may be due, at least in part to decrease in peripheral resistance resulting in stimulation of the renin-angiotensin-aldosterone system causing salt and water retention.[8]

Summary and Conclusions

Fourteen patients with massive splenomegaly and anemia have been studied with reference to plasma volume, red cell mass, hematocrit, portal venous pressure and clinical response to splenectomy. In every patient, the plasma volume was greater than normal prior to splenectomy and in most instances the red cell mass was normal or nearly normal. Thus, hemodilution was a prominent factor in the mechanism of the anemia.

Eleven patients have been studied subsequent to splenectomy and in eight the plasma volume contracted to normal or near normal. Continued elevation of the plasma volume in two of the remaining three may be due to persistent portal hypertension.

Anemia has been altered favorably by splenectomy in eight patients. Progression of the underlying blood disorder was judged responsible for failure to correct the anemia in two of the remaining three.

The immediate comfort afforded by removal of the massively enlarged spleen is striking, and the early clinical results have been good, athough progression of the primary disease has been responsible for early demise in four patients.

These observations support the findings of others that hemodilution due to an expanded plasma volume is a prominent factor in the anemia of some patients with massive splenomegaly. It is concluded that the dilutional anemia associated with splenomegaly is an indication for splenectomy.

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Discussion

DR. JAMES G. DONALD (Mobile): I would like to direct this discussion to the paper by Dr. Davis and Dr. Montero concerning the large splenic cysts.

In this discussion I shall present two patients from the private practice in Mobile of Dr. John Donald and myself in which we have encountered some splenic cysts of a different type from those that were presented by Dr. Davis, which might be interesting to show briefly.

(Slide) This first patient is a 33-year-old woman whom we first saw 3 weeks after the delivery of her fourth child. She had a history of a splenic mass in the left upper quadrant which had been left alone by her family doctor for 6 years.

Three weeks after delivery of this fourth child she suddenly had a severe pain and tenderness in this mass, and came to us for surgical treatment.

We felt that she had, possibly, a pre-existing cyst with subscapular hemorrhage. There was no history of trauma, except incident to the normal delivery and the factors of intraabdominal pressure related thereto.

We operated upon her promptly, and this is the specimen. (Slide) This cyst is on the hilar side of the spleen.

(Slide) On the convex surface is the subcapsular hemorrhage, which caused the increase in size and the pain and tenderness in the mass.

(Slide) This is the cross section of the spleen, which shows the cyst, containing yellowish, turbid fluid, and the clotted subcapsular hematoma—on the convex side. This cyst was lined with an endothelial lining which possibly might be some type of a lymphangiomatous congenital cyst.

It was calcified on preoperative x-ray which was done in 1951 and is not available now.

(Slide) This second patient, a 30-year-old woman, was injured in a skiing accident. She had a contusion, but the conventional scout film following her injury showed this mass in her left upper quadrant. Her injury proved to be insignificant. GI studies and IV pyelograms showed no connection of this to the gastrointestinal or urologic system. Operation was planned. We could feel a mass in the left subcostal area and at operation found that the spleen contained a cyst in the lower pole with two or three small satellite cysts near it. This is on the hilar surface of the spleen.

(Slide) The cross section shows again a calcified, thick wall with an endothelial lining and a turbid yellow fluid in the cyst. The satellite cysts had the same type of material. The pathologist reported lymphangiectasia in the spleen, and thought that this might possibly be one of the congenital lymphangiomatous cysts of the spleen.

DR. RICHARD C. CLAY (Miami): I shall limit my remarks to the presentation of Dr. Davis.

About 16 years ago I saw a 22-year-old man because of 30 pounds of weight loss and an enlarging mass in the left upper abdomen. Six months earlier he had been hospitalized for 19 days in another city with left upper abdominal pain which was never explained. The patient had an enlargement in his left upper abdomen at that time, which diminished. This was not commmented upon by the records from the other hospital.

Three months before I saw him, he had a recrudescence of pain, began to vomit, was able to eat very small amounts, and noticed a progressive and rapid enlargement in the upper abdomen.

(Slide) It was obvious that this young man had a tremendous mass which extended to the iliac crest. X-rays showed normal pyelograms, and the displacement of the viscera which was demonstrated in the film a moment ago.

(Slide) This will give you an idea of the degree of protuberance of this tremendous mass, which disappeared under the costal margin in all directions.

At thoraco-laparotomy, an enormous mass filled the upper abdomen. It proved to be a gigantic pseudocyst secondary to a perforation of a pre-existing cyst of the spleen. There was also another secondary cyst existing in the spleen. Four thousand cc. or so of fluid occupied the space in this cyst, which was partly walled off by spleen, both the upper pole and the lower pole were separated by the cyst, and the usual boundaries of the lesser sac.

(Slide) This will show the spleen itself. On top is an endothelial-like congenital cyst, such as the one that Jim Donald was talking about just a few moments ago. The tail end of tissue off to the right is the wall of the large pseudocyst, and down in the tip of that is the rest of the spleen.

This was treated, obviously, by splenectomy, and in addition the residual pseudocyst was drained to the outside. The patient made a very nice recovery, went home in 8 days, and when seen again 6 months later (Slide), he had gained 30 pounds in weight, back to his normal preoperative weight (Slide), and had certainly changed shape to a considerable extent.

Some years later, when he underwent plastic surgery on the hand, he had maintained this improvement. Here again is a disabling complication of splenic cyst which, fortunately, is amenable to surgery.

DR. JOHN CLARKE (Washington): I direct my remarks to Dr. Davis' paper also on large splenic cysts. We are called upon relatively infrequently to deal with this problem in surgery, and when I reviewed the literature several years ago, having had three cases, I was impressed with the rarity of these reports. After hearing this discussion it is possible that many of these cases are not reported.

At any rate, Dr. Davis' experience of encountering three of these cases in one year or less is probably unique.

I would not attempt to enlarge on his description of the pathologic change or classification of these lesions, and treatment is also not very amenable to discussion, about which no controversy seems to exist. Splenectomy is the treatment of choice, which almost always provides a satisfactory result.

My own experience involved three patients who were treated at the University of Florida, and I am presenting one instance briefly to complete the pathologic spectrum, since Dr. Davis' patients had secondary or false type of cysts and Dr. Donald and Dr. Clay also encountered the so-called neoplastic type.

(Slide) This 2-year-old boy had an abdominal mass that proved to be a splenic cyst.

(Slide) This cyst was intimately associated with the spleen, as you can see on section of the fixed specimen. My microscopic slide, unfortunately, did not survive the trip South, but this was lined with keratinized squamous epithelium,

and so fits into Fowler's classification of a neoplastic or epidermoid cyst.

(Slide) Although the diagnosis of these lesions has been relatively infrequent previous to operation, perhaps with the more frequent use of visceral angiography this is going to be improved upon.

(Slide) This woman had a calcified mass in the left upper quadrant, and the splenic origin of it is demonstrated by this aortogram.

DR. GARDNER W. SMITH (Baltimore) I direct my remarks to Dr. Sandusky's paper. I am not a hematologist and I am by no means an expert on the spleen. However, I did have an opportunity to look at the manuscript, and believe that this concept of splenic anemia is important, not only in elucidating the mechanisms of anemia associated with splenomegaly, but also, perhaps, in a different sense as it applies to cirrhotic ascites. Cirrhosis with portal hypertension has long been known to have a proclivity for hypersplenism, and this occasionally is a reason for surgical intervention; but the spleen has not thus far been involved in the causation of ascites. I would point out, however, that cirrhosis with portal hypertension and ascites fulfills virtually all the criteria which Dr. Sandusky has outlined for dilutional anemia of splenomegaly. The cirrhotic patient with ascites has splenomegaly; he has an increased plasma volume and often a non-iron deficient anemia. Kimber in 1965 showed that this anemia can at least occasionally be dilutional in nature. This patient also has hypervolemia, hyperdynamic circulation with an increased cardiac output and increased oxygen consumption; he has increased renin production and increased aldosterone activity and he clearly has portal hypertension.

I am not prepared to outline a complete pathophysiologic explanation for ascites based upon this mechanism, but I am convinced that these considerations, added to the known association between ascites and the renin-angiotensin-aldosterone mechanism merit serious investigation.

DR. T. BRANNON HUBBARD, JR. (Baltimore): Having encountered only two chronic hematomas or pseudocysts of the spleen over the last couple of decades, I thought they were rather rare, but after listening to all the discussers, I realize that probably we all have seen them. However, we have had two, at least one of which was an unusual occurrence.

A young 17-year-old girl was operated on by a very fine gynecologist for ovarian cyst. He found that this ovarian cyst had a pedicle that extended into the epigastrium, and he called us in.

(Slide) This was a so-called wandering spleen on a pedicle which was found in the pelvis. There was a chronic hematoma of the spleen. It weighed 2,130 Gm.

(Slide) This is the spleen open, showing the old clotted blood and the normal spleen in the upper left-hand corner at about eleven o'clock.

The only etiologic finding for this was that 4 months before this girl had had infectious mononucleosis, and in the progress notes of that admission, at least ten medical students and interns on medicine had enthusiastically stated that the spleen was slightly tender to fist percussion; I think perhaps this is the etiology of her problem.

(Slide) This next patient was a 29-year-old girl who, like Dr. Clay's patient, had lost 70 pounds in weight over the last 6 months and had a mass in the left upper quadrant. I believe you can see the cystic, calcified wall right there, pushing the stomach over here. She had epigastric pain, early filling after eating, nausea, and vomtiting. She had a calcified splenic cyst based upon an automobile accident that had occurred 15 years previously, when she had fallen out of a convertible and hit her lower left ribs.

(Slide) This is the splenic cyst. This is the calcified portion of the cyst. (Indicating) This is normal spleen. The specimen weighed 1,620 Gm.

(Slide) On section, the calcified wall of the cyst is filled with old blood and fatty, lipoid material.

(Slide) This shows the hematoma evacuated, normal spleen is shown through the trabeculations and the calcified lining of the cyst.

DR. B. F. BENTON (Memphis): (Slide) This 68-year-old woman with a lower abdominal mass and urinary frequency, had suffered a contusion of the shoulder 1 year before, when she fell in her garden. We know that she had an enlarged spleen, about two times normal size, at the time of operation 5 years previously. The spleen was noted to be in the normal subdiaphragmatic location.

(Slide) This shows the location of the mass, with both solid and cystic components apparent on physical examination.

(Slide) The pathologist found old, unclotted blood in the cyst, which was diagnosed as a false cyst. The patient made a good recovery, but 1 month after operation developed a mild episode of pancreatitis.

In spite of the rarity of this condition, we must consider it in our differential diagnosis of abdominal conditions, particularly where there has been a history of trauma and lower abdominal mass.

DR. J. M. MONTERO (Closing): In our review of the literature we found that approximately 14% of all the cystic spleens reported were calcified, which, of course, would hardly pose any preoperative problem with diagnosis.

DR. WILLIAM R. SANDUSKY (Closing): In the hypothesis relative to the mechanism, we noted the possibility of a vasoactive substance. We have investigated only one vasoactive substance; namely, bradykinin. Assays have been done on blood from the splenic vein, but apparently there is no activation of this system.