IDIOPATHIC SPONTANEOUS SEGMENTAL INFARCTION OF THE GREATER OMENTUM*

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VARIOUS DESCRIPTIVE TITLES are given in the literature for this condition. These include acute epiploitis, idiopathic segmental infarction and omental thrombosis.[6, 7, 12, 17] Regardless of the terminology used, this is a most interesting and rare entity. It causes acute abdominal distress and thereby adds to the differential possibilities in the etiology of acute abdominal conditions. It has been erroneously diagnosed as acute appendicitis, acute cholecystitis, acute pancreatitis and perforated peptic ulcer.

The insult causing this lesion is due to vascular disturbances, particularly of the greater omentum. This structure can be affected by primary torsion due to herniations or adhesions. Therefore, these entities should not be confused, even though the basic disturbance is vascular and the symptomatology essentially alike. Various attempts at classification have been made;[1, 16, 24] however, the most satisfactory, easiest and all inclusive one is that of Leitner, *et al.*,[16] and is 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).
2. Associated with vascular disease.
3. Due to external trauma.

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HISTORY

A review of the literature reveals about 20 previously reported authentic cases of spontaneous infarction without evidence of other contributory intraperitoneal pathologic changes.

Schomberg[24] in 1929 reported three cases of what he termed "acute hemorrhagic epiploitis," in all of which there were areas of infarction of the omentum without evidence of torsion or other omental disease. Johnson[12] is credited with reporting the first case in 1932; however, his patient had heart disease, plus the fact that the omental tip was adherent to a scar resulting from a previous herniorrhaphy.

Pines and Rabinovich[22] described six cases of "idiopathic segmental infarction" in 1940. None of these cases showed torsion and the areas of infarction occurred in portions of omentum where torsion was unlikely to occur. One of these patients had Buerger's Disease; however, the omental lesion failed to show evidence of vascular inflammatory disease. Totten[27] in 1942 presented two cases in which the distal segments of omentum were involved without trauma, torsion or associated disease. Joss and Pratt[13] in 1946 reported 12 cases from the literature and added one of their own. Seley[26] in 1951 reported a case occurring in a patient with polycythemia vera and could find only 16 authentic cases in the literature. Leitner *et al.*,[16] in an excellent article, presented a case in a patient who had been retired because of heart disease four

years prior to his infarction of the omentum. Higgins[10] presented the most recent case in 1952.

If one adheres strictly to the classification of Leitner *et al.*,[16] the cases of Bang-Dietrichsen,[2] Berger,[3] Fusco,[8] Hines,[11] Johnson,[12] Leitner, *et al.*,[16] Pines and Rabinovich,[22] and Seley[26] could be listed as due to systemic or vascular disease with secondary thrombosis and infarction, as against primary or idiopathic disease.

It is felt that the number of reported cases is not a true indication of the incidence of this condition, since it is believed that many cases are unreported, undiagnosed because no operation was performed, and not diagnosed even after surgery.

PATHOGENESIS AND ETIOLOGY

The mechanism in this condition is open to considerable speculation. Pines and Rabinovich,[22] in rabbit studies, showed that a forceful pull on the jugular veins caused clot formation at the site of injury to the vascular endothelium. They further advanced the theory that venous thrombosis occurs consequent to stretching of the omental veins secondary to some trauma with subsequent omental infarction. Totten[27] felt that a strain on the thin walled veins between the stomach and omentum increased vascular congestion after ingestion of a full meal, and that during this period, any additional abdominal tension, such as straining, coughing, sneezing, or lifting might result in rupture of a dependent vein, with hemorrhagic extravasation and secondary thrombosis. Trauma to the abdomen has preceded the onset of symptoms in a few cases.[19, 23, 25, 27] Vascular or systemic disease as a cause has already been covered.[2, 3, 8, 11, 12, 16, 22, 26] Other causes have also been reported.[5, 15, 20, 21]

CLINICAL FEATURES

The clinical features of this disease are not distinct, and the diagnosis is rarely, if ever, made preoperatively. In most cases, the symptoms, signs and laboratory findings simulate those of acute appendicitis.[9, 23, 24] However, in some instances, other conditions such as acute cholecystitis, acute pancreatitis and perforated peptic ulcer have been mimicked. All ages are involved and the sex distribution is apparently equal. Manfredi[18] in 1950 reported a case occurring in infancy.

Nausea and vomiting may be present, and constipation or diarrhea may frequently be observed. The constant complaint is abdominal pain. It can occur in the right upper abdomen, peri- or paraumbilically, in the right loin, occasionally as generalized pain, but most often it is present in the right lower quadrant of the abdomen. It is usually sudden, may be persistent or remitting, and may clear or ease when the patient is lying down or inactive. Tenderness invariably is associated with pain, especially in the lower right quadrant. Rebound tenderness is present and as usual, should be elicited last and with extreme care in acute abdominal conditions. Rigidity is present as well as skin hyperesthesia.[14] Occasionally, if the involved segment of omentum is sufficiently large, a mass or fullness may be felt in the abdomen and lead to a possible diagnosis of localized collection (abscess) or empyema of the gallbladder.

Fever and leucocytosis may or may not be present; however, they do occur in about the same frequency as one would anticipate in the usual case of appendicitis.

This disease does not progress in the same manner as acute appendicitis, despite fairly severe pain and tenderness at its outset.

PATHOLOGY

The picture is essentially that which is found with torsion of the omentum: Grossly, a discolored, hemorrhagic, indurated and congested segment of fatty tissue is noted. It varies in size and shape and its

location is characteristically at the free edge of the omentum. Microscopically, the adipose tissue shows marked congestion and thrombosis of venous channels with hemorrhagic extravasation into the omental fat. The surface shows mild inflammatory reaction with mixed exudate consisting of monocytes, polymorphonuclear leucocytes and plasma cells.

Expectant treatment of an acute abdomen with this diagnosis would be extremely hazardous because of the impossibility of making this diagnosis preoperatively. The appendix can be removed safely during the course of operation.

Aspiration of the sero-sanguineous fluid is performed and the abdomen closed without drainage.

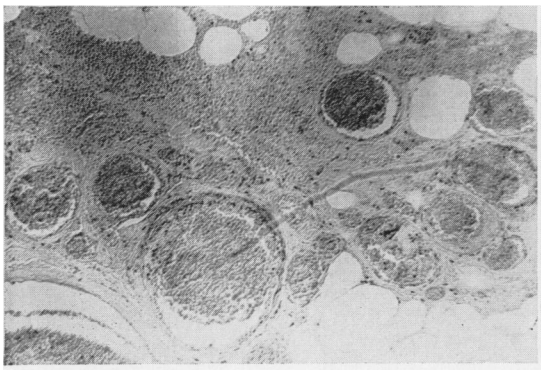

FIG. 1

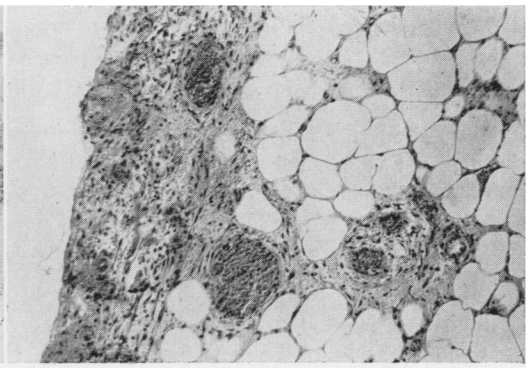

FIG. 2

FIG. 1.—H. & E. x 110 Omentum-thrombosed omental vessels with extravasation of erythrocytes.
FIG. 2.—H. & E. x 110. Omentum-fibrinous exudate with organization.

TREATMENT

The treatment is simple laparotomy with removal of the involved segment of omentum. A right rectus incision is recommended. It will permit more accurate diagnosis and also more definitive surgery in the event that other pathologic conditions are encountered. The finding of free sero-sanguineous fluid in the abdominal cavity and failure to explain the symptoms by obvious pathologic changes in the appendix, gallbladder, pelvic organs, etc., should make one suspicious of this lesion.

In all the reported cases, recovery was uneventful after removal of the infarcted segment of omentum, and only rarely have any untoward sequelae or postoperative complications been mentioned. A case is reported in which the involved segment was not removed with recovery.[4]

CASE REPORT

J. E., No. 4239. This patient was admitted to the hospital on January 17, 1952, with a chief complaint of pain in his right side for the past six days. He further stated that he was well until January 11, 1952, when he developed pain in his right flank which spread across his abdomen to the left. The pain became more severe and the patient returned home. He consulted his family physician who gave the patient some pills. These gave very little relief. He now felt nauseated but did not vomit. His kidneys and bowels functioned normally. After several days (Jan. 14, 1952) the patient felt somewhat better and returned to work. At this time the pain recurred and localized in his right side. After forcing himself to work for several days, the patient had finally to quit when he began to have diarrhea associated with the pain, even though he had not taken a laxative. He was again seen by his family doctor on January 17, 1952, and was sent to the hospital. He denied any injuries to his abdomen.

Past medical history, family history, social history and systemic review were non-contributory.

Physical examination was that of a 32-year-old, acutely ill white male of Italian extraction. T–99.8°, P–72, R–20, BP 120/70 (right arm). Tongue was coated. Heart and lungs were normal. Abdomen–symmetrical but with some anterior fullness in right lower quadrant. Tenderness, rebound tenderness and muscle guarding were elicited. Peristalsis was audible though hypoactive. Rectal examination revealed tenderness on the right side. It was felt that this patient had acute appendicitis with possible rupture and localized abscess formation.

Laboratory studies showed erthrocyte count of 4,600,000; Hemoglobin 13 Gm. (84.5 per cent); leukocyte count 6,500 with 62 per cent polymorpho-nuclear cells, 33 per cent lymphocytes, 4 per cent monocytes, 1 per cent eosinophiles.

He was operated upon via a McBurney incision shortly after admission, and was found to have edematous tissues in the layers of the internal oblique, transversalis muscles and fascia. The peritoneum was thickened and considerable serosanguineous fluid was encountered on entering the abdominal cavity. A specimen of this was obtained for laboratory study and the remaining aspirated as thoroughly as possible.

To the medial side of the incision, a mass of necrotic, gangrenous and hemorrhagic tissue was released from its fresh attachment to the anterior parietal peritoneum and delivered into the wound. It was noted to be a tongue-like portion of diseased fatty tissue, measuring 16 x 4 x 2 cm., which had auto-amputated itself from the remaining omentum, except for a small string-like attachment. This was clamped, ligated and cut, thereby permitting removal of the diseased tissue. The terminal ileum, cecum and appendix were acutely inflamed due to the aseptic peritonitis. The appendix was removed routinely. The abdomen was closed without drainage. The report on the aspirated material showed no growth. The convalescent period was uneventful and the wound healed primarily. Chest roentgenogram, blood sugar, urinalysis and complete blood count during the convalescent period were all within normal limits.

The patient was discharged on January 29, 1952.

PATHOLOGIC REPORT

Gross: Specimen was received in two parts. One specimen is omentum, measuring 16 x 4 x 1½ cm. and discolored throughout with hemorrhage. The other is appendix, and measures 7½ cm. in length. There is a sharp kink in the proximal third and the lumen is narrowed. The diameter of the appendix proximally is 7 mm., distally it is 4 mm. The serosa and meso-appendix are discolored a reddish black. On section, the lumen contains tan colored feces.

Microscopic: The specimen of omentum reveals widespread blood vessel thrombi. Throughout the connective tissue stroma there is an exudation of fibrin and neutrophilic leucocytes with occasional areas in which foreign body giant cells are present. The appendix reveals an intact mucosa. The submucosal lymphatics are hyperplastic with active germinal centers. The serosa is greatly thickened due to fibrinous exudate in which there are dilated blood vessels and enmeshed leukocytes. Throughout the meso-appendiceal fat there is blood vessel congestion and diapedesis of red cells.

Diagnosis: (1) Portion of omentum with venous thrombosis and early fat necrosis, (2) Peri appendicitis, secondary to above.

Comment: The specimen reveals widespread venous thrombosis (Figs. 1 and 2).

COMMENT

This case is one of primary idiopathic spontaneous segmental infarction of the greater omentum in an otherwise healthy white male. The diagnosis was not made, nor even suspected preoperatively, and it was felt that the patient had acute appendicitis with possible rupture and localized abscess even though the clinical features were somewhat unusual. The patient had pain, nausea, slight temperature elevation, tenderness, rigidity and rebound tenderness. There was a suspicion of an indistinct mass in the right lower quadrant due to the fullness in this area. There was diarrhea and no vomiting or leucocytosis. The condition was present six days prior to admission and its progress was not that of the usual case of acute appendicitis in a young adult male.

There was no history of trauma of any kind, nor was there any history of any antecedent or concurrent disease. The patient stated that he had always enjoyed good health.

SUMMARY

1. The subject of idiopathic spontaneous segmental infarction of the greater omentum is presented with a review of the literature.

2. This disease is rare, although it is felt that many cases are unreported and undiagnosed.

3. It is seldom, if ever, diagnosed preoperatively; requires surgical intervention and careful exploration, especially if free serosanguineous fluid is found in the abdominal cavity without other adequate cause.

4. The treatment is simple laparotomy with ligation and removal of the involved segment of omentum.

5. A case is reported with removal of the lesion and the appendix, with a rapid uneventful recovery.

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