

Inadequate detection of accessory spleens and splenosis with laparoscopic splenectomy

A shortcoming of the laparoscopic approach in hematologic diseases

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Abstract

Background: The ultimate goal of surgery for hematological disorders is the complete removal of both the spleen and accessory spleens in order to avoid recurrence of the disease. Whereas splenectomy by open surgery provides excellent results, the validity of laparoscopic splenectomy in this regard remains unknown.

Objective: The purpose of this study was to evaluate the detection of accessory spleens during laparoscopic splenectomy for hematologic diseases.

Methods: We therefore evaluated the pre-, intra-, and postoperative detection of accessory spleens in a consecutive series of 18 patients treated by elective laparoscopic splenectomy for hematological diseases by using computed tomography (CT) and denatured red blood cell scintigraphy (DRBCS).

Results: Preoperative CT, DRBCS, and laparoscopic exploration detected 25%, 25%, and 75% of accessory spleens, respectively. At time of laparoscopy, 16 accessory spleens were detected in seven of the 18 patients (41%). In two patients (11%), laparoscopic exploration failed to detect accessory spleens, whereas preoperative CT (one case) and DRBCS (one case) did reveal them. Postoperatively, during a mean follow-up of 28 months (median, 24; range, 12–44 months), nine patients (50%) showed persistence of splenic tissue by DRBCS, and three of them had signs of disease recurrence.

Conclusions: This prospective clinical study suggests that elective laparoscopic surgery for hematological diseases does not allow complete detection of accessory spleens. Moreover, after such a laparoscopic approach, residual splenic tissue is detectable in half of the patients during the follow-up.

Key words: Splenectomy — Laparoscopy — Accessory spleen — Thrombocytopenic purpura — Scintigraphy — Computed tomography

In the normal population, the incidence of accessory spleen(s) (AS) is 10% [18], but in patients with hematological diseases, it may be as high as 30% [6, 24, 30]. An association has been reported between residual AS and recurrent thrombocytopenia after splenectomy by open surgery [1, 9, 12, 30, 34, 35], but recurrence of the disease may take years to become clinically detectable [2]. Thus, for permanent treatment of patients suffering from idiopathic thrombocytopenic purpura (ITP), complete removal of all splenic tissue, including AS, is crucial. Although the technical aspects, safety, and short-term results of laparoscopic splenectomy have been assessed thoroughly [5, 13–15, 26, 28, 33], only a few reports have focused on the detection and complete removal of splenic tissue in patients with hematological diseases [13–15, 31]. In order to assess the efficacy of the laparoscopic approach in this regard, patients submitted to laparoscopic splenectomy for hematological disorders were carefully evaluated in terms of the pre-, intra- and postoperative detection of AS.

Patients and methods

Patients

Between October 1992 and June 1995, we treated 18 consecutive adult patients (14 female, four male) by elective laparoscopic splenectomy. The mean age was 33 years (median, 29; range, 15–73 years). Among these patients, 16 suffered from ITP, one from congenital spherocytosis, and one from autoimmune hemolytic anemia. In ITP patients, the site of platelet

sequestration was splenic in 13 patients and mixed (hepatosplenic) in three patients (19%), as assessed by ^{111}In -oxinate-labeled platelets.

Preoperative localization

In order to detect the possible presence of an AS, all patients underwent preoperative computed tomography (CT) and $^{99\text{m}}\text{Tc}$ heat-denatured red blood cell scintigraphy (DRBCS). CT examinations were performed with a Somatom DRH scanner (Siemens, Erlangen, Germany). Before the CT examination, all patients drank 500–1000 ml of 2% meglumine ioxitalamate (Telebrix, Guerbet, Aulnay-sous-Bois, France) or 1.5% w/v diluted barium (Micropaque scanner, Guerbet) over 1 h to opacify the upper gastrointestinal tract. Incremental scanning of the abdomen was performed with a slice thickness of 8 mm. Meglumine ioxitalamate, 150 ml intravenously, was administered in eight patients.

The technique of DRBCS was a modification of the Fisher method [10]. Pure red blood cells were labeled with $^{99\text{m}}\text{Tc}$ pertechnetate by means of a commercial kit (UltraTag, Mallinckrodt Medical, St Louis, MO, USA). Labeled red blood cells were damaged by incubation for 20 min in a 49.5°C water bath. Planar images were obtained 60–90 min after intravenous injection of 555 MBq $^{99\text{m}}\text{Tc}$ -labeled red blood cells with a large-field-of-view gamma camera equipped with a low-energy, high-resolution collimator. One million counts were obtained in anteroposterior, posteroanterior, and left oblique projections. Single-photon-emission CT (SPECT) was performed immediately after the planar images were obtained. Sixty-four 20-s views were obtained through a 360° arc on a 64*64 matrix.

Reconstruction was performed by means of an iterative "false likelihood algorithm" [37] with scatter correction [36]. This algorithm is an accelerated version of the "expectation of maximum likelihood" (EML) method, which preserves the pixel positivity inside the body and null activity outside. The algorithm results in improved image quality in low-count regions, especially when surrounded by areas with high-count rates. Transverse, coronal, and sagittal tomographic slices of 1 pixel (9 mm) were displayed for interpretation.

Results of preoperative studies were available to the surgeon before surgery, except in one patient for whom the definitive report of preoperative DRBCS was known only after surgery. Location of residual splenic tissue on postoperative scintigraphic studies was described by reference to the left kidney—above, at the same plane, or below the kidney, and anterior or posterior to it (Fig. 1).

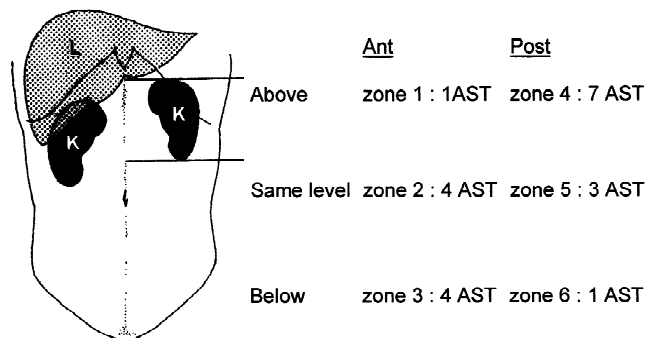
Surgical procedure

All patients underwent successful laparoscopic splenectomy, but one required a laparotomy to control hemorrhage from the splenic artery. In four additional cases, a splenic parenchymal injury was successfully treated during laparoscopy using an argon beam coagulator. During laparoscopic exploration, careful intraoperative search for AS was routinely achieved in the splenic hilum, the great omentum, and the left paracolic space, as well as in the lienocolic, splenorenal, and gastrocolic ligaments; the gastrocolic ligament was also opened to explore the lesser sac and the tail of the pancreas [8, 24, 30]. All splenic specimens were removed through a heavy plastic bag to avoid intraperitoneal spillage of splenic parenchyma.

Postoperative assessment

All patients had a normal platelet count at hospital discharge. The postoperative stay ranged from 2 to 12 days (mean, 4 days). During a mean of 28 months (median, 24; range, 12–44 months), all patients were followed by clinical examination and blood samples. Five patients suffering from ITP (31%) had recurrent thrombocytopenia (defined as a platelet count of $< 30 \times 10^3/\text{ml}$) 2, 8, 21, 25, and 35 months after laparoscopic splenectomy. Among 18 patients, 17 were studied by CT postoperatively (mean, one study per patient; range, one to two) after a mean interval of 10 months (median, 7; range, 2–22 months), whereas all patients underwent postoperative DRBCS (mean, two studies per patient; range, one to five) after a mean interval of 24 months (median, 22; range, 6–44 months).

FRONTAL VIEW



TRANSVERSE VIEW

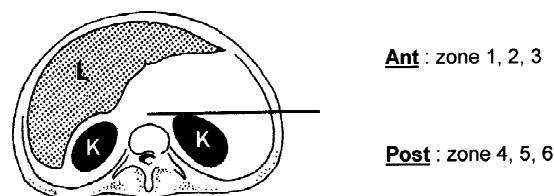


Fig. 1. Location of residual splenic tissue detected on denatured blood cell scintigraphy. L, liver; K, left kidney; Ant, anterior to the left kidney; Post, posterior to the left kidney; AST, accessory splenic tissue.

Results

Preoperative and intraoperative detection of AS

Preoperative CT, DRBCS, and careful intraoperative exploration did not reveal AS in 11 patients, whereas in seven other patients (41%), 16 AS were found during pre- and intraoperative exploration (Table 1). One AS was found in four patients and several (two, four, and six) in three others. In one of these seven patients, laparoscopic exploration failed to detect an 8-mm AS that was detectable on preoperative CT. This lesion was found by palpation in a fatty gastrocolic ligament during conversion to laparotomy (patient 4). In another case, preoperative CT and careful laparoscopic exploration revealed one AS. The surgeon completed the surgical procedure without having the information that a total of four AS were identified on preoperative DRBCS; sequential postoperative DRBCS confirmed the presence of three residual AS (patient 7).

Thus, laparoscopic exploration revealed 12 of 16 AS (75%). However, in at least two patients (11%), preoperative localization studies (CT and DRBCS in one patient each) were superior to careful laparoscopic search for AS. Preoperative CT demonstrated AS in four of seven patients (57%), but this finding represented only four of the 16 AS

Table 1. Pre-, intra-, and postoperative data on patients with accessory spleen or residual accessory splenic tissue in the course of laparoscopic splenectomy

Patient no.	Hematologic disease	Preop localization studies		Intraoperative AS detection		Clinical recurrence		Postoperative DRBCS	
		CT	DRBCS	No.	Size (mm)	Y/N	Delay (mo)	Number of hot spots	Delay (mo)
With AS									
1	ITP	0	0	1	6	Y	44	0	44
2	ITP	0	0	6	1–6	Y	25	1	24
3	ITP	0	0	1	5	N	19	3	18
4	ITP	1	0	0 (1) ^a	8	N	39	0	12
5	HS	1	0	2	11–15	N	35	1	35
6	ITP	1	0	1	10	Y	27	3	27
7	AIHA	1	4	1	18	N	24	3	9
Without AS									
1	ITP	0	0	0	^b	Y	40	2	31
2	ITP	0	0	0	^b	N	40	4	40
3	ITP	0	0	0	^b	N	38	2	38
4	ITP	0	0	0		N	17	1	6

AS, accessory spleen(s); DRBCS, denatured red blood cell scintigraphy; CT, computed tomography; ITP, idiopathic thrombocytopenic purpura; HS, hereditary spherocytosis; AIHA, autoimmune hemolytic anemia; Y, yes; N, no

^a Detected only during conversion to laparotomy for hemorrhage

^b Capsular splenic tearing occurred during laparoscopic surgery

present (25%). CT examination detected AS of 8 mm in four of five patients (80%), but none of the eight AS measuring < 8 mm. However, these AS represented 61% of all intraoperatively detected AS. Preoperative DRBCS demonstrated four of the 16 AS (25%), but failed to reveal any AS in six patients. The detection rate of combined preoperative CT and DRBCS was 44% (seven of 16 AS).

Postoperative residual accessory splenic tissue

Sequential postoperative DRBCS revealed persistence of splenic tissue in nine of 18 patients (50%). These residual hot spots on DRBCS were single in three patients and multiple in six (Table 1). The anatomical sites of these 20 spots, all located in the left abdomen, are shown in Fig. 1. It is worth noting that four of these 20 anatomical sites were not detected by planar imaging at DRBCS and were visible only on three-dimension iterative reconstruction tomographic display. Further, SPECT allowed a better delineation of the lesions. In comparison, postoperative CT examinations confirmed the presence of residual AS in only one patient, as shown on postoperative DRBCS. In another patient, sequential postoperative DRBCS were negative at 4, 11, and 16 months, but they became positive at 27 months and confirmed at 35 months. This single spot was located quite caudally in the left flank, which suggested seeding of splenic tissue.

These residual splenic tissues were detected in four of the 11 patients (36%) in whom AS was not detected by preoperative localization studies and intraoperative search (Fig. 2), and in five of the seven patients (71%) in whom AS was detected during surgery (Fig. 3). Three of the four patients (75%) who had splenic capsular injury during laparoscopic splenectomy exhibited detectable residual hot spots during the follow-up; whereas, in comparison, five of 14 patients (36%) who did not have splenic parenchymal injury during surgery demonstrated the same features. How-

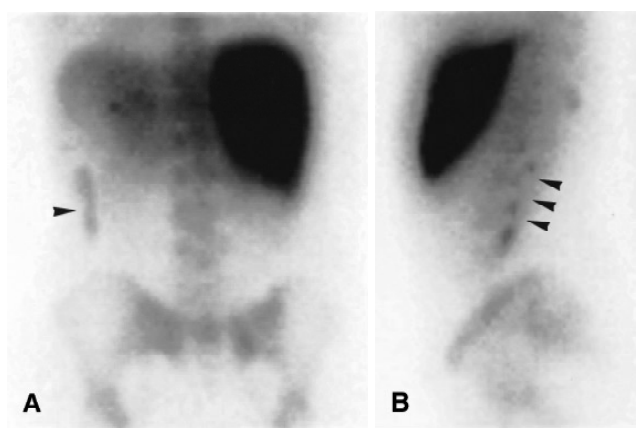


Fig. 2. Postoperative denatured blood cell scintigraphy demonstrated four foci of residual splenic tissues (arrow) in a 17-year-old female patient suffering from idiopathic thrombocytopenic purpura. No accessory spleen was detected pre- or intraoperatively. There has been no clinical recurrence at 40 months postoperatively. **A** Posterior view. **B** Left lateral view.

ever, the difference was not statistically significant (Fisher's exact test).

Early clinical recurrence (at 2 and 8 months postoperatively) was a consequence of hepatic platelet sequestration in two patients. In three patients, late recurrence (at 21, 25, and 35 months postoperatively) was associated with postoperative residual accessory tissue. One of these patients underwent surgical reexploration to confirm the presence of a single AS previously evidenced at DRBCS. An accessory splenectomy was performed successfully in this case.

Discussion

After splenectomy, persistent splenic tissue can be either congenital (undetected AS) or acquired as a result of sple-

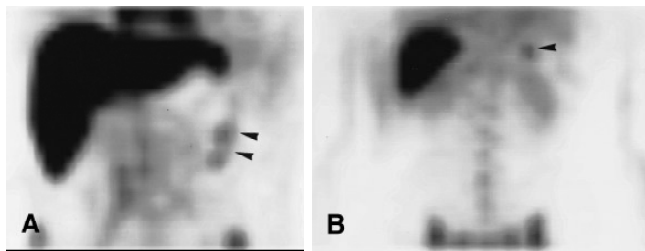


Fig. 3. Postoperative denatured red blood cell scintigraphy in a young woman with a single accessory spleen detected on preoperative CT and removed during surgery. At 27 months postoperatively, DRBCS demonstrated three foci of residual splenic tissue (arrow). Two foci are located in the left flank (**A**) and one in the splenic bed (**B**) overlying the left kidney. The patient had recurrent thrombocytopenia 21 months postoperatively.

nosis or autotransplantation of splenic tissue after accidental or iatrogenic injury of the spleen [23]. The main findings of this study are (a) that intraoperative laparoscopic search does not detect all AS and (b) that residual splenic tissue is found postoperatively in half of the patients following laparoscopic splenectomy.

We previously reported on the potential limitations of the laparoscopic approach for detecting AS in patients suffering from hematological diseases [13–15]. The present study only confirms these limitations, despite our close attention to this problem, with a detection rate of AS in 41% of patients during laparoscopy. The literature shows a wide variation of AS detection during laparoscopic splenectomy; various series report incidences of AS that vary from 0 to 14% [5, 6, 15, 26, 33] or up to 26% [13] and 30% of the patients [28]. In comparison, when using a laparotomy approach, the presence of AS has been reported in 18–43% of patients with idiopathic thrombocytopenic purpura [1, 8, 24, 30].

These results are undoubtedly related to the diligence of the surgeon in searching for this tissue [11]. Therefore, we must emphasize that routine, careful search for AS in the left upper abdominal quadrant and the left paracolic gutter is essential, as is the routine opening of the gastrocolic ligament to explore the lesser sac and the area of the pancreatic tail. Using this meticulous approach, intraoperative laparoscopic exploration provided a higher detection rate (75%) than combined preoperative localization examination (CT and DRBCS), which can only identify 25% of AS.

The dominant uptake of the orthotopic spleen at DRBCS represents the major limitation for detecting small adjacent accessory splenic tissue prior to surgery. Moreover, the limitation of CT for AS detection is related to the small size of the lesions, since undetected AS at CT were < 8 mm in 61% of all cases detected during laparoscopy. The use of more sensitive techniques, such as spiral CT, might improve detection in the near future. However, careful surgical exploration remains the most efficient way to detect AS. Therefore, we cannot recommend routine preoperative use of CT or DRBCS.

The second most striking feature of this series was the high incidence (50%) of residual splenic tissue detected on routine postoperative scintigraphy after laparoscopic splenectomy. To the best of our knowledge, this is the first report of such a high incidence using a laparoscopic ap-

proach. Of all scintigraphic studies, DRBCS, which improves the visualization of small foci of splenic tissue, is considered to be the most efficient technique for the detection of splenosis after splenectomy [17, 20, 22, 40]. Further, SPECT is mandatory to improve the delineation of small foci, especially in the splenic bed.

Because of the proximity of high liver activity, we found that iterative reconstruction [37] provided better results than standard filtered back projection. Underdetection of occult AS during laparoscopic splenectomy was the cause of these residual spots in at least one of our patients. In the other patients, however, a mechanism of splenosis related to intraperitoneal seeding of splenic tissue during laparoscopic surgery might be possible. Late detection of residual splenic tissue in one patient who had several previous negative postoperative scintigraphic studies could be regarded as support for this hypothesis. On the other hand, splenosis was also found in 30% of patients who had no evidence of AS pre- or intraoperatively.

Splenosis has been reported in rare cases of elective open splenectomy for hematological diseases (16 and 17%) [22, 32]. In comparison, the incidence of splenosis in patients splenectomized for trauma range between 44% and 76% [17, 22, 23, 25, 32, 40]. Nielsen et al. [22] reported a 45% rate of splenosis, after a median interval of 4 years, following accidental splenic injury during elective abdominal surgery. As in our patients, splenosis was usually located in the left upper quadrant [22, 23].

Several technical factors during laparoscopic surgery could be involved in the pathogenesis of splenosis. First, at least four of our patients had splenic parenchymal injury during laparoscopic splenectomy. Three of them presented residual hot spots on postoperative DRBCS at long-term follow-up. Therefore, great care must be taken to manipulate the spleen gently with an atraumatic retractor. Another mechanism of seeding, similar to that encountered in malignant diseases treated laparoscopically, is trocar site recurrence [38, 39]. However, none of our cases of residual splenic tissue involved a trocar site (Fig. 1).

Finally, the method of extraction of the surgical specimen during laparoscopic splenectomy is of utmost importance. We used intrabag morcellation, paying close attention to the resistance and impermeability of the plastic bag, in an attempt to avoid intraperitoneal seeding of splenic tissue. Other extraction techniques, such as intraperitoneal morcellation of the spleen, should be avoided. The use of an argon beam coagulator to control hemorrhage from capsular splenic tears may also be involved in the mechanism of splenosis, as a result of the local spray of splenic tissues.

The final question we must address is the clinical significance of postoperative residual splenic tissue in patients treated for hematological diseases. The clinical importance of accessory or residual splenic tissues and the relation between the presence of an accessory spleen and recurrent thrombocytopenia remains unknown. In ITP patients, the mechanisms of recurrence are multifactorial, but persistence of AS is known to be one of them [1, 9, 12, 30, 34, 35]. Facon et al. [9] reported a 12% incidence of residual AS in 65 patients with recurrent thrombocytopenia 1 year after open splenectomy. It is well known that a residual lesion may hypertrophy and become the cause of long-term clini-

cal recurrence, which may appear even years later [2]. In at least three of our ITP patients with recurrent thrombocytopenia, the clinical recurrence could have been related to persistent splenic tissue. However, because these patients were successfully controlled by medical therapy, accessory splenectomy was not performed in all cases. Time will tell whether progressive increase of residual splenic tissue results in late disease recurrence. Among a compilation of 84 patients who underwent accessory splenectomy for recurrent thrombocytopenia, 65% (55 patients) were successfully cured during a long-term follow-up [1, 9, 12, 30, 34, 35]. Moreover, the rate of recurrent thrombocytopenia in the present series was 33% in ITP patients and 31% in ITP patients with predominant splenic destruction of platelets. These results should be compared to those obtained with an open conventional approach. Among 1457 patients reported in the literature [3, 4, 7, 27], 65–79% of all ITP patients were successfully cured in the long term. In addition, an incidence of 78–93% was obtained for 409 ITP patients with predominant splenic destruction of platelets [16, 19, 21, 29]. Given the existence of long-term follow-up in these clinical series using an open surgical procedure, it is possible that we have underestimated the relapse rate in our patients based on the shorter follow-up.

When laparoscopic splenectomy is performed, we therefore suggest that postoperative DRBCS be performed routinely to identify patients with residual splenic tissue. These patients need to be assessed regularly for clinical recurrence. Until further information is available about the role of residual splenic tissue in the long-term clinical outcome of the patient, elective laparoscopic splenectomy for the treatment of patients with ITP should be used with caution.

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References

- Ambriz P, Munoz R, Quintanar E, Sigler L, Aviles A, Pizzuto J (1985) Accessory spleen compromising response to splenectomy for idiopathic thrombocytopenic purpura. *Radiology* 1: 793–796
- Appel MF, Bart JB (1976) The surgical and hematologic significance of accessory spleens. *Surg Gynecol Obstet* 143: 191–192
- Ben-Yehuda D, Gillis S, Eldor A, Israeli ITP Study Group (1994) Clinical and therapeutic experience in 712 Israeli patients with idiopathic thrombocytopenic purpura. *Acta Haematol* 91: 1–6
- Berchtold P, McMillan R (1989) Therapy of chronic idiopathic thrombocytopenic purpura in adults. *Blood* 74: 2309–2317
- Cadiere GB, Verroken R, Himpens J, Bruyns J, Efila M, Dewit S (1994) Operative strategy in laparoscopic splenectomy. *J Am Coll Surg* 179: 668–672
- Carroll BJ, Phillips EH, Semel CJ, Fallas M, Morgenstern L (1992) Laparoscopic splenectomy. *Surg Endosc* 6: 183–185
- Coon WW (1987) Splenectomy for idiopathic thrombocytopenia purpura. *Surg Gynecol Obstet* 164: 225–229
- Curtis G, Movitz D (1946) The surgical significance of the accessory spleen. *Ann Surg* 123: 276–298
- Facon T, Caulier MT, Fenaux P, Plantier I, Marchandise X, Ribet M, Jovet JP, Bauters F (1992) Accessory spleen in recurrent chronic immune thrombocytopenic purpura. *Am J Hematol* 41: 184–189
- Fisher J, Wolf R, Leon A (1967) Technetium-99 m as a label for erythrocytes. *J Nucl Med* 8: 229–232
- Friederici H (1979) Born-again spleens. *N Engl J Med* 300: 258
- Gibson J, Rickard KA, Bautovich G, May Y, Kronenberg K (1986) Management of splenectomy failures in chronic immune thrombocytopenic purpura: role of accessory splenectomy. *Aust NZ J Med* 16: 695–698
- Gigot JF, de Ville de Goyet J, van Beers BE, Reding R, Etienne J, Jadoul P, Michaux JL, Ferrant A, Cornu G, Otte JB, Pringot J, Kestens PJ (1996) Laparoscopic splenectomy in adults and children: experience with 31 patients. *Surgery* 119: 384–389
- Gigot JF, Healy ML, Ferrant A, Michaux JL, Njinou B, Kestens PJ (1994) Laparoscopic splenectomy for idiopathic thrombocytopenic purpura. *Br J Surg* 81: 1171–1172
- Gigot JF, Legrand M, Cadiere GB, Delvaux G, de Ville de Goyet J, de Neve de Roden A, Van Vyve E, Hourlay P, Etienne J, Njinou B, Collard A (1995) Is laparoscopic splenectomy a justified approach in hematologic disorders? Preliminary results of a prospective multicentric study. *Int Surg* 80: 299–303
- Gugliotta L, Isacchi G, Guarini A, Ciccone F, Motta MR, Lattarini C, Bachetti G, Mazzuconi MG, Baccarini M, Mandelli F, Tura S (1981) Chronic idiopathic thrombocytopenic purpura (ITP): site of platelets sequestration and results of splenectomy: a study of 197 patients. *Scand J Haematol* 26: 407–412
- Gunes I, Yilmazlar T, Sarikaya I, Akbunar T, Irgil C (1994) Scintigraphic detection of splenosis: superiority of tomographic selective spleen scintigraphy. *Clin Radiol* 49: 115–117
- Halpert B, Gyorkey F (1959) Lesions observed in accessory spleens of 311 patients. *Am J Clin Pathol* 32: 165–168
- Lamy T, Moisan A, Dauriac C, Ghandour G, Morice P, Le Prise PY (1993) Splenectomy in idiopathic thrombocytopenic purpura: its correlation with the sequestration of autologous indium-111 labelled platelets. *J Nucl Med* 34: 182–186
- Massey MD, Stevens JS (1991) Residual spleen found on denatured red blood cell scan following negative colloid scans. *J Nucl Med* 32: 2286–2287
- Najean Y, Dufour V, Rain JD, Toubert E (1991) The site of platelet destruction in thrombocytopenic purpura as a predictive index to the efficacy of splenectomy. *Br J Haematol* 79: 271–276
- Nielsen JL, Ellegaard J, Marqvorsen J, Hansen HH (1981) Detection of splenosis and ectopic spleens with ^{99m}Tc-labelled heat damaged autologous erythrocytes in 90 splenectomized patients. *Scand J Haematol* 27: 51–56
- Normand JP, Rioux M, Dumont M, Bouchard G (1993) Ultrasonographic features of abdominal ectopic splenic tissues. *Can Assoc Radiol J* 44: 179–184
- Olsen WR, Beaudoin DE (1969) Increased incidence of accessory spleen in hematologic disease. *Arch Surg* 98: 762–763
- Pearson HA, Johnston D, Smith KA, Touloukian RJ (1978) The born-again spleen: return of splenic function after splenectomy for trauma. *N Engl J Med* 298: 1389–1392
- Phillips EH, Carroll BJ, Fallas MJ (1994) Laparoscopic splenectomy. *Surg Endosc* 8: 931–933
- Pizzuto J, Ambriz R (1984) Therapeutic experience on 934 adults with idiopathic thrombocytopenic purpura: multicentric trial of the Cooperative Latin American Group on Hemostasis and Thrombosis. *Blood* 64: 1179–1183
- Poulin EC, Thibault C, Mamazza J (1995) Laparoscopic splenectomy. *Surg Endosc* 9: 172–177
- Ries CA, Price DC (1974) [⁵¹Cr] platelet kinetics in thrombocytopenia; correlation between splenic sequestration of platelets and response to splenectomy. *Ann Intern Med* 80: 702–707
- Rudowski WJ (1985) Accessory spleens: clinical significance with particular reference to the recurrence of idiopathic thrombocytopenic purpura. *World J Surg* 9: 422–430
- Rudowski WJ (1995) Laparoscopic splenectomy [Letter]. *Am J Surg* 169: 282–283
- Spencer GR, Bird C, Prothero DL, Brown TR, Mackenzie FAF, Phillips MJ (1981) Spleen scanning with ^{99m}Tc-labelled red blood cells after splenectomy. *Br J Surg* 68: 412–414
- Trias M, Targarona EM, Balague C (1996) Laparoscopic splenectomy: an evolving technique. *Surg Endosc* 10: 389–392

34. Verheyden CN, Beart RW, Clifton MD, Phyliky RL (1978) Accessory splenectomy in the management of recurrent idiopathic thrombocytopenic purpura. *Mayo Clin Proc* 53: 442–446
35. Wallace D, Fromm D, Thomas D (1982) Accessory splenectomy for idiopathic thrombocytopenic purpura. *Surgery* 91: 134–136
36. Walrand SHM, van Elmbt LR, Pauwels S (1994) Quantitation in SPECT using an effective model of the scattering. *Phys Med Biol* 39: 719–734
37. Walrand SH, van Elmbt LR, Pauwels S (1996) A non-negative fast multiplicative algorithm in 3D-scatter compensated SPET reconstruction. *Eur J Nucl Med* 23: 1521–1526
38. Wexner SD (1995) Port site metastases after laparoscopic colorectal surgery for cure of malignancy. *Br J Surg* 82: 295–298
39. Wibbenmeyer LA (1995) Laparoscopic cholecystectomy can disseminate in situ carcinoma of the gallbladder. *J Am Coll Surg* 181: 504–509
40. Zwas ST, Samra D, Samra Y, Sibber GR (1986) Scintigraphic assessment of ectopic splenic tissue localization and function following splenectomy for trauma. *Eur J Nucl Med* 12: 125–129