INTRAPARENCHYMATOUS HEMORRHAGE OF THE SPLEEN

HEMORRHAGIC SPLENITIS: INTRASPLENIC HÆMATOMA (NON-TRAUMATIC); APOPLEXY OF THE SPLEEN: FIBRINOUS TRANSFORMATION OF SPLEEN

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Two forms of hemorrhage of the spleen are well known in medical literature, viz.: that which accompanies external rupture, and the so-called blood cysts which are believed to be due chiefly to internal hemorrhage of traumatic origin. By some authors the great majority of non-parasitic cysts are placed in the latter category, and no absolute distinction is made between false and true cystic formations, the last named having a true epithelial lining. Profuse hemorrhage may occur within a true cyst.

But aside from these well-known forms there are others which appear to occupy a position midway between them, which are barely alluded to in standard works. It is understood at the outset that hemorrhagic infarctions, vascular tumors, aneurisms and perhaps other conditions are not to be considered in this connection. But few cases are on record, and it is unfortunate that these are very imperfectly described and illustrated. There is an absence of microscopic findings and of blood counts before operation. The case reports refer to intrasplenic hæmatomas, hemorrhagic splenitis, encysted hæmatomas, parenchymatous or diffuse hemorrhage, intracapsular hemorrhage, apoplexy of the spleen, etc. Generally speaking the extravasation of blood is large, often extreme. The whole organ, or one of the two poles, may be involved. The bleeding is apparently of the gravescent type in certain cases, oozing taking place gradually and compressing the splenic tissue. This is well shown in the character of the blood seen in some cases. We may see side by side fresh fluid and clotted blood, layers of unorganized fibrin and organized fibrin. In a so-called primary blood cyst in which escape of blood is limited to a single extravasation in one locality, the blood as a rule is quickly absorbed and the cyst which forms later contains only serum and blood detritus. In rare cases this absorption does not take place, or rather perhaps the lesion is seen at a very early stage before absorption could occur. Whether an encapsulated hæmatoma can become a true cyst seems to be undecided.

Much confusion is due to the fact that some of the interior hemorrhages of the spleen are really subcapsular ruptures. In any rupture of

the organ the blood is apt to accumulate beneath the capsule before rupture of the latter occurs, and the tear in the capsule only allows a part of the pent-up blood to escape into the peritoneal cavity. In a certain number of cases the capsular laceration does not occur until some time after the injury (two-stage rupture), and in a few cases does not occur at all. There is no doubt that some intrasplenic hemorrhages so-called are of this character. The accumulation of blood under the capsule may then compress the spleen and in the end the results will not differ from that produced by a purely intrasplenic hemorrhage. It has been the aim of the author to exclude all such cases from consideration.

Of special literature the chief authority is Camus's thesis on "Traumatic Intrasplenic Hæmatoma," which reproduces a large number of cases from literature. The great majority are cysts of traumatic origin and there are also a number of cases of subcapsular hemorrhage. But few cases are examples of internal hemorrhage in our conception of the term.

Of a considerable number of cases in literature which bear a close resemblance at first sight to the author's case, all have been rejected but four, either because of scantiness of data or because they were clearly subcapsular hemorrhages. Of the five cases not one gave any history of malaria, and the obscure nature of the disease in the five may have had some connection with the nature of the hemorrhage. A brief outline of the cases is appended.

Routier's patient was a woman of twenty-four, in excellent health aside from an obscure enlargement of the spleen of some years' duration. She went through a pregnancy without accident. Eight years after the original enlargement, the organ, hitherto quiescent, underwent further swelling and the size of the spleen then increased until it had to be removed as a mechanical nuisance. The organ weighed 3500 Gm. and its tissue had been partly replaced by fibrin and clots, chiefly at the lower pole. In the entire pulp could be seen fresh foci of hemorrhage and small blood cysts. The case was so unique that the author has no explanation to offer. The secondary enlargement of the spleen was apparently due to a diffuse parenchymatous hemorrhage.

A case of Jordan's was quite similar to the preceding. Jordan speaks of it as a "blood cyst," but R. H. Fowler in commenting on the case terms it a hæmatoma. The patient was a woman of forty-six without history of infection or trauma. The spleen had been enlarged for seven years, and was extirpated for mechanical reasons. It weighed nearly 9 pounds and on section hardly any parenchyma was visible—a connective-tissue wall contained only gelatinous masses of fibrin.

Martyn's case is altogether peculiar. He describes it as one of "acute hemorrhagic splenitis." There was no history of splenic trouble and the patient was

healthy until the third month of her fourth pregnancy, when she became acutely ill with symptoms which suggested acute pancreatitis and peritonitis. Laparotomy showed error in diagnosis. There was some blood in the lesser peritoneal cavity. The woman became worse and aborted. The abdominal wound was tamponed and the uterus curetted. Later the tampon was removed. The patient continued to grow worse until death set in. The spleen was found enlarged, and both subcapsular and parenchymatous hemorrhage was present. There were several large hemorrhages in the parenchyma, and several communications existed between these and a subcapsular clot. The pulp of the spleen was chiefly soft and necrotic. There was some normal pulp. No round-cell infiltration was found. The case is of special interest as showing that foci of internal hemorrhage may be associated with a subcapsular clot. A slight rupture of the capsule at this point had caused bleeding into the lesser peritoneum.

Di Bernardo's case is as follows: Male, aged sixty years, negative history. Sudden pain in splenic area followed by evidences of tumefaction. Two months later examination and puncture led to the diagnosis of hæmatoma. Laparotomy. Removal of two litres of bloody fluid through a small opening. The entire spleen had been disintegrated and altered by accumulation of blood within the capsule.

We are now in position to report our personal case and compare it with the preceding, with a view of learning whether the five cases represent a separate type of affection or are atypical throughout. As will be noted the author's case is very fully reported, with blood counts and microscopic findings.

CASE REPORT.—A. M., thirty-six years old, a native of Illinois; farmer; married, has two healthy children; height, 5 ft. 9 in.; weight, 150 pounds. Referred by Dr. Essex.

Family History.—Father died at age of seventy-three; cause unknown. Mother died of blood poisoning, aged about sixty years. Had one brother who had epithelioma of the lip, which was cured by extirpation. Has two brothers and three sisters, living.

Personal History.—Is without interest until three years ago when he was "threatened with malaria." No treatment was taken and no malaria developed. He was slight of build but never had to consult a physician, excepting for a bad tooth (pyorrhœa), about three years ago (this probably accounts for his threatened malaria), although for six years he had had periodical attacks of headache in right temple, for which no treatment had been sought.

Present Illness.—Patient had complained of feeling tired and weak for about one week; then seventeen days ago, while pulling weeds at 2 P.M., he was seized with a sudden pain in the pit of his stomach. Not a sharp and severe pain, but dull and continuous. He continued his work through the day and all the following day, and until noon of the third day. The pain was continuous and gradually increasing in severity, and on the evening of the third

day it was severe enough to require the administration of morphia, hypodermically. Following this he required from two to six hypodermics daily, and the last few days, hyoscine had been substituted for morphia. With nausea and vomiting the pain gradually worked its way to the left and beneath the edge of his ribs. No hæmatemesis was observed.

Physical Examination.—Skin pallid and moist, face drawn, thin and anæmic; tongue coated; pulse 86 and of fair volume; temperature 98.8; respiration 30 and shallow. Head: Features thin, eyes normal, teeth fair; tonsils, thyroid and pharynx negative. Chest: Dulness and decreased fremitus below the fifth rib on left side. Right side negative. Heart: Sounds normal; apex beat, 1 inch to left and above normal position. Abdomen: Left hypochondrium distended and very tender for 2 inches below edge of ribs. Other abdominal organs normal.

URINALYSIS

Color	Dark amber.
Reaction	Acid (100 per cent.)
Specific gravity	1024
Albumen	½ of 1 per cent.
Sugar	Negative
Indican	Present, large amount
Urea	0.03
Mucin	Present
Bile	Trace
Microscopic	Granular casts, few cells epithelium, leucocytes, crystals, calcium oxalate, bacteria (few).

BLOOD EXAMINATION

Red	3,176,000
Per cent. red cells	62
Hæmo index	.5
Hæmoglobin	40 per cent.
White blood-cells	130,000
Polymorphonuclear	71 per cent.
Large mononuclear	20 per cent.
Small mononuclear	8 per cent.
Eosinophiles	1 per cent.

Red cells, rouletted.
Many normoblasts present.

Diagnosis.—Hemorrhage into spleen, and decision to operate.

Preparation.—Hypodermic of hyoscine, gr. 1/100, morphia sulphate, gr. ¼, one-half hour before operation.

Operation.—Ether anæsthesia. Incision left rectus. Findings: Enlarged, tense spleen, adherent to parietal peritoneum,

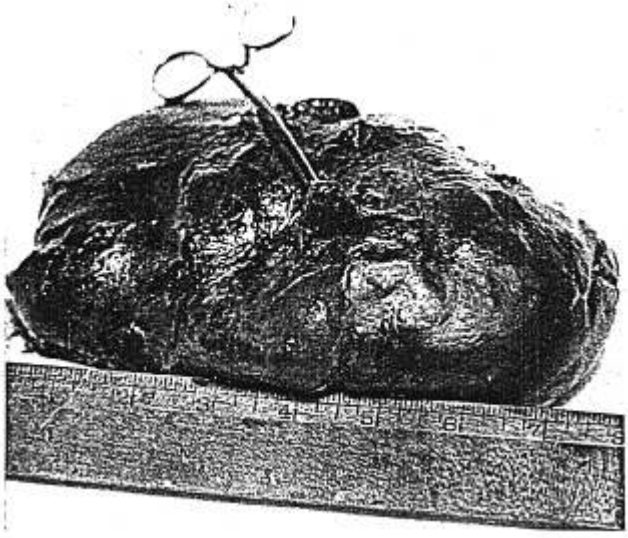

FIG. 1.—Showing condition of spleen after removal; forceps thrust into blood cavity.

FIG. 2.—Removed spleen laid open.

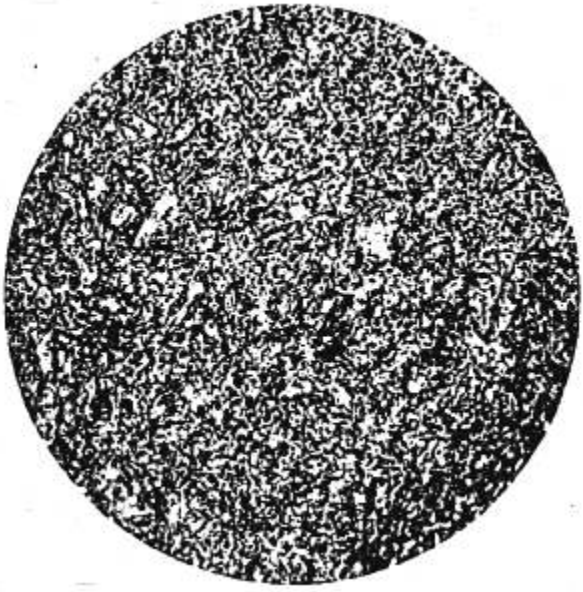

FIG. 3.—Section of spleen, showing round-cell infiltration and increase of connective tissue.

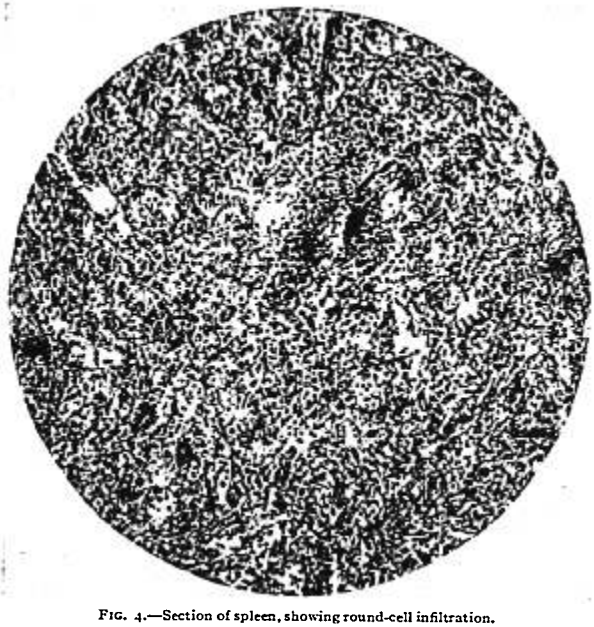

FIG. 4.—Section of spleen, showing round-cell infiltration.

colon, stomach, pancreas and diaphragm. Incision enlarged upward with resection of 2½ inches of eleventh and twelfth ribs for increased space. Spleen accidentally ruptured, allowing approximately 2000 c.c. of blood to escape. The spleen was delivered and pedicle clamped and ligated. The cavity mopped dry and packed with gauze strips. Weight of spleen three hours after removal, 1344 grammes, plus blood (estimated) 3344 grammes.

Unfortunately the hospital records covering the period between the time of operation and his death are not obtainable, but my recollection is that he did not suffer from surgical shock, his temperature did not run high and for three days his condition seemed favorable, but on the fourth day he showed signs of exhaustion, the symptoms becoming more pronounced, until death ensued on the fifth day from general exhaustion, superinduced by the prolonged period of pain and the starvation preceding his operation.

Pathological Findings (as reported by the Columbus Laboratories).—Sections made from spleen show that there is an increase in connective tissue and numerous round-cells (polymorphonuclear). There is no focal necrosis or other change. Our opinion is that this is splenitis simply. Eosinophilia and other special cells are absent.

It is of interest to note the changes in the blood picture for the five days between the time of operation and his death. Particularly the rapid fall in the white count and persistent polymorphonuclear leucocytosis.

Blood examination	First day	Second day	Third day	Fourth day	Fifth day
Hæmoglobin.....	.....	38 per cent.	36 per cent.	35 per cent.	40 per cent.
Red cells per cm....	.....	3,032,000	3,336,000	3,256,000	3,424,000
Red cells.....	.....	60 per cent.	66 per cent.	64 per cent.	68 per cent.
Corps. Hemo index	.....	.5	.4	.4	.4
White cells per cm..	158,000	150,400	118,000	106,400	100,000
Differential					
Polymorphonuclear	.....	60 per cent.	100 per cent.	88 per cent.	92 per cent.
Large mononuclear.	.....	34 per cent.	.....	1 per cent.	4 per cent.
Small mononuclear..	.....	6 per cent.	.....	11 per cent.	4 per cent.
Eosinophiles.....	.....	0	.....	0	0

If we accept the statement of Anders and Boston that in over 80 per cent. of all cases of splenitis the symptoms are indefinite or absent, we can reconcile ourselves to the belief that this was one of the 80 per cent. of symptomless cases in which the disease had insidiously affected the splenic artery until its walls became friable and so weak that the slight effort of pulling weeds ruptured the artery, and we had a rapidly

forming hæmatoma, the blood extravasating through the pulp with its subsequent pressure symptoms and enormous distention of the capsule and perisplenic adhesions.

This would be an easy and perhaps satisfactory explanation of the condition found, if we did not attempt going farther in our efforts to discover why he had splenitis in the first place. Here we confront a stone wall.

A reference to his history shows no past illnesses, excepting pyorrhœa three years before. We then have what appears to be a primary simple splenitis.

ANALYSIS OF FIVE CASES

History of Trauma.—In no case was there a history of any marked traumatism; in the author's case stooping over may have acted as a minimal traumatism.

History of Infection.—None.

History of Pain, Shock, Etc.—In three of the six cases there was pain at the onset—Di Bernardo's, Martyn's and the author's. In the last two it was severe, and led to fatally ending symptoms. Both Martyn and the author regarded their cases as examples of "hemorrhagic splenitis."

History of Prolonged Splenomegaly.—This was absent in the cases of Di Bernardo, Martyn and the author, and present in a high degree in the cases of Routier and Jordan (splenectomy having been done for mechanical reasons only).

Thus far in the analysis the cases of Routier and Jordan compare very well, and there are strong points of resemblance between the cases of Martyn, Di Bernardo and the author (all recently reported).

The cases of Routier and Jordan are examples of a latent hemorrhagic process of a highly diffuse character, in which the organ was almost destroyed. There was no symptomatology. Jordan's case seems to have been furthest advanced, as hardly any parenchyma remained. Enough was present in Routier's case to show that the process was focal in character, the smaller foci probably coalescing in the pole first attacked. Since small foci of hemorrhage may be seen in chronic splenitis, these cases might be termed chronic hemorrhagic splenitis.

There remain for comparison the three related cases of Martyn, Di Bernardo and the author. In none was there any suspicion of any serious trauma (mere stooping over in the author's case), nor any history of infection or splenomegaly. Sudden, severe pain ushered in the condition in spleens doubtless diseased but not known to be so.

After the initial symptoms evidences of an enlarging spleen were noted. In the cases of the author and Martyn the pain was not in the splenic region, but rather in the middle line. Di Bernardo's patient does not appear to have been a sick man, he had no complications. The entire (enlarged) spleen had been almost transformed within two months into a sac of bloody fluid, yet no rupture had been caused. This suggests a slow, gravescent type of bleeding. The author's case resembled the preceding in this respect, for only seventeen days after the onset of the disease, 2000 c.cm. of liquid blood were accidentally evacuated from the spleen. Martyn's case agreed with the author's in the severity of the symptoms and fatal termination. Both Martyn and the author looked upon their cases as examples of splenitis, but Martyn calls his case acute splenitis, a term hardly compatible with the findings. An acute septic abscess of the spleen may be accompanied with hemorrhage, but this case was not of that stamp.

As already stated, there are numerous reported cases which are comparable with the preceding in certain respects.

Two are very old cases incompletely reported. Sangalli describes a case of copious extravasation of blood in a hypertrophied spleen without any history of infection. The splenic tissue was permeated with small areas of capillary hemorrhage. Verga describes an autopsy case of fibrinous transformation of the enlarged spleen. Heurtaux describes a case of an enormous blood cyst in a woman of twenty-seven, following an injury eight years previously. Nine litres of thick altered blood were drawn off. The lesion, however, seemed to be a true cyst, and was readily cured by marsupialization and drainage. Similar cases could be cited. In this connection it must not be forgotten that profuse hemorrhage may occur secondarily in a cyst with serous contents.

A case which was originally thought to possess points of marked resemblance to the author's was that of Solieri. Solieri's case showed notable differences; there is a possibility that it represented a subcapsular rupture. This point is not made sufficiently clear. The patient, aged forty-eight, was an old malarial subject and had just experienced a two-day attack. Upon sneezing he at once experienced pain and shock, suggesting a sudden rupture. The symptoms disappeared but the spleen enlarged steadily for ten weeks. An exploratory incision revealed a spleen tense with blood, which was allowed to escape through a buttonhole opening. The cavity was then closed. The author regards his case as an encysted hæmatoma.

The fact that the splenic tissue aside from the collection of blood was intact and that it was not necessary to extirpate the organ is sufficient to exclude this case from the group cited.

In regard to the real nature of intrasplenic hemorrhages, there are several causes which can account for part of them—as a central laceration, rupture of a diseased blood-vessel, hemorrhage into a pre-existing

cyst. None of these causes, however, can account for diffuse parenchymatous hemorrhage which is most readily explained by the view of a hemorrhagic splenitis, such as is known to occur at times on a relatively small scale. In a large number of sections of extirpated spleens at the Mayo Clinic we find mention of chronic splenitis with small foci of hemorrhage, and there seems to be no reason why this process may not occur on a larger scale. At the same clinic it is admitted that not a few enlarged spleens cannot be classified because of the total absence of any causal factors or associated lesions.

The term splenitis seems to have disappeared largely from the nomenclature. Acute splenitis, if it means anything at all, means a condition terminating in abscess, especially in general sepsis. It is said that septic splenitis of this sort may be hemorrhagic. Aside from this type, diffuse splenitis at its outset is usually termed tumefaction or congestion, and eventually there should be a small cell proliferation. Chronic splenitis has as a synonym fibrosis of the spleen, a condition due to metamorphosis of the new-formed connective tissue.

So great an authority as Baccelli does not apparently recognize simple splenitis as a cause of extravasation of blood. He holds that in a diseased spleen hemorrhage may occur from traumatism, while in non-traumatic cases the efficient cause is a varicose dilatation of the blood-vessels, such as occurs in typhoid and malarial spleens. Other authorities not quoted fail to mention such a condition as hemorrhagic splenitis. In the report of the author's case the pathologist found evidences of simple splenitis, while Martyn's case, termed acute hemorrhagic splenitis, was apparently an acute necrosis. As far as can be made out the spleen in Di Bernardo's case was the seat of a primary degenerative process, while in the remaining cases there is no reference to splenitis or necrosis, but normal splenic tissue was preserved in places. The very extent and long duration of these cases doubtless made it difficult to determine the exact nature of the morbid process involved.

The only conclusion we can reach is that extensive intrasplenic extravasations are very rare and not associated with any one special morbid process. No doubt in the past they have been regarded as false blood cysts, although the latter have almost invariably been reckoned as of traumatic origin, and in the vast majority of cases they have occurred in infected spleens. These two factors have been practically absent in the material we have analyzed. The slow and progressive as well as the diffuse character of the process is in evidence in several of the cases not only clinically, but pathologically.

The author has studied a number of recently reported cases of external rupture of the spleen in the hope of finding some basic resemblance to the cases detailed in the paper. In the traumatic cases violence was always marked and often extreme, while spontaneous ruptures practically always occurred in the presence of severe infections.

In conclusion the author acknowledges the assistance of Dr. Edward Preble, of New York City, in collecting the references and arranging the material for the paper.

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