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Inadequate detection of accessory spleens and splenosis with laparoscopic splenectomy

A shortcoming of the laparoscopic approach in hematological diseases

We read with interest the recent paper by Gigot et al. [3]. In a prospective series of 18 laparoscopic splenectomies (LS), Gigot et al. show the existence of residual splenic tissue, as demonstrated by denaturated red blood cells scintigraphy and single photon emission computerized reconstruction (DRBCS-SPECT), in up to 50% of cases. They also discuss the difficulties associated with the perioperative localization of accessory spleens and the safety issues related to LS for treatment of hematological diseases.

For normal-sized or slightly enlarged spleens, LS is a feasible and reproducible procedure that has all the advantages of laparoscopic surgery [5]. However, because of port site recurrences following laparoscopic surgery for malignant conditions, concern exists about the risk of tissue dissemination if the spleen capsule is broken and cell spillage occurs. Another problem is that it is difficult to identify accessory spleens (AS) during LS. We recently have found evidence of the existence of residual splenic tissue after LS. However, both problems need to be analyzed separately and compared with the previous experience in open surgery.

Splenic function can be assessed by several means. Image studies can identify fragments of tissue measuring $\leqslant 1$ cm (CT scan, US) [1]. DRBCS can show the existence of tissue able to captate the isotope. SPECT reconstructions may be too sensitive to nonbinded isotopes and therefore offer false positive images. Truly functioning splenic architecture is evaluated by the rate of pitted cells or Howell-Jolly bodies seen in peripheric blood smears. Residual splenic tissue should have enough quantity as well as adequate architecture to restore splenic function and potentially to induce relapsing of the hematological diseases. The treatment of residual accessory spleens after splenectomy for idiopathic thrombocytopenic purpura (ITP) has shown that the disease is cured after accessory splenectomy in only half of the cases [6].

We evaluated spleen function by counting the peripheral pitted cells in a series of 37 LS performed for several hematological conditions. These cases had a mean follow-up of 36 months, and our evaluation was done 2 months after the LS. In all cases but five, pitted cell count was >16%; the lower rate was considered as asplenic [2, 7], and that confirmed the efficacy of splenectomy. We then reevaluated with DRBCS or CT scan 10 patients who had no response (total, <100,000 platelets mm^3) or partial response (<50,000) after LS. In three we found a hot spot by DRBCS; in two, we observed a splenic nodule by CT. Two of them had no pitted cells in peripheric blood; but in the other seven, we could not identify any splenic function by any of the three methods.

One controversial issue is the role of accessory spleens. How can we explain the wide variability in the incidence of AS between series, what is their physiological role, and what is the critical size that will induce relapse of an hematological disease? The incidence of accessory spleens ranges between 0 and 41% in both open and laparoscopic series. In our series, in which we searched carefully for them and opened the omental pouch, the incidence was 12%. On the other hand, reported series of LS for ITP showed a clinical success similar to open series, even though follow-up has not been as long up to now as for open series [3, 4].

Gigot et al. contend that the laparoscopic evaluation of AS is less efficient. However, we seriously doubt that the search for AS during open surgery (a 15-cm subcostal incision for splenectomy for ITP) is any better than the view achieved during LS, where it is possible to have a magnified access to retrogastric pouch and, indeed, to areas that cannot be visualized during open splenectomy, such as the posterior face of the spleen.

The seeding of spilled splenic cells in a high-pressure pneumopertineum during the slightly longer operation is a new and specific problem to LS. In our series, one case, which required conversion due to splenic bed oozing, relapsed ITP, and CT, DRBCS and pitted cell count showed residual splenic function. In the Gigot series, in three of four cases where the spleen capsule was turned out, they found splenic seeding. A more worrisome finding was the presence of positive isotopic scans in cases without AS or capsule rupture.

The spleen has a capsule, and if it is maintained intact, there is no reason for cell spillage. The current LS technique, with a lateral or semilateral approach, allows the surgeon to open the lesser sac, search for the AS, and clip the artery with minimal handling of the spleen. Mobilization of the posterior face of the spleen and stapling of the hilum can be done without damage to the capsule, thanks to the

wide mobility of the spleen that is obtained. Careful exposure with probes, rather than just grasping the spleen and adjacent tissue, may avoid capsules tears. One hypothetical mechanism that must be considered is that residual venous bleeding coming from the spleen after total devascularization can carry splenocytes able to implant in the splenic fossa.

In summary, LS seems to have several advantages over the open approach, with initial clinical results very similar to open series. LS should include a careful search for AS, as well as use of a delicate technique to avoid splenic tears or bleeding. The true importance of AS and the clinical consequences of the risk of splenic spillage are not known. The only way to address these questions, now that there are several centers where LS is currently performed, is through a prospective randomized trial comparing LS to open surgery for ITP, which is probably the most frequent and best-suited hematological condition for this inquiry.

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