Yes—6 months	Left pleural effusion	0.6	Yes—multiple	33% larger at 2 years then at 6 months Infarcts present	Less problems with bone infarcts after 1st year. Improved activity; 2 years postop.
26	38	8.0	10.2	2.9	8.2	62	151	Yes	None	0.2	No—2 episodes of great toe gout	33% larger at 1 year than at 1 month	Excellent; 2 years postop.
27	20	9.0	13.2	1.6	12.5	24	339	No—1 month No—2 years	Abdominal Wall Cellulitis	0.8	No	Not visible on scan	Excellent; 2 years postop.
28	17	9.3	11.5	2.2	6.5	52	199	Yes	None	0.2	Yes—1 episode of femur pain	Doubled in size in 1 year	Excellent; 2 years postop.
29	25	8.8	10.7	1.9	9.2	38	129	Yes	None	—	No	Only 1 scan done	Excellent; 3 months postop.

RBC, red blood cells.
* At the time of most recent visit (see current status).
† Normal pitted RBC % is less than 1%.

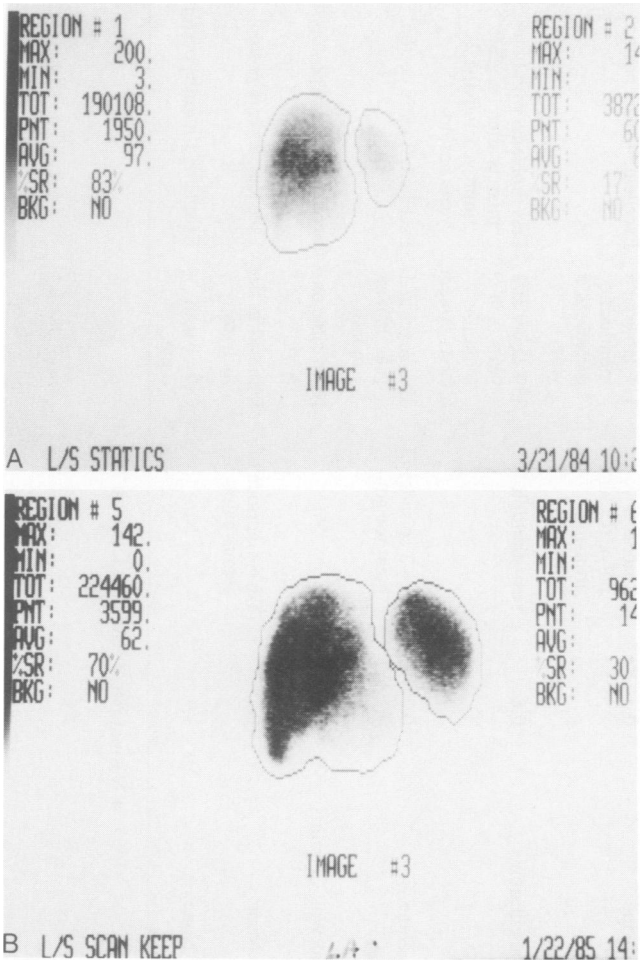

FIG. 4. Seven-year-old patient with type I Gaucher disease liver spleen scan at (A) 4 months and (B) 14 months after STSx, which demonstrates the increased size of the splenic remnant during that time.

tration of the neutrophils. It has been established that total splenectomy lessens the incidence of azathioprine-induced neutropenia.[15] Before 1981 approximately one third of our pediatric renal transplant patients had temporary cessation or reduction of their azathioprine dosage for neutropenia after transplantation. In 1981, because of this problem, we investigated partial splenic ablation by percutaneous embolization as described by Moses et al.[16] The development of postsplenic embolization syndrome,[16] which is similar to acute pancreatitis, in two of our five patients and the recurrence of the neutropenia in the two patients in whom partial splenic embolization initially appeared to be successful, encouraged us to seek a surgical solution to the neutropenia.[17]

The long-term outcome of the 20 patients undergoing STSx before renal transplant is not significantly different from those patients having a transplant without STSx during the same time period. It is impossible to say whether we improved the STSx group's allograft function by the procedure because we do not have a control group at risk for azathioprine-induced neutropenia whose members did not have STSx between 1981 and 1984. It is clear that the incidence of neutropenia without concurrent CMV infection (1 of 18 patients at risk [6%]) is substantially lower than rate we saw before 1981; however this low incidence of azathioprine-induced neutropenia apparently did not result in improved allograft survival rate.

It is encouraging that there have been no episodes of sepsis in the transplant patients with a 5-to-8-year follow-up, but the population may be too small or the follow-up period may be too short to safely conclude that they are not at increased risk for sepsis after STSx.

Hypersplenism due to Type I Gaucher disease has been treated traditionally by total splenectomy.[18] The first report of partial splenectomy for Gaucher disease appeared in 1980[19] and subsequent reports with short-term results have also appeared.[8,20,21] Partial splenic embolization has been used to treat hypersplenism due to Gaucher disease;[22] however we see no benefit to that technique over STSx because it has little effect on the splenic mass, which is the source of many patient complaints.

Our experience with nine Gaucher patients, eight of

TABLE 3. *Subtotal Splenectomy Patients with Other Diseases Requiring Splenic Resection*

Patient	Age (Years)	Diagnosis	Viable Remnant	Outcome
30	3	Chronic myelogenous leukemia	No	Tolerated subtotal splenectomy well. Died 1 month after discharge from leukemia relapse
31	11	Thalassemia major	Yes	Lessened transfusion requirements for 1 year, but thereafter returned to preoperative requirements
32	10	Cholesteryl ester storage disease	Yes	Hypersplenism improved: WBC Preop. 2200 Postop. 4100; Hgb 8.9 11.4; Platelets 74,000 156,000. 2 years postop liver disease has progressed
33	14	Posttraumatic splenic cyst	Yes	Excellent; 6 years postop.

whom are alive more than 1 year after surgery, has given us certain insights that have not been previously emphasized. Although a review of 239 Gaucher disease patients concluded that splenectomy does not increase the risk of bone infarction,[23] it appears that children in the first decade of life with Type I Gaucher disease are particularly susceptible to bone infarcts after total splenectomy[24] and we have found a similar high incidence of bone infarcts in our children after STSx. This problem can be debilitating in some patients, particularly if the vertebrae are involved. Indications for STSx in the young child must be stringent because of the potential for these bone crises. Efforts to identify patients at risk for this problem before surgery are ongoing.

We believe that there is a specific salutary time period in which to perform STSx in patients with Type I Gaucher disease. The mere presence of a large spleen is not an indication for surgery; however prolonged delay may result in multiple painful splenic infarcts that make partial splenic preservation more difficult, and recurrent spontaneous hemorrhages requiring transfusions. In children there may be improvement in growth after STSx, which must also be taken into consideration. The optimal time for surgery in both adults and children is when symptoms increase in frequency and severity and before large segments of the spleen become infarcted.

The question of the ultimate fate of the splenic remnant in the Gaucher disease patient remains unanswered. Clearly at 5 years there is significant regrowth of the remnant, but the hypersplenism remains in check. Again there seems to be a difference between children and adults, with the remnant increasing in size more quickly in the children than in the adults. Adults followed for more than 3 years have shown only a 30% increase in remnant size. It is conceivable that another STSx may be necessary in some of these patients in the future. Management of Gaucher disease patients, particularly children, must take into consideration that the STSx is only palliative treatment for hypersplenism and any procedure must not interfere with future treatments such as enzyme replacement or bone marrow transplantation[25] directed at the underlying enzyme deficiency.

Partial splenectomy is the procedure of choice for a splenic cyst, regardless of whether the cyst is of congenital, traumatic, or infectious origin.[26–28] The child with cholesteryl ester storage disease[29] was similar to the patients with Gaucher disease in indications for surgery; however she also had severe cirrhosis and portal hypertension. Although we would not recommend routine STSx for hypersplenism due to portal hypertension, the patient clearly shows that the procedure can be performed safely under those circumstances. Successful management of hypersplenism due to portal hypertension by partial splenic embolization has been reported.[30]

The patient with chronic myelogenous leukemia had a very unsatisfactory course. Not only did he fail to show a viable splenic remnant, he died of leukemia relapse 1 month after surgery. He was considered a candidate for surgery only after chemotherapy and radiation to the spleen were unsuccessful in managing constant, severe splenic pain. Results with total splenectomy in this form of leukemia are also poor because of the limited life expectancy for these patients.[31]

Thalassemia major has been treated by total splenectomy when transfusion requirements become excessive. Our patient was an 11-year old who was receiving two to three units of blood every 2 weeks. After STSx he required three units of blood every 2 months, but within 1 year he needed his preoperative transfusion requirements. His parents refused further surgical therapy. We removed approximately 80% of the spleen and we believe that a greater amount of spleen should be resected for this disease. Partial splenic embolization has been used in thalassemia major.[32]

The technique we used for STSx has remained essentially unchanged during the 8-year period. Others have advocated the use of the laser[33] or the ultrasonic dissector[34] for splenic transection. The raw splenic surface has been covered with fibrin glue[35] or a mesh of absorbable suture.[36,37] We believe these are acceptable alternatives but not necessarily improvements on a technique that has proved completely safe and effective in our hands.

From experimental studies it is clear that maintenance of some sepsis protection after STSx requires retention of the splenic artery and vein as the blood supply to the remnant,[38,39] and at least 25% of the original normal-sized spleen.[40,41] We tried to resect 80% or more of the spleen because that amount is necessary to control the hypersplenic state. In those patients with massive splenomegaly due to an infiltrative process, it is difficult to estimate how much spleen must be left for sepsis protection because the spleen is so distorted and the remnant is not composed entirely of normal spleen. We have chosen to leave a remnant no larger than 8 cm × 8 cm × 5 cm, even when the spleen occupies the majority of the abdominal cavity. To date we have seen no episodes of sepsis in these 33 patients, 27 of whom are alive 4 or more years after their STSx.

The safety and effectiveness of STSx has been conclusively demonstrated for a variety of nontraumatic conditions by this series of patients. In addition careful patient selection and attention to surgical technique, particularly in those patients with massive splenomegaly, offers excellent and long-lasting control of hypersplenism in a variety of nontraumatic conditions.

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