Splenic Rupture: With Consideration Of The So-called "Spontaneous Rupture Of Normal Spleen"

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Among the acute intra-abdominal conditions splenic rupture is one of the most dramatic and dangerous. The surgeon must use all his clinical experience in making an accurate and prompt diagnosis of the condition in view of the imperative need for adequate surgical treatment.

The history of splenic surgery has been traced back to ancient times (23-79 B.C.) when Pliny related that splenectomy was performed in an effort to improve the wind of runners. The earliest ruptures in the world historically have been ascribed to the Chinese ability to punch the enlarged spleen, and for a considerable period of time this was a frequent mode of execution.

The credit for the first splenectomy has been given to Zicarelli, a Neapolitan surgeon, who successfully performed the operation, assisted by Fioravantini, on the wife of a sea captain affected with splenomegaly due to malaria.

For about 300 years (1549-1809) only 17 cases of splenic rupture were reported. In 1881 the condition was still considered a very rare entity. In the latter part of the 19th century, however, the surgeons considered the spleen to be as vulnerable an organ as the heart because of its vascularity.

Baudet, in 1902, gave the classical description of the clinical picture of delayed rupture and described the so-called “quiescent period” after trauma, which has become known as the “latent period of Baudet”, and which McIndoe (1932) described in detail as the so-called “delayed rupture of the spleen after trauma”. McIndoe collected the first 45 cases of this clinical entity in 1932. Since that time many reports have appeared in the literature dealing with the general subject of splenic injury, traumatic, delayed and spontaneous rupture. Late in 1943, Zabinski and Harkins published a review of the world literature on the subject of delayed rupture. By that time 179 cases had been collected. In 1955 Fultz and Altemeier, of the Cincinnati General Hospital, added 17 more cases. More recently (1956) Terry, Milton and Howard, reported 102 consecutive cases of splenic rupture over a period of ten years. Very recently (January, 1958) Orloff and Peskin reported 28 acceptable cases of spontaneous rupture of normal spleen, and 48 as dubious or unacceptable. At the Henry Ford Hospital from 1946 to 1955 a total of 23 cases of ruptured spleen have been seen, six of them classified as spontaneous rupture, of which five were apparently normal at the time of surgery or autopsy.

TRAUMATIC IMMEDIATE RUPTURE

The diagnosis of ruptured spleen when trauma has been present is usually entertained especially when there has been injury to the left side. Generalized abdominal pain, with tendency to localize usually to the left upper quadrant and shoulder
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pain, left, which is most often due to diaphragmatic irritation and known as “Kehr’s sign”, are present in about 50% to 70% of the cases. Tachycardia hypotension, nausea and syncope may occur concomitantly with the acute blood loss. Splenic rupture would be suspected with the fulfillment of the following criteria:

1. history of left sided chest or abdominal trauma
2. symptoms and signs of peritoneal irritation
3. clinical picture of acute blood loss

In most cases the white blood count is elevated and the hemoglobin is depressed. The value of paracentesis has been a subject of controversy. Roentgenological studies offer little help.

The correct diagnosis is accomplished only in 25% of the cases (Roettig11). When associated injury to other organs occurs, such as liver or kidney trauma, there is more difficulty in establishing the correct diagnosis, although the severity of the signs and symptoms is so marked that the surgeon is prone to act quickly, with the result that too many of the diagnoses are made at the operating table.

DELAYED RUPTURE OF SPLEEN AFTER TRAUMA

The possibility of delayed splenic rupture must be kept in mind in all patients sustaining injury to the left side of the chest and abdomen. McIndoe states in this regard that the patient who gradually exsanguinates should not be included in the category, even though the duration of symptoms is more than 48 hours. The postulates of McIndoe have been followed in all the English literature in regard to the proper classification of delayed rupture.

The symptoms are those previously described. The history of trauma is obtained in all cases. What must be mentioned here is that in the quiescent period a false sense of security may be initiated in the surgeon by the recession of the primary signs. During this quiescent stage roentgenological studies should be made. Wang and Robbins14, were able to make a diagnosis in 50% of the cases from x-ray findings. In order of frequency they consist of:

1. complete or partial loss of the splenic outline
2. enlargement of the area usually occupied by the spleen
3. medial displacement of the fundus of the stomach
4. downward displacement of the splenic flexure of the colon
5. slight elevation of the left hemi-diaphragm
6. loss of the outline of the kidney and psoas muscle
7. irregularity or localized indentation of the greater curvature of the stomach

This study is more accurate and precise when a small amount of thin barium is given by mouth through a Levine tube. The period of delayed rupture may last
from 48 hours to as long as a month. Patients should be observed carefully for a period of one month at least after the initial trauma. When the rupture occurs with the patient hospitalized prompt and adequate treatment may usually be undertaken. When it occurs with the patient outside, sometimes far away, a goodly number of these patients will arrive in a most precarious condition and the situation will be out of hand. Because of this, we believe close observation of the patient for a prolonged period of time is indicated.

**SPONTANEOUS RUPTURE OF THE NORMAL SPLEEN**

This term is used less frequently in recent years since the knowledge of the condition has deepened and clinical histories are taken in more accurate detail. Because of this it has been possible to elicit a history of trauma in the majority of cases. In addition, when these specimens are re-examined microscopically, a degree of pathology has been found in those previously reported as a normal spleen. A goodly number of the earlier cases reported in the literature as spontaneous rupture of a normal spleen were dubious even to the contemporary pathologists. Wohl agrees in the physiological aging of the spleen. He believed that after the age of thirty-six 50% of the spleens showed degenerative change but that these changes should be considered as normal and explains the spontaneous rupture on this basis.

In 1937 Zuckerman and Jacobi refuted Wohl's theory on the basis of the following postulate. If 50% of the normal spleens undergo degeneration, sufficient to account for rupture of spontaneous type, why then are not more frequent spontaneous ruptures seen? They accordingly believed that these spleens classified previously as normal were pathologic in some manner at certain points, probably very close to the place of fracture. A diseased spleen is more subject to trauma and to rupture than is the normal spleen. Even a mild degree of trauma may be sufficient to precipitate rupture and more frequently in the patient with a spleen diseased by malaria, infectious mononucleosis, leukemia, congestive splenomegaly, Gaucher's disease, aneurysm of splenic artery, or pregnancy. The latter is particularly important because of easy confusion, particularly if present at the moment of labor. The mortality of spontaneous rupture of spleen in pregnancy is recorded as from 50% to 60%.

Susman in 1928 collected 6 cases of spontaneous rupture of normal spleen. In 1930 Zuckerman and Jacobi, after a critical review of the cases reported up to that time, admitted 20 only. In 1939 Rankin, from Philadelphia, reported one case. Jones, from England, in 1944 and Nicol, in 1952, each reported one case.

A number of factors have been considered as predisposing and others as precipitating in regard to the possible etiology of these splenic ruptures. The Susman postulates are of the most interest. He states that from his observations the following occur:

1. softening of all the structures of the spleen
2. congestion of portal vein and radicles and inability of the narrow splenic vein to accommodate itself
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3. blood being thus forced between the spleen and investing peritoneum which finally gives way

4. perisplenic adhesions which eventually, by fixing the organ, predispose to rupture

In one case he described some arteriolar degeneration and suggested that the rupture is secondary to intrasplenic hemorrhage, but it is really unlikely that only one common cause for the hemorrhage will be found to explain all the cases. Failure in development of the ligaments which maintain the spleen in position has been pointed out as another etiologic factor. J. G. Whitcomb, from our Surgical Research Laboratory, was unable to obtain rupture by intrasplenic injection of blood. The author made a study of five dogs. In two of them ligation of the splenic vein was done in order to obtain congestion. No results are achieved other than hypertrophy of the short gastric veins. Three other dogs were subjected to ligation of all the veins of the hilum. In these only a shrinkage of the organ was obtained without congestion. Actually we would say that the existence of such an entity must be considered very dubious and that only time will give a final answer. The explanation of the cause of splenic rupture will be found in the organ itself, seen by a pathologist who is thoroughly familiar with diseases of the spleen.

A word must be said about the differential diagnosis of a spontaneous rupture. There may be confusion in certain cases, not often but often enough to consider, with two clinical entities of medical type, myocardial infarction and angina pectoris. A sudden onset of chest and shoulder pain with some radiation to arm, tachycardia, cyanosis, dyspnea, marked drop in blood pressure, a pale, cold and clammy patient often semi-stuporous and unable to give a coherent history, all warrant consideration of the heart as the site of origin. An electrocardiogram is obviously indicated in these instances and in most will resolve the problem.

From the review and summary of our six cases of spontaneous rupture we will get a more complete idea of the uncertain situation in which a surgeon may find himself.

Case No. 1. (D.R., 746283, white, male, age 57)

This patient was observed for one year in the out patient department because of marginal ulcer after gastrojejunostomy. He was admitted on 5-31-55 for elective surgery of this condition. Ten days prior to operation (while undergoing observation and medical therapy in the hospital) he developed shoulder and abdominal pain during the night following a liver punch biopsy. The symptoms were first ascribed to the previous procedure; however, the next day pain increased, spreading over the entire upper abdomen and lower chest. Blood pressure dropped to 90/70 mm Hg. Pulse was 120. Hemoglobin dropped to 10 grams. His oral intake was restricted and he was given supplementary blood and parenteral fluids. Several transfusions were required to raise the hemoglobin to 13 grams prior to surgery. On opening the abdomen 1000 cc of dark, old blood were encountered. Hematoma of the spleen was noted. A splenectomy was done with a take-down of the gastrojejunostomy and gastroduodenostomy at the same time. Recovery was uneventful.
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Case No. 2 (M.C., 198905, white, male, age 47)

This patient was admitted to the Henry Ford Hospital on 2-15-46 for medical treatment of a chronic ulcerative colitis. This being unsuccessful he was transferred on 3-8-46 to the surgical service, at which time a phlebothrombosis of the left leg was subsiding. Ileostomy was performed, and on 3-13-46 thrombotic manifestations occurred in the right leg. The temperature was elevated and there was tachycardia with the onset of nausea and vomiting on 4-3-46. The edema of both extremities was classified as "phlegmasia alba dolens". On 4-22-46 there was suspected involvement of the iliac vein by the thrombotic process. The tachycardia was persistent. There was severe dyspnea, increase in circulation time and elevation of venous pressure. The patient was digitalized. On 5-11-46 there was hemoptysis; he was weak, nauseated, vomited and in frank congestive failure. Oxygen was given nasally. Hemoglobin had dropped to 7.4 grams. From 5-11-46 to 6-11-57 he received nothing by mouth, but plasma, blood and fluid replacements were given. Patient became semi-stuporous,

Figure 1

Case No. 2. M.D. 198905. 170 x.
Trabeculae with degenerative staining characteristics. The sinusoids are markedly enlarged with peripheral blood in different stages of decomposition.

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pulseless, blood pressure became unobtainable and death intervened. Autopsy showed “2000 cc of blood in abdominal cavity, thrombosis of iliac vein and vena cava, pulmonary emboli bilateral; spleen with large subcapsular hematoma.” Pathological examination showed “Spleen weight 660 gm. Microscopic: Pulp space and sinusoids are markedly distorted, with large amounts of peripheral blood in different stages of decomposition. Trabecular elements, as well as the stromal, are minimal and show degenerative staining characteristics. The capsule shows no stromal elements, only blood. Sinusoids markedly engorged. In no portion was seen normal spleen.”

Case No. 3. (D.L., 541911, white, male, age 58)

This patient was seen in our Emergency Room on 5-26-55. The previous night he developed left upper quadrant pain which became more severe at 12:47 a.m. There was no nausea or vomiting. No history of trauma or gastrointestinal disease was elicited. Physical examination revealed marked tenderness in the left upper quadrant with possible presence of a mass. Rectal examination was negative. Temperature was 99°, pulse 92, respirations 20, blood pressure 120/80 mm Hg. White blood count was 14,000, hemoglobin 11 grams. Electrocardiogram was negative. Urinalysis was negative. Films of the abdomen showed questionable free air under the diaphragm. Spleen shadow was enlarged. Kidney shadow was not seen. A diagnosis of perforated

Figure 2

Infiltration of trabeculae by mononuclear lymphocytes. Giant cell granuloma formation.
ulcer was made, with rupture of spleen as a second choice. The patient was taken to the Operating Room and a large splenic hematoma was found. Splenectomy was performed. Pathological examination revealed “splenomegaly, due to infiltration of fibrous trabeculae by mononuclear leukocytes resembling lymphocytes.” These slides were reviewed with Dr. Rebuck and it was reported that “fibrous splenic trabeculae smaller and less frequently present. There are large mononuclear leukocytes resembling lymphocytes. Malpighian corpuscles are increased in size due to increased number of cells; one of the lymphomas should be suspected. Conclusion: 1. splenomegaly due to infiltration of fibrous trabeculae by mononuclear lymphocytes; 2. giant cell granuloma.” Patient made an uneventful recovery.

Case No. 4. (J.F., 311047, white, male, age 49)
This patient was seen in the Emergency Room on 12-13-46, complaining of pain in the left shoulder, sudden in onset, which radiated to the elbow, chest and abdomen.

Figure 3

Case No. 4. 311047 F.T. 170 x.
Granulomata formation in the pulp. Sinusoids are dilated with hyperplasia, of the lining cells. lining cells.
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On admission the pain moved to the epigastrium. No previous history of ulcer or angina was obtained. On physical examination the patient appeared to be acutely ill. He was cold, perspiring profusely, and there was slight cyanosis of the skin of face and lips. Heart was regular; tones were normal. Lungs were clear to percussion and auscultation. There was a generalized abdominal tenderness, more marked in the left hypochondrium, and a rebound tenderness referred upward from the left abdomen. Temperature was 96°, pulse 60, blood pressure 80/46 mm Hg., white blood count 12,900. Films of the chest and abdomen were negative. A tentative diagnosis of perforated peptic ulcer was made. On operation a large subcapsular hematoma of the spleen was found with intra-abdominal hemorrhage. Splenectomy was performed, 1500 cc of blood being given during the surgical procedure. Patient’s recovery was uneventful and he was discharged in ten days. Platelet count was normal on discharge. Pathological examination, at that time, revealed a normal spleen. On reviewing the slides Dr. Rebuck noted “multiple granulomata scattered through the pulp with necrosis, marked hyperplasia of the cells lining sinusoids and dilation of sinusoids beneath the capsule.”

Case No. 5. (B.T., 545287, white, male, age 32)

This patient was seen for the first time on 7-13-48 with the main complaint of chest and abdominal pain which had begun at 4:00 a.m. the morning of admission. At that time he was awakened by severe, constant, epigastric pain which radiated to the left shoulder. This was accompanied by some nausea. He had two episodes of diarrhea but no melena. The pain then became intermittent with radiation into the right shoulder and left cervical region and was aggravated by respiration. There was no past history of upper gastrointestinal symptoms. Physical examination revealed a well developed, thin male lying still in bed obviously in severe pain. There was a right pararectal scar from an appendectomy eleven years previously. Temperature was 98.6°, pulse 110, respirations were rapid and shallow. Pain, worse on deep inspiration, was noted in the left lower costal region. Chest was clear to percussion and auscultation. Blood pressure was 130/80 mm Hg. There was no abdominal distention; peristalsis was present; no masses were present. Except for a bronze colored pigmentation of both extremities the remainder of the examination was negative. Following admission the patient’s condition changed. The abdomen became board-like with marked rebound tenderness in the upper abdominal muscles. There was nausea and vomiting. A Levine tube was inserted, and suction was initiated. X-rays of the chest and abdomen were negative. Hemoglobin was 10.9 gram; white blood count was 11,850. A diagnosis of perforated ulcer was made. On surgery a ruptured spleen was found with 1000 cc of fresh and clotted blood present in the peritoneal cavity. Splenectomy was done. Spleen weighed 515 gm. Pathological diagnosis was “cavernous hemangioma of spleen.”
Case No. 5. B.L. 545287. 170 x.
Cavernous hemangioma of spleen. The blood filled cystic space is lined by distinct endothelium. Small sinusoids are filled with RBC's.

Case No. 6. (N.W., 525555, negro, male, age 58)

This patient was admitted to the Emergency Ward on 11-24-47 with the complaint of chest pain and upper abdominal pain, two hours after onset. A co-worker stated that he had complained of pain in the chest with radiation to the neck and left arm. The patient was cold, clammy, semi-stuporous, cyanotic and pulseless. The blood pressure dropped from 80/40 mm Hg. to unobtainable on admission and examination revealed cyanosis of the extremities. Heart tones were normal, with no murmurs or gallop. The abdomen was tender, with the tenderness more marked on the left side. His dyspnea became progressively worse and shortly after admission the patient expired. A tentative diagnosis of massive myocardial infarction was considered. Coroner's autopsy showed "rupture of spleen with massive intra-abdominal bleeding".

As can be seen by this last case, confusion with a massive myocardial infarction clinically may very easily arise. An electrocardiogram is invaluable in these cases.
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SUMMARY
A brief review of the history of surgery of the ruptured spleen is presented with a discussion of the clinical manifestations of the immediate type of traumatic rupture and the delayed syndrome. The so-called “spontaneous rupture” is evaluated. The clinical importance of this syndrome is stressed in a warning to the medical man and the surgeon. A review of six cases seen at Henry Ford Hospital is done, with special emphasis placed on diagnosis and pathological findings.

CONCLUSIONS
1. Immediate (traumatic) rupture of the spleen is more frequently diagnosed.
2. The delayed type of rupture is diagnosed in only 50% of the cases.
3. Spontaneous rupture of the spleen is the most difficult to diagnose and has the highest mortality rate.
4. Spontaneous rupture may be mistaken in some cases for the clinical picture of massive myocardial infarction or angina pectoris.
5. From the present study we found no evidence to support the theory of the so-called “spontaneous rupture of normal spleen”.
6. Splenectomy is the treatment of choice.

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BIBLIOGRAPHY