TORSION, INFARCTION AND HEMORRHAGE OF THE OMENTUM AS A CAUSE OF ACUTE ABDOMINAL DISTRESS*

MOSES J. LEITNER, M.D., CLAUS G. JORDAN, M.D.,
MORTON H. SPINNER, M.D., AND EVAN C. REESE, M.D.

EAST STROUDSBURG, PENNSYLVANIA

FROM THE GENERAL HOSPITAL OF MONROE COUNTY, EAST STROUDSBURG

VASCULAR DISTURBANCES of the omentum, particularly of the greater omentum, occasionally cause acute abdominal distress, diagnosed variously as due to acute appendicitis, acute cholecystitis, ruptured peptic ulcer, etc. Fortunately, most cases present such persisting symptoms that laparotomy is performed and the correct diagnosis is usually reached. However, the surgeon must recognize the possible occurrence of this lesion and look for it if the cause of the symptoms is not found in the organ or organs preoperatively suspected of producing the clinical symptoms.

These cases are reported in the literature under several descriptive titles including primary omental torsion, omental volvulus, acute epiploitis, idiopathic segmental infarction, omental thrombosis and similar headings; and usually the cases caused by torsion are segregated from those produced by spontaneous thrombosis as separate and distinct entities. Since the basic disturbance is vascular regardless of etiology, and since the symptomatology is essentially alike in all cases, we propose an inclusive classification or grouping as follows:

A. Omental infarction (with or without gangrene) due to torsion.

1. Primary (idiopathic)
2. Secondary

B. Omental infarction (with or without gangrene) due to thrombosis.

1. Idiopathic (spontaneous) infarction.
2. Associated with vascular disease.
3. Due to external trauma.

OMENTAL INFARCTION DUE TO TORSION

Omental torsion was first decribed in the literature by deMarchetti in 1858, and primary torsion of the omentum by Eitel in 1899. Anton *et al.*[1] in 1946 published an excellent review in which they discussed the embryology, anatomy, physiology and pathology of the omentum. They listed 104 references and proposed the following classification for this lesion:

A. Primary (idiopathic, cryptogenic, pure intra-abdominal).

B. Secondary.
   1. Hernial (external, in sac)
   2. Abdominal
      a. Intrinsic (cysts and tumors)
      b. Extrinsic (associated with pathology of abdominal and pelvic organs and peritoneum)

Caron[5] had found 70 cases of primary omental torsion reported until 1946, and since then a brief survey of the literature indicates about 30 additional cases.

Primary Torsion. The etiology is not immediately apparent. The omentum does not have inherent powers of motion so that its displacements are determined by outside forces; *e.g.*, intestinal peristalsis, intra-abdominal tension, intestinal distention, trac-

* Submitted for publication April, 1951.

tion by adhesions. The causes of primary torsion have been grouped by several authors[3, 14, 29] as (1) predisposing and (2) exciting.

1. The predisposing causes include the following (a) *Anatomical variations*—tongue-like projections, longer right half, bifid structure. (b) *Obesity*—causing irregularly distributed accumulations of excess fat. (c) *Epiploitis* (omentitis)—thought by some authors always to occur, possibly fugitive in nature and not evident on examination, but sufficient to initiate the torsion. This change may be inflammatory, post-traumatic or postoperative. (d) *Arrangement of blood vessels*—the omental veins are longer than the arteries and are thin-walled. Kinking of the veins may cause dilatation and twisting around the shorter tense artery, initiating a self-perpetuating torsion. (e) *Tumors* or *cysts.*

2. The exciting causes are as follows: (a) *Trauma*—including blunt trauma to the abdomen, violent exercise, sudden changes in body position. (b) *Hyperperistalsis*—with resultant increase in passive movements of the omentum. (c) *Vascular changes* — areas of infarction caused by thrombosis or embolism initiate a twisting due to the increased weight of the infarcted area.

Secondary torsion is much more common than the primary type and is associated with adhesions of the free end of the omentum to the peritoneum. This occurs especially to the neck of hernial sacs, although it may occur in many types of intra-peritoneal lesions, or following laparotomy. The secondary torsion is usually bipolar; that is, a torsion of the central portion between two fixed points, but it may be unipolar, with torsion of the free end. The latter type occurs most frequently within hernial sacs into which the omentum has prolapsed or into which it has become incarcerated. The twisted omentum may be entirely within the hernial sac or may be partly intra-abdominal.

Pathology. The twist of the omentum occurs in the long axis, producing a neck of varying length in which a variable number of turns occur around the pivotal point. The pedicle may become considerably attenuated and in a few instances there has occurred auto-amputation.[10] It is interesting to note that the direction of rotation is usually clockwise. The torsion is usually complete and permanent. However, incomplete torsion and self-restitution apparently occurs, or there may be recurring partial torsion, as shown by old irregular areas of scarring and fibrosis in the omentum. In fact, the acute episode at times may be the end result of previous partial or incomplete twisting, with final venous thrombosis initiating a permanent torsion. The basic changes subsequent to the torsion are essentially the same as those due to infarction to be subsequently discussed. The distal or free end of the omentum becomes congested and edematous, hemorrhagic extravasation takes place into the interstitial tissue, and thrombi form in the omental veins. There is inflammatory cell infiltration of varying degrees and if the process is of sufficient duration, gangrene may occur. Aseptic peritonitis usually is present, with varying amounts of free sero-sanguineous fluid in the peritoneal cavity. This latter finding is so characteristic that it should lead to the immediate suspicion of an omental infarction if other obvious pathology does not exist.

In addition to torsion of the greater omentum a few isolated instances are reported in which the torsion has involved the gastrocolic or the gastro-hepatic omentum (more properly termed "ligament"). Thus Eliason[8] found one of his 11 cases to involve the gastro-colic omentum and Etherington-Wilson[9] mentions a case in which the gastro-hepatic omentum was adherent to the surrounding structures and had undergone sec-

ondary torsion. Von Klimko[19] describes the presence of a so-called "third omentum" extending from the anterior surface of the gastro-colic omentum, and describes a case with torsion producing the same clinical picture as that noted in greater omental torsion.

OMENTAL INFARCTION DUE TO THROMBOSIS

Idiopathic (Primary Idiopathic Segmental) Infarction. Schomberg,[26] in 1929, described three cases of what he termed "acute hemorrhagic epiploitis," in all of which there were areas of infarction of the omentum and no evidence of torsion or other omental disease. Johnson,[15] in 1932, reported a case of what he called "primary omental thrombosis" which was associated with heart disease and would probably be more accurately classified as secondary infarction due to vascular disease. In 1940 Pines and Rabinovich[24] described six cases under the title of "idiopathic segmental infarction." One of these patients previously had Buerger's disease, although the authors specifically state that the omental lesion showed no evidence of vascular inflammatory disease. They called attention to the fact that none of these cases showed torsion and also that the infarcted areas occurred in portions of the omentum where torsion was unlikely to occur. Totten,[28] in 1942, presented two cases in which localized distal segments were involved with no trauma, torsion or associated diseases. A number of other cases[7, 11, 16, 20, 25] have been reported since, totalling about 20 to the present date.

Pathogenesis. Pines and Rabinovich[24] showed that forceful pull on the jugular veins of rabbits caused clot formation at the site of injury to the vascular endothelium. Totten[28] suggested that increased vascular congestion occurs after a heavy meal, with resultant tension on the thin-walled omental veins. Additional strain caused by increased intra-abdominal pressure, such as coughing, sneezing, heavy lifting, might cause a local injury or rupture of these thin dependent veins, with hemorrhagic extravasation and secondary thrombosis.

Pathology. Except for the failure to demonstrate a torsion as the cause of the vascular occlusion, the picture is essentially the same as in cases of torsion. A thrombus may be evident, although it may not be discernible in the nondescript blue-black mass of a long-standing infarct. The location is characteristic, usually being noted as a more or less wedge-shaped area at the free edge of the omentum.

Associated with vascular or cardiac disease. A few cases reported as segmental infarction have been associated with vascular or cardiovascular disease. Although in these cases the omental vein thrombosis may have been truly spontaneous, it is quite reasonable to predicate primary changes due to the systemic diseases (venous stasis, embolism) with secondary thrombosis and infarction. Among these cases may be mentioned that of Hines[12] which was associated with thrombosis of the portal vein, and those reported by Berger,[4] Bang-Dietrichsen,[2] Johnson[15] and Fusco,[10] in which there was cardiovascular disease of long duration with varying degrees of cardiac decompensation. One of the cases reported by Pines and Rabinovich had Buerger's disease. It is interesting to note that in one of our cases the possibility of polyarteritis was considered in view of the increased percentage of eosinophiles in the differential white count. However, subsequent observation of the patient has shown no evidence of any other vascular involvement or of persisting eosinophilia.

Due to external trauma. In a few cases a definite history of blunt trauma to the abdomen has preceded the onset of symptoms. Morris[21] describes such a case which occurred three days following an injury sustained at work. He noted that Hertzler was

able to collect 34 such cases in the literature up to 1919 and calls attention to the fact that a severe injury to the abdomen is apt to produce perforation of a hollow viscus or laceration of a solid intra-abdominal organ, overshadowing any possible damage to the omentum or sparing it entirely. Schottenfeld and Rubinstein[27] report such a case in a seven-year-old boy who had been kicked in the abdomen six days before laparotomy, when a large portion of purplish-blue indurated omentum was removed. They were able to find six other cases of omental lesions produced by trauma in a 15-year period at their hospital (Jewish Hospital, Brooklyn, N. Y.) but they do not describe the appearance or the exact type of lesion present in these cases. Schaff and Stephenson[25] suggest the possibility of repeated small trauma in their patient, who was a bus driver and so might have been subject to minor injury to the abdomen in the course of his work.

A very few lesions somewhat different from infarction caused by torsion or thrombosis are reported in the literature. Since they produce essentially the same symptom complex and require the same treatment they are mentioned here. Mourgue-Molines and Cabanac[22] report a case of severe intraperitoneal hemorrhage originating from a hematoma of the gastro-hepatic omentum in which thrombosis was found in the gastric coronary artery. In a case reported by Eberts,[6] spontaneous hemorrhage had occurred from the greater omentum of a young man who had a history of recurrent epistaxis.

In Klein's case[18] the omentum had become entrapped in the foramen of Winslow and had become gangrenous. Timbal, in 1910, described two cases of hemorrhagic cysts of the omentum occurring in soldiers who had sustained trauma to the abdominal wall. Nixon[23] reports a similar case in which a hematoma was found in the gastro-colic omentum and which apparently had been spontaneous in origin, since no history of trauma could be obtained.

CLINICAL FEATURES

There are no significant features to distinguish one type of lesion from the other. This is understandable, since the basic alteration is the same; namely, infarction with or without subsequent necrosis. The preoperative diagnosis is rarely made, the usual clinical impression being, in order of frequency, acute appendicitis, acute cholecystitis, ruptured peptic ulcer and acute pancreatitis. Actually, preoperative diagnosis is more or less of academic interest only, since the symptoms and physical findings inevitably warrant a laparotomy even in the absence of a definitive diagnosis. It is here that the surgeon must be aware of the possible occurrence of this condition, since inadequate exploration through a small McBurney type of incision might permit him to overlook the actual disease. The finding of free sero-sanguineous fluid in the peritoneal cavity and the failure to explain the symptoms by obvious pathologic changes in the appendix, gallbladder, pelvic organs, gastro-intestinal or genito-urinary tract should cause him to suspect the presence of some omental lesion.

The leading complaint is, of course, abdominal pain. This frequently is present in the right lower quadrant of the abdomen, although it is often described as peri- or para-umbilical. It may occur in the right upper quadrant, the right loin, less commonly in the same locations on the left side, and occasionally as generalized pain. The onset is usually sudden, and the pain may be persistent or remitting, and frequently may subside or diminish when the patient lies down.

Tenderness is almost invariably present, most commonly in the right side, especially the lower quadrant. *Rebound tenderness* occurs often and moderate rigidity may be present. However, the rigidity is usually

voluntary rather than the involuntary muscle spasticity of intraperitoneal infection. Special emphasis is laid on marked hyperesthesia of the skin (Jurado[17]) with little muscle guarding, in contrast to the intense pain elicited on touching the abdominal surface. Of some significance is the fact that despite fairly intense pain and tenderness at the onset, the rapid progression that one would expect to find in acute appendicitis is not observed in the same period of time.

Occasionally, if the involved portion of omentum is sufficiently large, a mass may be felt, most often under these circumstances in the right lower or upper quadrants. This has several times led to the diagnosis of localized abscess of the appendix or empyema of the gallbladder.

Fever and leukocytosis occur with about the same frequency as one would anticipate in the usual case of appendicitis. Nausea and vomiting are frequently observed, and diarrhea or constipation may occasionally develop.

It is seen, then, that neither the symptoms nor the physical findings present any characteristic pattern to suggest the diagnosis, since any or all of these features are common to several other acute abdominal diseases. The treatment is simply laparotomy with ligation and excision of the involved area of omentum, or, if necessary, of the entire omentum. The prognosis is excellent and only rarely have any untoward sequelae or postoperative complications been mentioned.

CASE REPORTS

Case 1.—*Infarction Due to Primary Torsion.* H. G., a white male, age 49, was admitted December 3, 1950. Two days before admission he had a feeling of abdominal distention and pressure in the right lower abdominal quadrant. The next evening he was examined by his physician, who apparently found no particular abdominal tenderness. The day of hospitalization he felt fairly comfortable on a liquid diet but at night was awakened from sleep by a severe pain in the lower part of the abdomen. He had no nausea or vomiting, no diarrhea and no urinary complaints.

The temperature, pulse and respirations were normal. Blood count showed WBC's 15,700 with 68 per cent polymorphonuclear leukocytes, 31 per cent lymphocytes and 1 per cent basophiles. There was some abdominal distention, marked localized tenderness and rebound tenderness but no rigidity over McBurney's point. There was no inguinal hernia. The preoperative diagnosis was acute appendicitis, possibly with rupture and localization of an abscess by omentum. At operation some cloudy free fluid was found in the peritoneal cavity. The appendix was localized with difficulty, but did not appear sufficiently diseased to account for the symptoms and the leukocytosis. During further exploration an indurated portion of omentum was felt. This was found to be twisted, blue-black and hemorrhagic, measuring about 2 inches in length and 1½ inches in width. The liver and gallbladder were normal and no other pathologic changes were noted in the abdomen. The infarcted portion of omentum was ligated at the pedicle and excised. Postoperative recovery was rapid and uneventful except for the development of a dermatophytosis of the feet and a severe dermatophytid reaction of the hands.

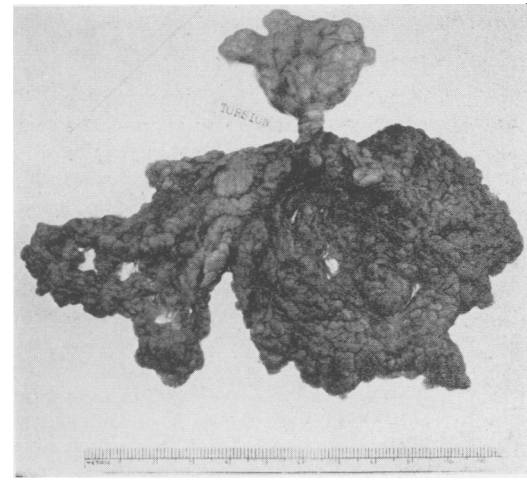

FIG. 1.—(Case 2.) Typical primary torsion. Note formation of pedicle and intense congestion of infarcted portion.

Pathologic report. The specimen consisted of a disc-like mass 5 cm. in diameter, the surface of which was dark-red and hemorrhagic. On cutting it was seen to consist of fat tissue which was markedly congested. Microscopic examination showed adipose tissue with marked hyperemia, areas of hemorrhage and thrombi in the small

veins. Diagnosis was infarction of the omentum due to primary torsion.

Case 2.—*Infarction Due to Primary Torsion.* H. H., a white female, age 53, was admitted January 27, 1951, complaining of pain in the right lower quadrant of the abdomen. She became ill 2 days before with epigastric distress which soon localized to the right side of the abdomen and along the right flank. There was no nausea, vomiting or diarrhea, and the bowels moved after a colonic irrigation. The pain persisted until admission.

The temperature, pulse and respirations were respirations 24. The abdomen was obese. There was tenderness along the right flank, most marked near the anterior superior spine of the ilium, with marked rebound tenderness but no rigidity. A large, firm mass was felt in the hypogastrium, which on vaginal examination appeared to be part of a large uterus containing fibroid tumors. The preoperative diagnosis was retrocecal appendicitis and fibroids of uterus. At laparotomy the appendix was found to be normal. The uterus was enlarged and contained several fibroid tumors. In the right upper quadrant a mass of omentum was found which had twisted on its long axis in a counter-clockwise direction, with hemorrhage and congestion of the distal portion. The pedicle was clamped, and the mass excised. No other pathologic changes were found in the abdomen. The patient made an uneventful recovery.

Pathologic report. The specimen consisted of omentum, which was fanshaped when spread out and measured 5 by 1.5 cm. At the upper end there was a pedicle 1.5 cm. in length which exhibited a definite twisting on its long axis with three complete counter-clockwise turns. The distal portion was deep red in color and congested, and showed tensely distended veins. Microscopic examination showed extravasation of red cells into the fat and thrombi in all the veins. There was no evidence of vascular inflammatory disease. Diagnosis was infarction of the omentum due to primary torsion.

Case 3.—*Infarction Due to Primary Torsion.* (Courtesy of Dr. Walter C. Levering, Abington Memorial Hospital, Abington, Pa.)

E. M., a white female, age 51, was admitted October 22, 1949. For about 17 years the patient had complained of recurring pains in the right upper abdominal quadrant, suggesting gallbladder disease. Four days before admission she developed an attack of right upper quadrant pain with nausea and vomiting. At first she refused hospitalization but with the persistence of the pain finally consented to admission.

The temperature was 100.4 F., pulse 108 and normal. The white blood count also showed no unusual changes. Abdominal examination showed marked tenderness over the right upper quadrant but no rigidity. A small mass was felt which was thought to be the gallbladder and the preoperative diagnosis of acute cholecystitis was made. At laparotomy a purplish red mass, 2 by 2½ inches, was found beneath the liver. It was made up of a twisted portion of the omentum. It was ligated and excised and the patient made a rapid recovery.

Pathologic report. The specimen consisted of a mass of indurated and hemorrhagic fat 3.5 by 3 x 1.5 cm. Microscopic examination showed extensive hemorrhage and degeneration and a moderate infiltration with polymorphonuclear leukocytes. Diagnosis was infarction of the omentum due to primary torsion.

Case 4.—*Infarction of the Gastro-hepatic Omentum Due to Torsion.* G. G., a white male, age 27, was admitted January 5, 1951, complaining of pain in the right upper quadrant of the abdomen. The day before admission he began to have persistent discomfort in the upper abdomen, mostly on the right side. There was no nausea, vomiting, diarrhea, chills or fever. Since the patient was subject to frequent colds and some type of allergic reactions, and since there was slight aggravation of the pain on deep breathing, it was thought he had an early respiratory infection with diaphragmatic pleurisy.

The temperature was 99.8 F., pulse 120, respirations 20. The white blood count was 12,800 with normal differential. On examination he was found to be rather apprehensive and excited. There was moderately severe tenderness in the epigastrium and the right upper abdominal quadrant, and some rigidity over the entire right side of the abdomen. Peristalsis was normal. There was no evidence of hernia. The preoperative diagnosis was indefinite, the possibilities of acute gallbladder disease, pancreatitis, leaking duodenal ulcer and acute appendicitis all being considered. Because of the persisting pain and tenderness, laparotomy was performed on January 9, 1951.

At operation a portion of the gastro-hepatic omentum (ligament) was found which had become twisted and gangrenous. This formed part of an adhesion between the liver, gallbladder and the lesser curvature of the stomach. There was considerable congestion of the round ligament, and of the lesser curvature of the stomach where the infarcted tissue was attached. There was no evidence of twisting. No other intra-abdominal ab- or gastric ulcer and no appendicitis. The infarcted tissue was ligated and excised and the patient made a rapid and uncomplicated recovery.

Pathologic report. The specimen consisted of a roughened, hemorrhagic irregular mass 2 by 1.5 by 1 cm., made up of fibro-fatty tissue and blood clot. Microscopic examination showed extensive hemorrhage and blood clot, and areas of fat necrosis in which there was a proliferation of fibroblasts, suggesting beginning repair. There was no evidence of vascular disease. Diagnosis was infarction of omental tissue (torsion of portion of gastro-hepatic omentum).

Case 5.—*Infarction Due to Secondary Torsion* (Courtesy of Dr. Damon B. Pfeiffer, Abington Memorial Hospital, Abington, Pa.)

K. R., a white male, age 82, was admitted December 19, 1945. Two days before admission he developed generalized crampy abdominal pain with nausea and vomiting. He became constipated and the pain persisted. He had had a small umbilical hernia for years, which, for the past 2 weeks had become non-reducible and now was very tender.

The temperature was 99 F., pulse 96 and respirations 20. The white blood count was 21,000 with 92 per cent polymorphonuclear leukocytes and 8 per cent lymphocytes. At operation some black fluid was found in the sac of the umbilical hernia and a mass of twisted hemorrhagic omentum was mobilized. The omental mass was twisted at the neck and was not adherent to the peritoneal aspect of the sac, and no bowel was found in the hernia. The infarcted area was ligated and resected, and, despite his age, the patient made an uneventful recovery.

Pathologic report. The specimen consisted of a mass of fatty tissue 9.5 by 8 by 3 cm. Some portions were dark red, others brownish and others yellow in color. The blood vessels were engorged. Microscopic examination showed hemorrhagic extravasation into the fatty tissue and thrombi in the small veins. Diagnosis was infarction of the omentum due to torsion (secondary).

Case 6.—*Infarction Due to Primary Thrombosis* (Idiopathic Segmental Infarction).

C. S., a white male, age 64, was admitted November 17, 1950. The patient had been complaining of pain in the left side of the abdomen for 2 days prior to admission. The pain had lessened slightly but had then recurred with greater intensity, and was associated with some nausea but no vomiting. There was also some soreness under the left costal margin. The patient had been retired from active work for about 4 years because of a "heart condition" but, except for slight dyspnea on exertion, had been feeling quite well prior to the present illness.

The temperature, pulse and respirations were normal. The white blood count showed a total count of 11,900 with 72 per cent polymorphonuclear leukocytes, 17 per cent lymphocytes and 11 per cent eosinophiles. On examination he was found to have generalized tenderness and rebound tenderness over the entire abdomen with some rigidity. The rigidity was most marked on the left side and there was a point of maximum tenderness on the left side just above the umbilicus. The preoperative diagnosis was possible leaking gastric ulcer, possible acute pancreatitis or mesenteric thrombosis. At operation a wedge-shaped hemorrhagic mass was found within the greater omentum measuring 3¼ by 1¼ inches and showing no evidence of gallbladder or liver disease, no duodenal normality was found. The involved area of omentum was ligated and excised. The patient made a rapid and uneventful recovery and has remained well to date, 5 months after operation. Repeated blood counts showed no further eosinophilia.

Pathologic report. The specimen consisted of a tongue-like mass 6 by 3 by 1 cm. The distal portion was made up of fat resembling omentum and was markedly hemorrhagic. Microscopic examination showed intense hemorrhage into fatty tissue, and thrombosis of all the veins. There was no evidence of vascular or perivascular inflammatory disease. Diagnosis was infarction of the omentum (idiopathic thrombosis).

COMMENT

These six cases illustrate four different types of omental infarction. The first three are typical of primary omental torsion. The fourth case illustrates a rather uncommon lesion in which the infarction occurs within the gastro-hepatic ligament. The exact cause is obscure, since the ligament does not occur as a structure with a distal free portion. It is possible that the torsion develops on a loose, redundant congenital accessory fold lying between and attached to the round ligament and the lesser curvature of the stomach. The fifth case, secondary torsion associated with incarceration into a hernial sac or adhesions to the neck of a sac, is the most common type and of itself would not warrant reporting. The last case appears to fit into the group recently reported as idiopathic segmental infarction. The eosinophilia in this case is unexplained.

SUMMARY

Acute occlusion of omental blood vessels presents the same clinical features whether due to torsion, idiopathic thrombosis, abdominal trauma or vascular disease. Preoperative diagnosis is rarely made. However, the surgeon must remain aware of the disease and search for it if, at laparotomy, other adequate cause is not found to explain the symptoms, especially if free sero-sanguineous fluid is found in the peritoneal cavity.

Treatment consists of ligation and resection of the involved portion of the omentum and recovery is usually rapid, uneventful and complete.

Six cases are reported illustrating several of the types which occur and a classification is suggested to include all forms of infarction of the omentum.

BIBLIOGRAPHY

[1] Anton, J. I., J. E. Jennings and M. B. Spiegel: Primary Omental Torsion. Am. J. Surg., **68**: 303, 1945.

[2] Bang-Dietrichsen, O.: Infarction of Greater Omentum. Nord. Med., **12**: 3413, 1941.

[3] Barsky, E. K., and A. M. Schwartz: Primary Omental Torsion; Review of Literature and report of two cases. Am. J. Surg., **38**: 356, 1937.

[4] Berger, A. R.: Hemorrhagic Infarction of Greater Omentum. Arch. Surg., **36**: 497, 1938.

[5] Caron, W.: Primary Torsion of the Omentum. Laval Méd., **12**: 481, 1946.

[6] Eberts, E. M.: Case of Spontaneous Hemorrhage from the Great Omentum. Canad. M. A. J., **10**: 461, 1920.

[7] Eger, S. A., and R. E. Barto, Jr.: Primary Idiopathic Segmental Infarction of the Greater Omentum. Am. J. Surg., **78**: 518, 1949.

[8] Eliason, E. L., and J. Johnson: Primary Acute Epiploitis. Surgery, **6**: 68, 1939.

[9] Etherington-Wilson, E.: Torsion of the Great Omentum; Report of Four Cases. Brit. J. Surg., **33**: 142, 1945.

[10] Fusco, E. M.: Surgical Lesions of the Greater Omentum. Virginia M. Monthly, **73**: 371, 1946.

[11] Harris, F. I., T. Diller and S. A. Marcus: Hemorrhagic Infarction of the Greater Omentum Simulating Acute Appendicitis. Surgery, **23**: 206, 1948.

[12] Hines, L. E.: Hemorrhagic Infarction of the Greater Omentum. Illinois M. J., **66**: 166, 1934.

[13] Jackson, A. S.: Primary Torsion of the Omentum. Am. J. Surg., **75**: 849, 1948.

[14] Jaroslavsky, L., G. Stutman and E. Sapisochin: Acute Torsion of the Greater Omentum. Diá. méd., **18**: 2036, 1946.

[15] Johnson, A. H.: The Great Omentum and Omental Thrombosis. Northwest Med., **31**: 285, 1932.

[16] Joss, C. S., and J. S. Pratt: Primary Idiopathic Segmental Infarction of the Greater Omentum. Minnesota Med., **31**: 996, 1948.

[17] Jurado, P.: Idiopathic Hemorrhagic Infarct of the Great Omentum. Bol. Acad. Argent. Cir., **33**: 215, 1949.

[18] Klein, L.: Strangulation of the Omentum. New York State M. J., **50**: 341, 1950.

[19] v.Klimko, D.: Torsion of the Third Omentum. Arch. f. Klin. Chir., **155**: 685, 1929.

[20] MacKenzie, W. C., and J. Small: Primary Idiopathic Segmental Infarction of the Greater Omentum. Canad. M. A. J., **55**: 144, 1946.

[21] Morris, G. N.: A Case of Omental Necrosis Following Trauma. Austral. N. Zealand J. Surg., **18**: 229, 1949.

[22] Mourgue-Molines, E., and J. Cabanac: Abundant Intraperitoneal Hemorrhage from Infarction of the Gastrohepatic Omentum. Bull. et. Mem. Soc. Nat. de Chir., **59**: 720, 1933.

[23] Nixon, W. C. W.: Hematoma of the Gastrocolic Omentum. Brit. M. J., **1**: 797, 1932.

[24] Pines, B., and Rabinovich, J.: Idiopathic Segmental Infarction of the Greater Omentum. Surg., Gynec. & Obst., **71**: 80, 1940.

[25] Schaff, B., and H. U. Stephenson, Jr.: Omental Infarction Simulating Acute Appendicitis. N. Carolina M. J., **10**: 361, 1949.

[26] Schomberg, H.: Acute Hemorrhagic Epiploitis with the Picture of Acute Appendicitis. Beitr. z. Klin. Chir., **146**: 89, 1929.

[27] Schottenfeld, L. E., and H. Rubinstein: Hemorrhage and Thrombosis of the Omentum; Their Etiology in the Acute Abdomen. Am. J. Surg., **51**: 449, 1941.

[28] Totten, H. P.: Primary Idiopathic Segmental Infarction of the Greater Omentum. Am. J. Surg., **56**: 676, 1942.

[29] Zuckerman, I. C.: Primary Torsion of the Omentum. Am. J. Surg., **75**: 637, 1948.