Splenosis: The Autotransplantation of Splenic Tissue Following Injury to the Spleen *

Report of Two Cases and Review of the Literature

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Introduction

THE AUTOPLASTIC transplantation of splenic tissue following traumatic rupture of the spleen in man is termed "splenosis" by Buchbinder and Lipkoff.[4] Only 34 cases are included in world reports since 1896, the year Albrecht [1] published what is probably the first recorded case in the human. Two cases reported here will bring the total to 36. True incidence cannot be ascertained because of the necessity to re-examine the abdominal cavity of previously splenectomized patients, either during a later operative procedure, or at postmortem examination. This condition, except in rare instances, has been asymptomatic, noted only as an incidental finding at operation or autopsy.

The purposes of this article are to (1) report two additional cases of splenosis, (2) present the first instance of intestinal obstruction directly due to a solitary splenic transplant, (3) review the literature on autoplastic splenic transplants in man, and (4) discuss briefly some experimental aspects of splenic autografts.

Albrecht,[1] in 1896, reported that on autopsy of a 25-year-old subject with nephritis, there were approximately 400 small splenic nodules scattered over the peritoneum from both diaphragms to the pouch of Douglas. He considered these accessory spleens which originated from a shattering of the splenic anlage during the embryologic period. Schilling [36] reported, in 1907, similar accessory splenic tissue on the greater omentum, serosal surface of the gallbladder, and descending colon. Although neither Albrecht nor Schilling reported trauma in their subjects, the principal spleen in each case was smaller than normal, scarred, distorted in shape, and adherent to surrounding structures.

Küttner,[20] in 1910, reported a patient who had splenectomy for traumatic rupture of the spleen four years earlier and, at autopsy, splenosis was found. This was the first case of traumatic splenic transplantation. In the following year, Faltin [10] reported a young patient in whom splenic nodules were found over the large and small bowel during operation for appendicitis. Although the patient had splenectomy for traumatic rupture six years prior to the appendectomy, Faltin believed that the etiology of the scattered splenic tissue was not autotransplantation, but stimulation of dormant embryonic rests of splenic tissue by splenectomy.

Von Stubenrauch [45, 46] accepted Küttner's case as traumatic autotransplantation but explained his own by his "Splenoid" theory. He believed that the peritoneum forms new organs, "Splenoids," when the normal constituents of the reticuloendothelial sys-

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tem fail to compensate for the loss of the main spleen.

Experimental Studies

Splenic tissue is readily transplanted into the peritoneal cavity of dogs,[45] rabbits,[34] mice and rats.[6] Splenic pulp emboli, transmitted via the splenic vein, implant in the liver and remain viable.[45] Subcutaneous splenic transplants in rabbits and albino rats develop into fully differentiated and functionally active splenic tissue.[23, 24, 30, 31, 32, 40] Growth rate and survival time of these transplants is increased in young and in splenectomized animals.[24] In nonsplenectomized rats, however, regeneration of splenic transplants is nevertheless complete within three weeks.[30] It is apparently the reticulum cell of the adult spleen which retains the potentiality for differentiation into the various splenic elements.[6, 30]

Specific studies indicate the functional ability of splenic transplants. Rats which develop Bartonella muris anemia following splenectomy are protected in 50 per cent of instances if subcutaneous splenic transplants are performed seven weeks prior to splenectomy.[31] Such transplants also reduce the severity of Trypanosoma lewisi infection in splenectomized rats.[32] Phagocytosis of particulate matter and red blood cells, reservoir ability of the sinusoids, fragmentation of white blood cells, and other splenic activity, can be seen microscopically when splenic tissue is transplanted into transparent chambers installed in rabbits' ears.[50]

An attempt to revascularize the heart in dogs by transplanting splenic tissue into the pericardial cavity has been reported.[12] Further studies may provide an acceptable surgical procedure for the treatment of myocardial ischemia.

Case Reports

Case 1. On April 7, 1945, L. P., a 12-year-old white male, was admitted to the surgical service of Charity Hospital of Louisiana at New Orleans. Approximately 4 hours prior to admission, he fell 15′ from a ladder. Unconsciousness resulted, but he revived quickly. Thirty minutes later, he had left-sided abdominal pain radiating to the left scapular area, which increased in intensity until the time of admission. The only significant fact in the history was that when he was 7-years-old he was found to have an enlarged spleen.

Physical examination revealed abdominal rigidity, generalized tenderness, most severe in the left upper quadrant, distention, and absence of bowel sounds. Laparotomy was performed, and there was a large laceration of the spleen with a hemoperitoneum of over 1,000 cc. Splenectomy was performed.

On June 6, 1950, approximately 5 years later, the patient was readmitted to Charity Hospital. He had a 2 day history of colicky epigastric pain, increasing in severity, and associated with nausea and vomiting during the 8 hours prior to admission. He had a similar episode 1 year earlier which subsided spontaneously. On physical examination, there was tenderness over the upper part of the left-sided splenectomy scar, hyperperistalsis, and slight abdominal distention. Abdominal x-ray films revealed a mechanical small bowel obstruction in the left upper quadrant. He was operated upon and found to have a volvulus of the jejunum in the upper one-third, secondary to adhesions. Lysis of these adhesions easily relieved the obstruction.

Over the greater omentum, small intestines, and the mesentery of the small bowel, were numerous nodules, resembling splenic tissue, varying in size from "freckles to as large as the end of the thumb." The mesentery containing this tissue was biopsied. The patient did well postoperatively and when last seen, in 1955, he had no intra-abdominal complaints.

Pathology Report: Specimen received for examination consists of small portions of tissue, dark red in color, resembling splenic tissue.

Microscopic Examination (Fig. 1): Tissue is identified as being of splenic origin by virtue of its possessing a capsule, Malphighian corpuscles, trabeculae, and red pulp. The capsule, lacking the normal smooth muscle and elastic elements, is composed of dense fibrous connective tissue, and is invaded by many thin-walled blood vessels at the periphery. No hilum is identified. The red pulp appears normal but the Malphighian corpuscles are decreased in number. The trabeculae are poorly developed and also appear numerically reduced.

Diagnosis: Autoplastic splenic transplant resulting from traumatic rupture of the spleen.

Case 2. On November 8, 1955, W. W., 18-year-old white male, was admitted to the Newell Hospital, Chattanooga, Tennessee. One hour ear-

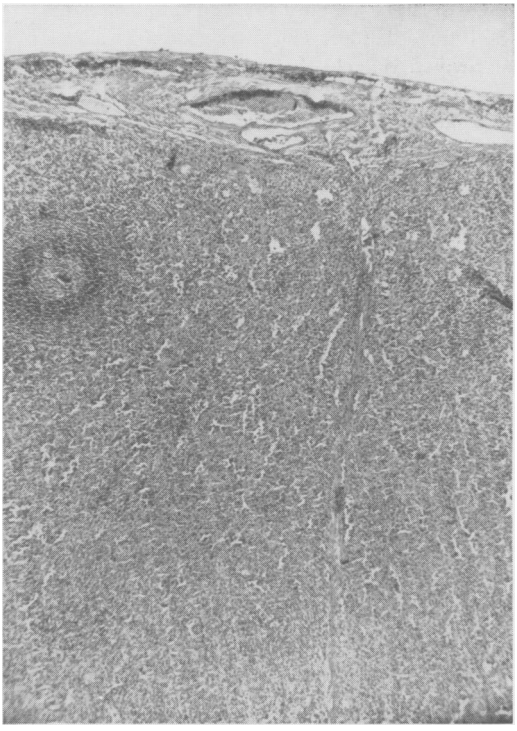

FIG. 1. Representative microscopic field from Case No. 1 showing capsule at upper margin and other features described in text.

lier, he was struck on the posterior portion of his left rib cage while playing football. Pertinent findings on admission were tenderness and splinting of the left side of the chest, flank, and abdomen, and some pain in the left shoulder. Vital signs were normal. X-ray and laboratory studies were normal except for many red blood cells in the urine.

During the following 8 days, the patient improved except that he continued to have pain in the left chest and left shoulder region. Blood pressure, pulse, and hemogram remained stable. The hematuria subsided slowly and intravenous pyelogram revealed no extravasation or injury to the left kidney.

On November 16, 1955, the patient suddenly developed mild shock with marked tenderness in the left upper quadrant and pain radiating to the back and left shoulder. Laparotomy was immediately performed. The peritoneal cavity contained old and fresh blood. Two lacerations of the spleen were found and splenectomy was performed. The postoperative course was complicated by a transient serosanguinous pleural effusion. The patient was discharged from the hospital 2 weeks following operation.

On January 1, 1958, at 9:00 p.m., 2 years and 1 month later, this patient was admitted to Touro Infirmary at New Orleans with a 4 hour history of nausea, vomiting, abdominal cramps, and loose stools. Physical examination revealed only extreme anxiety with hyperventilation. No abdominal tenderness or distention was present. The diagnosis of acute gastroenteritis was made and therapy instituted. Over the next 24 hours, the patient became progressively worse. Nausea and vomiting persisted, and mild abdominal distention with tenderness over the lower part of the splenectomy scar developed. Bowel sounds were increased in pitch and intensity, especially in the left upper quadrant of the abdomen. X-ray films of the abdomen clearly demonstrated mechanical small bowel obstruction. White blood count was 22,950 with 86% neutrophiles and 14% lymphocytes.

Twenty-four hours after admission, the patient underwent emergency laparotomy. At operation, the diagnosis of intestinal obstruction was verified and was due to a firm adherent band, attached at one end to the serosal surface of the distal jejunum and, at the other end, to the left lateral peritoneal wall. On the serosa of the jejunum, forming the base of the obstructing band, was a small tumefaction, oval in shape, approximately 1 cm. in diameter, and dark red in color. The adhesion was lysed and the nodule excised for histologic examination. No other nodules were found.

The patient had an excellent postoperative course and has remained well since operation.

Pathology Report: Specimen labeled mass from wall of jejunum consists of a circumscribed, oval-shaped portion of tissue. The tissue is soft, dark red and measures 1 cm × 8 mm.

Microscopic Examination (Fig. 2): Sections of the tissue identify it as splenic tissue. It has a capsule, Malphighian corpuscles, trabeculae, and a red pulp. The capsule is abnormal due to the presence of irregular masses of dense thick collagenous fibrous connective tissue resembling scars. Smooth muscle and elastic fibers are absent in these areas. In some areas, the capsule is replaced by masses of telangiectatic thin-walled blood vessels that could represent a source of blood supply to this ectopic splenic tissue.

The white pulp or Malphighian corpuscles appear numerically normal. However, the small central arteries and small arteries of the surrounding red pulp are absent.

The red pulp has normal cellularity. The sinusoids are few in number and largely collapsed. The trabeculae are less numerous and less vascular than in normal spleen. The center of the red pulp contains a Gamma-Gandy body with coarse hemo-

FIG. 2. Section from microscopic slides of Case No. 2 showing area of capsule in upper left margin of field containing blood vessels and gamma-gandy body in lower portion.

siderin pigment surrounded by fibrous connective tissue.

In view of the atypical histology noted, especially in the capsule, and the history of previous splenectomy for traumatic injury to the spleen, one is justified in considering this splenic tissue as representing an autogenous transplant rather than a congenital accessory spleen. The location of the tissue situated on the serosa of the jejunum also supports this diagnosis as it would be very unusual to have an accessory spleen in this location.

Diagnosis: Autogenous transplantation of splenic tissue following traumatic injury to spleen.

Clinical Splenosis

Among the 36 reported cases of splenosis, there is no single instance of this condition being diagnosed prior to visualization of the tissue. In some cases, the possibility of transplanted splenic tissue was not considered before histologic examination of the autopsy or surgical specimen was reported. Splenosis, then, has no characteristic clinical picture. Jolly [17] attributed vague intra-abdominal complaints to the presence of the transplanted splenic tissue on the greater omentum. Most authors, however, have denied that splenosis produces signs and symptoms. Those who believe that abdominal discomfort can be produced by this condition, offer as supportive evidence, the gross and microscopic hemorrhage, infarction, hemosiderosis, and other changes in the splenic transplants. Further regarding symptomotology, reference should be made to Gill's case,[13] in which a subcutaneous splenic transplant enlarged and became tender in response to a malarial attack, and to Stobie's[43] which may have represented a recurrence of congenital hemolytic icterus due to splenosis.

Nine of the 36 cases of splenosis reported have been diagnosed at operation for intestinal obstruction. Case report number two in this paper, represents the only known case of intestinal obstruction directly due to the splenic transplant. In the remaining eight cases of obstruction associated with splenosis, postoperative adhesions were responsible for the mechanial ileus. In Table 1 are presented other circumstances under which the diagnosis of splenosis has been made.

Twenty-five cases of splenosis have occurred in males, ten in females, and in one report the sex is not stated. The condition has been diagnosed most commonly in the second and third decades of life. Of the 36

total cases, two-thirds have involved patients between the ages of 12 and 30 years. The youngest patient in whom the diagnosis has been made was age eight, and the oldest, age 56 (Table 2).

In 29 cases, the patient had previous splenectomy for traumatic rupture of the spleen (Table 3). Shaw and Shafi [39] reported nodules on the parietal peritoneum confined to the area of the surgical scar, suggesting that splenic tissue was grafted into the incision site during delivery of the ruptured spleen. In one case in which the transplantation was due to a gun-shot wound,[13] splenectomy was not performed until months after the subcutaneous splenic implant was first palpated. Two cases involve patients who had splenectomy for nontraumatic disease of the spleen.[7, 43] Though in the remaining four of the 36 cases the spleen had not been removed, there was evidence in three that previous splenic trauma had occurred. The time interval between splenic rupture or injury and establishment of the diagnosis of splenosis varied from six months [19] to 30 years.[25] Approximately one-half of the cases, however, have been discovered within five years following trauma, and two-thirds within ten years (Table 4).

TABLE 1. *Occasion for the Diagnosis of Splenosis in the 36 Reported Cases*

Occasion	Number of Cases
1. Operation for intestinal obstruction	9
2. Autopsy	8
3. Appendectomy	5
4. Exploratory laparotomy	5
5. Incisional hernia repair	2
6. Subcutaneous mass excision	2
7. Inguinal hernia repair	1
8. Hysterectomy for leiomyomata	1
9. Cesarean section	1
10. Exploratory thoracotomy	1
11. Not known	1
Total	36

TABLE 2. *Frequency of Diagnosis of Splenosis in the Various Age Groups*

Age Group	Number of Cases
0–10 years	1
11–20 years	13
21–30 years	11
31–40 years	2
41–50 years	4
51–60 years	2
Not known	3
Total	36

With the meager number of cases reported in the literature, it is not possible to assign to splenosis any characteristic clinical picture. Careful follow up studies of patients in whom splenosis was discovered at operation would do much to define this clinical syndrome as the majority of these patients had only biopsy of the splenic nodules, with the greater part of the transplanted tissue left within the abdomen.

Pathology of Splenosis and Comparison with Accessory Spleens

Table 5 compares true accessory spleens with splenic autotransplants.

Splenosis: Splenic implantation most often occurs, in order of frequency, at the following sites: (1) small intestines, (2) greater omentum, (3) parietal peritoneum, (4) large intestines, (5) mesentery of the bowel. The autografts may also involve the diaphragm, pleura, liver, stomach, gall bladder, appendix, kidneys, ureters, lesser omentum, uterus, ribs, urinary bladder and fallopian tubes. In two cases, splenic transplants have been found in the subcutaneous tissue,[7, 13] and in one case, in lung.[40] In most cases, a large number of transplants closely resembles normal splenic tissue. The nodules may vary in color, however, from pink to dark red to greenish black. Two immediately adjacent implants may so differ that one simulates a mass of normal splenic tissue, while the other appears as a small avas-

cular, fibrotic remnant of a splenic nodule. A feature of splenic transplants is the absence of inflammatory reaction in the adjacent tissue.

Peritoneal splenosis has been mistaken initially for endometriosis, metastatic carcinoma, sarcoma, and angiomata of the bowel wall.

Microscopically, the implant may have all of the constituents of normal spleen, or only the red pulp to identify the tissue as splenic in origin. As in Shaw and Shafi's case,[39] in which miliary tubercles were found in the splenic autografts, there will be occasional microscopic evidence of function in the transplants.

Accessory Spleen: The true accessory spleen arises during the embryological period of development and resembles, in miniature, the structure of the principal spleen. Its microscopic anatomy is that of normal splenic tissue. Arising, as does the main spleen, from the left side of the dorsal mesogastrium, accessory spleens are limited to the following areas: (1) hilus of the principal spleen, (2) splenic pedicle, (3) gastro-splenic ligament, (4) retroperitoneal region about the tail of the pancreas, (5) greater omentum, (6) splenocolic ligament, (7) mesentery of the large and small bowel, and (8) left adnexa in the female and left scrotum in the male.[24, 47] Over 75 per cent of accessory spleens are found in the immediate vicinity of the splenic hilus and pedicle.

TABLE 3. *History of Injury to the Spleen in the 36 Reported Cases of Splenosis*

History	Number of Cases
1. Splenectomy following traumatic rupture of the spleen	29
2. Splenectomy for purpura hemorrhagica	1
3. Splenectomy for congenital hemolytic icterus	1
4. Gun-shot wound	1
5. No splenectomy*	*4
Total	36

* Definite evidence of splenic trauma in three of the four cases.

TABLE 4. *Interval Between Splenic Injury and the Diagnosis of Splenosis*

Interval	Number of Cases
6 mos. to 5 yrs.	18
6 yrs. to 10 yrs.	5
11 yrs. to 15 yrs.	2
16 yrs. to 20 yrs.	1
21 yrs. to 25 yrs.	1
26 yrs. to 30 yrs.	2
Not known	7
Total	36

Usually, the number of accessory spleens in a patient is limited to one or two. Curtis and Movitz [8] found that of 56 patients possessing accessory spleens, 26 had only one and 13 only two of the ectopic organs. 85 per cent of patients had accessory spleens in only one location. In no instance were more than two sites involved and in all cases of double location, the hilus constituted one site.

The recurrence of thrombocytopenic purpura and congenital hemolytic anemia due to accessory spleens is well known. These ectopic organs may also be the site of inflammatory lesions, such as malaria, and neoplasms, especially of the lymphoblastoma type.

Settle,[38] in 1940, reported mechanical small intestinal obstruction secondary to adhesions produced by an accessory spleen which had twisted upon its pedicle. The organ, located on the gastrosplenic ligament, had undergone strangulation, hemorrhage, and necrosis. This is not unlike the splenosis induced obstruction reported in this present publication.

Summary

1. Two additional cases of splenosis, the autoplastic transplantation of splenic tissue in man, are presented bringing the total number of recorded cases to 36.

TABLE 5. *The Comparison of Normal Accessory Spleens with the Splenic Autotransplants of Splenosis*

	Accessory Spleens	Splenic Autotransplants
1. History	Not significant	History of trauma to the spleen
2. Location	Determined by the embryological development from the left side of the dorsal mesogastrium	May be widespread throughout peritoneal cavity, in lung, rib, and subcutaneous tissue
3. Number	Usually only one or two—rarely as many as eight or ten	Usually numerous—not infrequently over one hundred
4. Size	Average size larger than transplants. Two to five cm. or greater	Small; pin-point to two cm. in diameter generally
5. Pedicle	Present	Rare
6. Hilum	Present	Absent
7. Blood supply	Enters at hilum as in principal spleen	Small vessels enter capsule at periphery
8. Capsule	Contains normal amount of smooth muscle and elastic tissue	Thick and fibrous—smooth muscle and elastic tissue largely decreased in amount or absent
9. Red pulp	Normal	Normal or undergoing various stages of degeneration—often increased amounts of hemosiderin
10. Malphighian corpuscles	Normal	Development and number may be decreased. Usual absence of small central artery
11. Trabeculae	Normal	Normal or decreased in number or development
12. Functional ability	Same as principal spleen	Evidence that well-developed transplants possess some of the functions of normal splenic tissue

2. The first case of intestinal obstruction directly due to splenosis is reported. This is also the initial report of a solitary intra-abdominal splenic transplant.

3. Experimental aspects of splenic autografts are discussed. Splenic tissue is readily transplanted in laboratory animals, and these transplants function as normal splenic tissue.

4. Clinical splenosis on the basis of the small number of reported cases is reviewed. At present, no typical clinical picture can be assigned to this condition.

5. The autoplastic splenic transplants are compared with true accessory spleens, and the differentiating characteristics developed.

6. A detailed tabulation of all of the cases of splenosis reported to date is included.

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* Deceased.

TABLE 6. *Tabulation of the Cases of Splenosis Presented in the World Literature since 1896*

Case Number	Author(s)	Year	Sex	Age at Time of Splenectomy	Age at Diagnosis of Splenosis	Interval	Occasion of Diagnosis	Location of Implants	No.	Size
1	Albrecht	1896	M	No history of splenectomy	25 yrs.	—	Autopsy	Parietal peritoneum, diaphragm, liver, root of mesentery, rectum, omentum, pouch of Douglas	400	Hirshkorn to hazelnut
2	Schilling	1907	F	No history of splenectomy	47 yrs.	—	Autopsy	Greater omentum, colon, gall bladder	44	Head of knitting needle to cherry pit
3	Küttner	1910	M	?	?	4 yrs.	Autopsy	Small and large intestines	80–100	Pea to cherry
4	Faltin	1911	F	9 yrs.	15 yrs.	6 yrs.	Appendicitis	Cecum, appendix, colon, small intestine	Numerous	Lentil to cherry
5	Stubenrauch	1912	M	?	?	10 mos.	Intestinal obstruction	Greater omentum, transverse mesocolon, small intestine	Numerous	Hempseed to pea sized
6	*Oltmanns	1919	?	?	?	?	?	Throughout peritoneal cavity	Multiple	?
7	Jolly	1919	F	No history of splenectomy	15 yrs.	—	Exploratory laparotomy	Greater omentum	Numerous	Millet grain to cherry seed
8	Lee and Sheff	1923	M	14 yrs.	29 yrs.	15 yrs.	Intestinal obstruction	Small intestine and mesentery	200–300	Pinhead to 1 × .5″
9	Kupperman	1936	M	15 yrs.	15 yrs.	6 mos.	Incisional hernioplasty	Scattered over abdominal cavity	100	Bean-sized
10	Shaw and Shafi	1937	M	?	20 yrs.	?	Autopsy	Parietal peritoneum, pleura, liver, diaphragm, retrovesicle pouch, greater omentum, mesentery, intestines, ureter and gall bladder	82	.2–2 cm. in diameter

* Quoted by Stubenrauch (46)

TABLE 6—*Continued*

Case Number	Author(s)	Year	Sex	Age at Time of Splenectomy	Age at Diagnosis of Splenosis	Interval	Occasion of Diagnosis	Location of Implants	No.	Size
11	Jarcho and Andersen	1939	M	6 yrs.	8 yrs.	18 mos.	Autopsy	Left diaphragm, liver, stomach, lesser omentum, transverse colon, right kidney, rectum, and bladder	Several doz.	1 to 12 mm. in diameter
12	Jarcho and Anderson	1939	M	12 yrs.	20 yrs.	8 yrs.	Autopsy	Peritoneum, greater omentum, diaphragm, stomach	80	1 mm. to 3 cm. in diameter
13	Buchbinder and Lipkoff	1939	F	9 yrs.	28 yrs.	19 yrs.	Exploratory laparotomy	Parietal peritoneum, greater omentum, ileum, ascending colon, sigmoid colon	Numerous	1 mm. to 2 cm. in diameter
14	Hamrick and Bush	1942	M	9 yrs.	12 yrs.	3 yrs.	Autopsy	Parietal peritoneum, pelvis, omentum, large and small intestine, left diaphragm	75	.5 to 1 cm. in diameter
15	Krueger and Mast	1942	M	11 yrs.	16 yrs.	5 yrs.	Intestinal obstruction	Both surfaces of the greater omentum	20 to 30	1 to 2 cm. in diameter
16	Gill	1944	M	52 yrs. gun-shot wound left chest—16 mos. later splenectomy with hiatal hernia repair	54 yrs.	2 yrs. following gunshot wound	Excision of mass in bullet wound scar	Chest wall-posterior axillary line at 10th rib	1	3 × 2 cm.
17	Boggs	1945	M	11 yrs.	14 yrs.	3 yrs.	Acute appendicitis and intestinal obstruction	Entire intestinal tract including 3 nodules on appendix	25 to 30	1 to 2 cm. in diameter
18	Waugh	1946	F	32 yrs.	37 yrs.	5 yrs.	Hysterectomy for fibroids	Small intestine and cecum	10 seen	Pinhead to pea sized

TABLE 6—*Continued*

Case Number	Author(s)	Year	Sex	Age at Time of Splenectomy	Age at Diagnosis of Splenosis	Interval	Occasion of Diagnosis	Location of Implants	No.	Size
19	Bunch and Spivey	1946	M	9 yrs.	12 yrs.	3 yrs.	Acute appendicitis	Retroperitoneum, mesentery, greater omentum, pelvis	100	Pea to acorn
20	Stobie	1947	F	25 yrs.	29 yrs.	4 yrs.	Cesarean section	Parietal peritoneum, uterus, omentum, pelvis	over 100	Pea to plum
21	Trossero	1949	F	37 yrs.	41 yrs.	4 yrs.	Inguinal hernioplasty	Anterior parietal peritoneum and pelvic peritoneum	Numerous	Head of a pin to a bean
22	Trossero	1949	M	No splenectomy—left upper quadrant trauma age 28	56 yrs.	28 yrs.	Laparotomy for stomach carcinoma	Greater omentum and parietal peritoneum	Numerous	Size of a bean or larger
23	Sampaio	1950	F	9 yrs.	19 yrs.	10 yrs.	Laparotomy for chronic appendicitis	Mesentery, cecum, ileum, posterior peritoneum	Considerable	Not stated—small
24	Raper	1951	M	?	30 yrs.	?	Autopsy	Abdominal scar, parietal peritoneum, diaphragm, foramen of Winslow, stomach, ileum, omentum	70	2 mm. to 1 cm. in diameter
25	Wise	1953	M	24 yrs.	24 yrs.	10 mos.	Intestinal obstruction	Greater omentum	10 to 20	Pinhead to 2 cm. in diameter
26	Storsteen and Remine	1953	M	17 yrs.	22 yrs.	5 yrs.	Intestinal obstruction	Small bowel, colon, omentum, mesentery, parietal peritoneum, both kidneys, pelvis	300 to 400	1 mm. to 2 cm. in diameter
27	Berry and Erdman	1953	M	12 yrs.	14 yrs.	2 yrs.	Intestinal obstruction	Small bowel, mesentery, omentum, adhesive band	Not stated	Up to 5 mm. in diameter

TABLE 6—*Continued*

Case Number	Author(s)	Year	Sex	Age at Time of Splenectomy	Age at Diagnosis of Splenosis	Interval	Occasion of Diagnosis	Location of Implants	No.	Size
28	Hartman	1953	F	30 yrs.	31 yrs.	1 yr.	Exploratory laparotomy	Small and large bowel, Fallopian tubes, uterus, bladder	Multiple	.1 to 1 cm. in diameter
29	Garamella and Hay	1954	M	?	22 yrs.	?	Acute appendicitis	Jejunum, right lateral peritoneal wall, meso-appendix	Multiple	2 removed 2 × 1.6 cm. and .5 cm. × 9 mm. in diameter
30	Cohen	1954	M	8 yrs.	30 yrs.	22 yrs.	Excision of mass in scar	In subcutaneous tissue of splenectomy scar	1	1.5 cm. in diameter
31	Ferry and Cabanne	1954	F	13 yrs.	23 yrs.	10 yrs.	Incisional hernioplasty	Free border of jejunum	Multiple	Size of a cherry
32	Pequinela	1954	M	34 yrs.	46 yrs.	12 yrs.	Intestinal obstruction	Distal ileum and cecum	18–20	1.5 × .5 cm. to 3 × 1 cm.
33	McCann	1956	M	15 yrs.	45 yrs.	30 yrs.	Laparotomy for G.I. bleeding	Small intestine, mesentery, greater omentum, cecum, ascending colon, lesser omentum	Numerous	Very small to 1.5 cm. in diameter
34	Skinner and Hurtean	1957	M	19 yrs.	29 yrs.	10 yrs.	Exploratory thoracotomy	Left visceral and parietal pleura, lung, ribs	6	.5 cm. in diameter to 3.5 × 2 × 1 cm.
35	Cotlar and Cerise	1958	M	12 yrs.	17 yrs.	5 yrs.	Intestinal obstruction	Small bowel mesentery, greater omentum	Numerous	"Freckle to end of thumb"
36	Cotlar and Cerise	1958	M	18 yrs.	20 yrs.	2 yrs.	Intestinal obstruction	Serosal surface of distal jejunum	1	1 cm. × 8 mm.

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