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Splenosis and sepsis
The born-again spleen provides poor protection

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Key words: splenosis, sepsis, overwhelming post-splenectomy infection, accessory spleen, splenic physiology

Abbreviations: OPSI, overwhelming post-splenectomy infection

Splenosis describes ectopic splenic tissue found in patients after rupture of the spleen. These implants are commonly located on the omentum but can be scattered throughout the body in varying number and size. Although splenosis was first documented over a century ago, the precise mechanism for its development remains unknown. The degree of immunoprotection offered by this tissue remains unclear. Much of the human data is in the form of case reports documenting failure of splenotic tissue to protect against septicemia. Even accessory spleens may not offer complete protection once the primary spleen is removed. This review of the literature demonstrates that no amount of splenosis should be considered protective against overwhelming post-splenectomy infection.

Introduction

Commonly, the spleen has been considered an accessory organ without vital function; more recently however there has been renewed interest in preserving the spleen in circumstances in which splenectomy is thought to be necessary.1 Splenectomized patients are at higher risk of many long-term sequelae including cardiovascular disease,2 portal vein thrombosis,3 and possibly an increased rate of malignancy.4 The best-known complication of splenectomy may be overwhelming post-splenectomy infection (OPSI), where the body, lacking the spleen’s critical immunologic function, is quickly overrun with infection. It has been suggested that splenic nodules may offer a degree of immunologic defense when created serendipitously or intentionally during surgery.5 Our review examines the evidence that such nodules significantly influence host response to infection and specifically attempts to answer whether the condition of splenosis can protect against OPSI.

Splenosis describes ectopic splenic tissue found in patients after rupture of the spleen. These implants are commonly located on the omentum but can be scattered throughout the body in varying number and size. The term “born-again spleen” was coined by Pearson et al.4 to describe reincarnated splenic nodules following splenectomy for trauma. Using red cell surface indentations as a marker for splenic hypofunction, splenosis resulted in fewer “pits” suggesting a return of splenic function. Historically, splenosis is discovered in patients during either autopsy or surgical exploration. With an increase in more advanced imaging techniques, splenosis may also be detected as an incidental mass.

The regenerative properties of the spleen were first described in 1883 by Griffini and Tizzoni, who found numerous implants bespeckling the peritoneal surface of splenectomized dogs.7 Concordant findings in splenectomized humans were documented in 1896 by Albrecht in a case report illustrating more than 400 peritoneal splenic implants.8 Since the first description of splenosis, many additional cases have been reported and theories proposed regarding the pathophysiologic implications of this condition. Splenosis was so named in 1939 by Buchbinder and Lipkoff,9 and differs from accessory spleens in that accessory spleens develop along the dorsal mesogastrum and are vascularized by the splenic artery. Accessory spleens are also fewer in number, have a hilum, and resemble an intact spleen.10

Splenic Anatomy and Physiology

In its French linguistic origins, “spleen” refers to melancholy, a name bestowed upon the organ historically containing that most melancholic humor, black bile. Galen felt that the purpose of the spleen was to harbor this black bile and to purify the contents of the liver. Modern understanding of the function of the spleen includes the filtration of blood, polishing of erythrocytes, pooling of cells, hematopoiesis, and most notably its immunoprotective function.

The human spleen is an organ encapsulated in a fibrous shell that has a sparse amount of smooth muscle in its inner layer (Fig. 1). The spleen (about 175 g in the normal human adult) receives 5% of the body’s cardiac output via the splenic artery,11 which enters the spleen and arborizes, each branch surrounded by an armament of lymphocyte-rich white pulp and, further out, the macrophage-rich red pulp (Fig. 2). Blood enters the splenic cords and returns to the circulation by squeezing through fenestrations between venous sinus endothelial cells (Fig. 3).12 The normal erythrocyte exhibits great compliance as it maneuvers through these fenestrae, where imperfect cells are confronted by
the scrutinizing splenic macrophages and subjected to culling (cellular destruction) or pitting (the removal of cellular inclusions). An erythropoietic organ, the spleen is the primary source of blood cells when the fetus is between six and seven months of age. Although the spleen may have “islands” of hematopoiesis in certain infiltrative and hemolytic states and is a known site of extramedullary hematopoiesis, the organ appears to lack the ability to initiate this process; rather, the spleen supports the growth of circulating immature erythrocytes. There is, however, recent evidence to suggest that the spleen can support endogenous myelopoiesis.

The spleen contains a variety of cells involved in the immune response. Encircling the arteries are the lymphocytes, a quarter of the body’s total lymphatic mass. As the contents of the blood travel through the sinuses, the periarterial lymphatic sheath (PALS) and lymphatic nodules are exposed to immunogenic stimuli (Fig. 1). The spleen is the major site for the production of IgM, which may increase ten-fold in splenomegalic patients; IgM levels are conversely diminished in asplenia or hyposplenia, and splenectomized patients may have a 75% decrease in circulating B-1a B cells. The spleen has been implicated in the storage and acute release of monocytes in the face of inflammation, and Swirski and colleagues recently demonstrated monocytes moving from a splenic reservoir to the myocardium after infarction.

The consequence of splenic loss for immunologic function is seen in the incidence of OPSI. Splenectomy may carry a relative risk of OPSI as low as 2.03–2.07 when due to trauma or idiopathic thrombocytopenic purpura (ITP), or as high as 11.6 when removed for thalassemia. Other splenectomy indications such as spherocytosis, Hodgkin’s lymphoma and portal hypertension, have an intermediate risk. There is an estimated 58 times increased mortality or higher in septic patients with splenosis compared to normosplenic individuals. In a patient series by Hansen and Singer, 2.4% of patients splenectomized for...
Opsonized antigens are efficiently disposed of by the splenic macrophages, more extensively than in other reticuloendothelial organs such as the liver. The intact spleen is the way in which its reticular structure facilitates the immune response. The endothelial cells lining the venous sinuses rest atop a basal plasma membrane network of stress fibers, composed of actin and myosin-like filaments. These

Figure 2. Diagram of the splenic artery. Arterial blood pools in the splenic cords before entering the splenic sinuses and returning to systemic circulation. (From Schiffman FJ, ed. Hematologic Pathophysiology. Philadelphia, PA: Lippincott-Raven, 1998. Used with permission).
fibers create tension in the fenestrations through which cells must squeeze, facilitating greater scrutiny of deformed or infected erythrocytes (Fig. 3). Cells infected by parasites or worn from old age become trapped between these stress fibers and exposed to the immunologic response. Moreover, splenic endothelial cells express mannose receptors and Toll-like receptors, helping to capture and present antigens, including encapsulated bacteria. The very infrastructure of the spleen thus appears to play a role in the overall immune response.

Etiology of Splenosis

From early studies, it was clear that traumatic or surgical disruption of the spleen was a crucial step in the seeding process. Although the earliest reported cases of splenosis did not note a history of trauma—and in fact postulated an embryonic etiology—they did find a small, scarred, adherent primary spleen in the patients. While hypotheses such as activation of embryonic rests of splenic tissue have been proposed, there is almost invariably a history of trauma or surgical intervention in patients with splenosis.

Drawing upon the observation that splenosis appears to preserve some degree of splenic function, surgical autotransplantation at the time of total splenectomy was pursued beginning in the early 1980s. These implantations do not require a vascular anastomosis at their implantation site, and the process by which the tissue regenerates has been well documented in various animal species. Initially, a portion of the transplant undergoes necrosis; later, in a centripetal manner, it repopulates the mass with splenic tissue. The central portion may remain fibrotic or degenerative, with the periphery acting as a partially functioning spleen.

Rat models show that splenic tissue implanted in an omental pouch has improved pneumococcal clearance compared to intraperitoneal and intramuscular implants, suggesting preserved function of splenic tissue that retains a connection with the portal circulation. Surgical pseudosplenosis typically utilizes the mesentery as a receptive tissue bed in order to maximize the probability of success. Physiologic splenosis occurs primarily throughout the abdomen and pelvis, but can also be found elsewhere. It does not appear to have the same requisite connection to the portal circulation as do surgical treatments; however, it is unclear whether tissue implanted away from the portal circulation has the same degree of immune function. While patients with splenosis are usually asymptomatic, the condition may result in gastrointestinal bleeding, hemoptysis, chest pain, bowel obstruction, hydronephrosis, or be mistaken for malignancy.
There is a long latency between splenic rupture and any manifestations of splenosis.\textsuperscript{46} Intrahepatic splenosis, an extremely rare manifestation of this condition, is often found as a 2–4 cm nodule and not apparent for years following initial splenic trauma.\textsuperscript{39,47–49} Since an acute embolic splenic nodule of such diameter would be expected to result in severe compromise of the portal circulation, this suggests that tissue from the ruptured spleen expanded after deposition within the liver.

In the face of acute erythropoietic stress, such as severe anemia, the spleen may resume erythropoiesis. Tissue hypoxia stimulates erythropoietin secretion, which in turn induces the BMAP/Madh5 signaling pathway to cause differentiation of splenic progenitor cells.\textsuperscript{50} Splenic stem cells in these splenunculi may be an important factor in the etiology of splenosis.

Splenotic implants differ from normal spleen, suggesting that these tissues are the result of cellular growth and are not simply redistributed portions of disrupted spleen. Splenotic tissue has a decreased amount of white pulp,\textsuperscript{51,52} and while it contains many elements of the normal spleen, the splenic nodules are smaller in mass with reduced individual compartments.\textsuperscript{47} The reduced immune function of these ectopic implants may be partially related to the decreased amount of white pulp.\textsuperscript{49} There is no regeneration of the delicate but efficient arterial and venous circulating beds that are also important in the spleen’s immunologic role. Timens and Leeman\textsuperscript{53} offer a plausible explanation for the lack of full protection from sepsis by ectopic splenic tissue: “First the total amount of blood that is filtered is low, despite an acceptable vascularization. Second, the microanatomy of the splenic fragments is probably not suited for the specific local low flow that is characteristic for the normal spleen and is essential for the close contact between antigen and phagocytes and immunoresponsive cells.” It would appear then, that splenosis occurs following traumatic disruption of the spleen, which sends progenitor cells throughout the viscera, implanting as ectopic rest and further differentiating into tissue that resembles, but does not recapitulate, the human spleen.

**Imaging and Identification of Splenunculi**

Investigations of the prevalence of splenosis on imaging have suggested that this condition may actually occur in one-third to two-thirds of patients following splenectomy for trauma.\textsuperscript{42,54,55} Even thoracic splenosis, a less common presentation, may exist in up to one-fifth of patients with simultaneous spleen and diaphragmatic injury.\textsuperscript{56} When imaging reveals a splenic nodule that may have otherwise gone unnoticed, the clinician faces a diagnostic dilemma, particularly in the case of intrathoracic or intrahepatic masses.\textsuperscript{42,57,58} Previously unrecognized splenic nodules may result in unnecessary biopsy and interventions, including thoracotomy for intrathoracic splenosis or abdominal exploration for intraabdominal masses. A trauma history and tumor markers may be useful in distinguishing between benign and more serious lesions.\textsuperscript{47,59}

While biopsy remains the gold standard for diagnosis, imaging in combination with a history of splenic trauma or splenectomy can avoid excessive intervention. Currently, the preferred imaging modality to identify ectopic splenic rests is with Technetium Tc 99m heat-damaged RBCs.\textsuperscript{60} Damaged RBCs deposit in splenic tissue throughout the body as they are scrutinized for defects; radionuclide tagging of these erythrocytes achieves increased specificity in identifying splenic rests and decreased background when compared to older techniques such as Technetium Tc 99m sulfur colloid imaging.\textsuperscript{60,62} There are several ultrasonographic findings that suggest splenic tissue, including multiple feeding vessels and a homogeneous inner pattern, but these require confirmation using radionuclide studies.\textsuperscript{62} Computed tomography and magnetic resonance imaging may also identify splenic masses;\textsuperscript{63} in particular, the use of super-paramagnetic iron oxide particles, which localize to sites of phagocytotic reticuloendothelial cells, has been used to identify splenosis on MRI.\textsuperscript{44} In the future, MRI may become more commonly used, as it allows for increased resolution compared to radionuclide uptake while still targeting reticuloendothelial tissue.\textsuperscript{65,66}

**Splenosis and Infection**

**Animal studies.** In studies using partially splenectomized laboratory animals, retention of 30–50% of whole splenic tissue mass has protected against experimental pneumococcal sepsis.\textsuperscript{30,67–71} Leonard and colleagues noted that splenic opsonic and complement activities were normal even when two-thirds of the whole spleen was removed; however, actual clearing of bacteria from the blood was delayed when 50% of the spleen was removed.\textsuperscript{72} In these experiments, unlike in post-splenectomy splenosis, a significant amount of native splenic tissue and its unique blood flow were preserved.

Willfuhr et al.\textsuperscript{73} found that splenic autotransplantation resulted in better regeneration of splenic tissue in young rats when compared to old rats. Inuma et al.\textsuperscript{35} found that the most effective site for autotransplantation in rats was the omental pouch, and that 50% of the whole spleen mass was necessary for protection against sepsis. Steely et al.\textsuperscript{74} reported that autotransplantation of 80% of the spleen in rats is the optimum amount to maximize survival in a model of pneumococcal sepsis. Indeed, Moxon and Schwartz\textsuperscript{75} found that rats with heterotopic splenic autotransplantation were not protected against blood stream dissemination but were protected against bacterial meningitis. More recently, Smith et al.\textsuperscript{76} measuring the phagocytosis of labeled bacteria with flow cytometry, found that autotransplanted splenic tissue did not restore the splenic phagocytic capacity which was lost following splenectomy. Tang et al.\textsuperscript{77} found that splenic tissue autotransplantation in rabbits did not restore host defense as measured by pneumococcal clearance and serum lysozyme levels.

**Human studies.** Much of the human data is in the form of case reports documenting failure of splenotic tissue to protect against sepscemia, even at times when there was a large amount of splenosis. Articles involving splenosis and OPSI are summarized in Table 1.\textsuperscript{78–86} while those involving accessory spleens and OPSI are summarized in Table 2.\textsuperscript{79,87–90} Zarrabi and Rosner\textsuperscript{91} reported serious infections in adults following splenectomy for trauma: Four out of their 47 patients died...
had died from OPSI involving pneumococcus. One had a single accessory spleen weighing 4 g while the other had several ectopic spleens—the largest 12 g in weight—seven years after splenectomy. Navarro and Kondlapoodi\textsuperscript{90} have also reported on the failure of accessory spleens to prevent infection following splenectomy. Singer\textsuperscript{21} reported a fatal case of \textit{H. influenzae} infection in a child with approximately 15 g of splenic implants. He postulated that the critical quantity of the spleen needed for protection was 20 g (approximately \( \frac{1}{4} \) of the expected weight of despite ectopic splenic tissue found at autopsy. Rice and James\textsuperscript{82} reported two patients who died of post-splenectomy sepsis. One was a 12-year-old girl who expired with over 100 splenunculi, most 2–4 mm in diameter—the largest weighing 3 g. The other was a 19-year-old man who died of post-splenectomy sepsis with 92 g of splenunculi.

Even accessory spleens may not offer complete protection once the primary spleen is removed. Gopal and Bisno\textsuperscript{79} reported accessory spleens at autopsy in two of their five post-trauma cases who had died from OPSI involving pneumococcus. One had a single accessory spleen weighing 4 g while the other had several ectopic spleens—the largest 12 g in weight—seven years after splenectomy. Navarro and Kondlapoodi\textsuperscript{90} have also reported on the failure of accessory spleens to prevent infection following splenectomy.

Singer\textsuperscript{21} reported a fatal case of \textit{H. influenzae} infection in a child with approximately 15 g of splenic implants. He postulated that the critical quantity of the spleen needed for protection was 20 g (approximately \( \frac{1}{4} \) of the expected weight of

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**Table 1. Reports of OPSI and splenosis**

<table>
<thead>
<tr>
<th>Reference</th>
<th>Article type</th>
<th>Patient Age/Gender</th>
<th>Years post-splenectomy</th>
<th>Size of splenunculi</th>
<th>Infectious organism</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reay and Nakonechny\textsuperscript{81}</td>
<td>Case Series</td>
<td>19-year-old female</td>
<td>3 months</td>
<td>unknown</td>
<td>pneumococcus</td>
<td>death</td>
</tr>
<tr>
<td>Reay and Nakonechny\textsuperscript{81}</td>
<td>Case Series</td>
<td>21-year-old male</td>
<td>4 years</td>
<td>unknown</td>
<td>pneumococcus</td>
<td>death</td>
</tr>
<tr>
<td>Holmes et al.\textsuperscript{83}</td>
<td>Case Series</td>
<td>17-year-old female</td>
<td>4 years</td>
<td>unknown</td>
<td>meningococcus</td>
<td>death</td>
</tr>
<tr>
<td>Holmes et al.\textsuperscript{83}</td>
<td>Case Series</td>
<td>27-year-old female</td>
<td>5 years</td>
<td>Estimated 25 g of splenic tissue</td>
<td>meningococcus</td>
<td>death</td>
</tr>
<tr>
<td>Rice and James\textsuperscript{82}</td>
<td>Case Series</td>
<td>12-year-old female</td>
<td>8 years</td>
<td>Most were 2–4 mm in diameter; the largest was 2.6 by 0.8 cm and weighed 3 g; another aggregate was 4 cm by 0.5 cm</td>
<td>pneumococcus</td>
<td>death</td>
</tr>
<tr>
<td>Rice and James\textsuperscript{82}</td>
<td>Case Series</td>
<td>19-year-old male</td>
<td>9 years</td>
<td>42 g splenunculus in the splenic bed, scattered deposits up to 3 cm in diameter; total splenic weight was 92 g</td>
<td>pneumococcus</td>
<td>death</td>
</tr>
<tr>
<td>Gopal and Bisno\textsuperscript{79}</td>
<td>Case Series</td>
<td>11-year-old female</td>
<td>7 years</td>
<td>Several ectopic spleens, the largest being 12 g</td>
<td>pneumococcus</td>
<td>death</td>
</tr>
<tr>
<td>Sher, Block and Gomperts\textsuperscript{85}</td>
<td>Case Study</td>
<td>29-year-old male</td>
<td>17 years</td>
<td>-</td>
<td>-</td>
<td>death</td>
</tr>
<tr>
<td>Widmann and Laubscher\textsuperscript{86}</td>
<td>Case Study</td>
<td>27-year-old female</td>
<td>17 years</td>
<td>More than 40 lesions 0.2–1.2 cm in diameter; 1.2 x 1.0 x 0.8 cm accessory spleen</td>
<td>enterococcus</td>
<td>death</td>
</tr>
<tr>
<td>Sass et al.\textsuperscript{83}</td>
<td>Case Study</td>
<td>37-year-old female</td>
<td>11 years</td>
<td>Multiple nodules, the largest 1 cm in diameter, and a 6 cm diameter spleen remnant; 40 g total</td>
<td>pneumococcus</td>
<td>death</td>
</tr>
<tr>
<td>Breisch and Krous\textsuperscript{78}</td>
<td>Case Study</td>
<td>10-year-old female</td>
<td>10 months</td>
<td>2.1 x 1.5 x 0.9 cm infarcted wandering spleen, 1.5 x 0.5 x 1.5 cm accessory spleen weighing 2 g</td>
<td>pneumococcus serotype 22F</td>
<td>death</td>
</tr>
<tr>
<td>Scully, Galdabini and McNeely\textsuperscript{84}</td>
<td>Case Study</td>
<td>23-year-old male</td>
<td>10 years</td>
<td>The largest nodule was 3.5 cm in diameter; total estimated weight 15 g</td>
<td>pneumococcus</td>
<td>death</td>
</tr>
</tbody>
</table>

**Table 2. Reports of OPSI and accessory spleens**

<table>
<thead>
<tr>
<th>Reference</th>
<th>Article type</th>
<th>Patient Age/Gender</th>
<th>Years post-splenectomy</th>
<th>Size of accessory spleens</th>
<th>Infectious organism</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Iwemark\textsuperscript{89}</td>
<td>Case Series</td>
<td>9-month-old female</td>
<td>9 months</td>
<td>2–3 g</td>
<td>-</td>
<td>meningitis</td>
</tr>
<tr>
<td>Gopal and Bisno\textsuperscript{79}</td>
<td>Case Series</td>
<td>27-year-old male</td>
<td>10 years</td>
<td>4 g accessory spleen</td>
<td>pneumococcus</td>
<td>death</td>
</tr>
<tr>
<td>Navarro and Kondlapoodi\textsuperscript{90}</td>
<td>Case Study</td>
<td>74-year-old male</td>
<td>10 years</td>
<td>14 g total splenic tissue</td>
<td>pneumococcus</td>
<td>death</td>
</tr>
<tr>
<td>Coler\textsuperscript{88}</td>
<td>Case Series</td>
<td>8-year-old female</td>
<td>2 years</td>
<td>3 cm diameter accessory spleen</td>
<td>-</td>
<td>death</td>
</tr>
<tr>
<td>Coler\textsuperscript{88}</td>
<td>Case Series</td>
<td>13-year-old male</td>
<td>7 years</td>
<td>unknown</td>
<td>-</td>
<td>death</td>
</tr>
<tr>
<td>Balfanz et al.\textsuperscript{87}</td>
<td>Case Study</td>
<td>8-year-old female</td>
<td>3 years</td>
<td>1.5 cm diameter accessory spleen</td>
<td>pneumococcus</td>
<td>death</td>
</tr>
</tbody>
</table>
this child’s spleen). Van Wyck noted that splenosis or accessory splenic tissue occurred in 26% of 39 fatal infections he reported.

Traub et al. studied splenic reticuloendothelial function after splenectomy, spleen repair and spleen autotransplantation. They showed that partial splenectomy and splenic repair were better than splenic autotransplantation in protecting against OPSI. They hypothesized that the amount of autotransplanted tissue and location of splenic slices were probably quite important in preservation of splenic function, consistent with animal models. Autotransplanted slices were 30 grams and placed in extraperitoneal pockets. Splenic reticuloendothelial dysfunction was measured using a pocked erythrocyte assay (as gauged by interference-phase microscopy) and erythrocyte clearance using \(^{51}\)Cr-labeled autologous antibody-coated erythrocytes. Splenic dysfunction was indicated by an increase in pocked erythrocytes and less erythrocyte clearance.

The presence of pocked or pitted erythrocytes or Howell-Jolly bodies have been used as signifiers of splenic dysfunction. Observation of such changes on or in the red blood cells, how- ever, does not fully correlate with functioning splenic tissue. Studies reveal that pitted erythrocytes more reliably reflect splenic function than Howell-Jolly bodies. The absence of Howell-Jolly bodies on peripheral blood smear cannot be considered evidence of regeneration of adequate splenic tissue to provide protective immune function.

Physicians should not become complaisant about aggressively treating infections in patients with known splenosis. Efforts should be made to repair or preserve injured spleens and perform partial splenectomy in selected cases of congenital hemolytic anemias.

References

Conclusion

In 2006, Backhus and Bremner suggested that splenosis affords normal immune function based on the absence of Howell-Jolly bodies in a patient with thoracic splenosis. They further stated that their patient was no longer “functionally asplenic and no longer required immediate antimicrobial therapy when he had a fever.” While animal data suggest some amount of splenosis is protective, human case reports and series do not support this conclusion—there is a paucity of data to support full immunologic recovery. Many animal studies and human anecdotal data suggest that splenotic nodules do not regenerate full immunologic defense against infection.

The frequency of OPSI is affected by the post-splenectomy time period and the indications for splenectomy, as well as the patients’ general health. Human observational studies do not show an exact threshold for ectopic splenic tissue mass that would prevent against overwhelming sepsis. Experimental studies that use a model of autotransplantation followed by bacterial challenge do indicate that some, but not complete, protection may occur when a large amount of splenic tissue is autotransplanted.

For protection against infection, an intact spleen is better than a repaired or autotransplanted spleen, while an accessory spleen and splenosis seem to be only slightly better than asple- nia. Surgical or interventional radiologic techniques should be employed to maximize residual splenic tissues in cases of splenic injury. No amount of splenosis should be considered protective against OPSI. Therefore, post-splenectomy patients with signs of infection—even those with known splenosis—should be aggressively treated by their physicians.


50. Kashgari AA, Kashgari AA, Al-M .\n


