Primary Idiopathic Segmental Infarction of the Greater Omentum:

Case Report and Collective Review of the Literature

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PRIMARY idiopathic segmental infarction of the greater omentum is an uncommon cause of acute abdominal symptoms that has been recognized with increasing frequency in the past decade. Although hemorrhage into the greater omentum was first described by Bush [5] in 1896 and Eberts [8] in 1920, Johnson [14] is generally credited with the first authenticated description of omental infarction in his report of a case in 1932. Wrzesinski and his associates [34] in 1956 outlined the diagnostic criteria that distinguish primary idiopathic segmental infarction from omental infarction due to other causes such as torsion and strangulation in hernias. There has been a total of 87 cases reported, two-thirds within the last 15 years. It is our purpose in this report to add a case to those previously reported, to analyze the clinical manifestations of this entity useful in its recognition and management, and also to present certain observations that suggest a congenital contribution to its pathogenesis.

Case Report

J. V., a 37-year-old white man, was admitted to the Veterans Administration Hospital of Indianapolis, Indiana on 9–6–66 with pain in the right lower quadrant of the abdomen of 3 days duration. Onset was not related to strenuous exertion or the ingestion of a large meal. Discomfort was ingravescent, nonradiating, and increased by moving or coughing. He had no nausea, vomiting, diarrhea, constipation, dysuria, frequency of micturition, chills, or fever. He had not previously had any similar difficulty. The past medical history was unremarkable.

On physical examination he was an obese man not in any distress. His blood pressure was 150/90 mm. Hg., pulse rate 92, and temperature 37.9° C. Pertinent physical findings were confined to the abdomen. There was no scar or hernia. Direct and rebound tenderness and moderate rigidity of the abdominal muscles were present in the right lower quadrant and right paraumbilical areas of the abdomen. Rovsing's sign was positive. No organomegaly or mass was palpable, and rectal examination was unremarkable.

Results of the laboratory tests were as follows: hemoglobin 15.3 g.%, hematocrit 49%, white blood cell count 14,100/cubic mm., with 66% neutrophilic leucocytes, 2% bands, 29% lymphocytes, and 3% monocytes. Urinalysis did not reveal any abnormality.

A preoperative diagnosis of acute appendicitis was made. The patient was operated upon through a transverse right lower quadrant incision. Two-hundred to 300 cc. of serosanguinous fluid were aspirated. The cecum was in a high position at the level of the umbilicus. The appendix appeared normal. Immediately above the cecum and overlying it was a firm purple mass which could not be visualized well through the original incision. The wound was closed, and an upper right paramedian incision was made. The mass overlying the cecum and the ascending colon appeared to be infarcted omentum and was adherent to the colon and the anterior peritoneum. The infarcted omentum was separated from the colon and dissected from the viable omentum along a plane of cleav-

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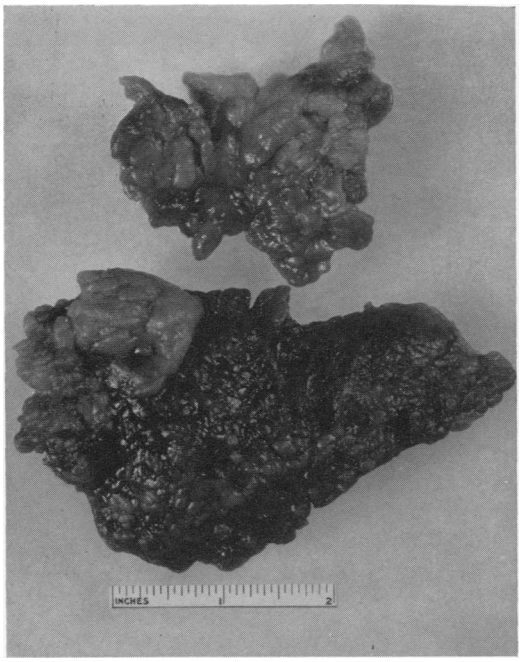

FIG. 1. Infarcted omentum; the larger specimen was adherent to the cecum, the smaller to the ascending colon.

age not traversed by blood vessels. A second small portion of the omentum with a possibly compromised circulation overlay the ascending colon and was also resected (Fig. 1). An incidental appendectomy was performed. There were no other abnormalities in the abdomen. The postoperative course was uncomplicated.

The two specimens of omentum measured 9.5 × 7.5 × 2.5 cm. and 7 × 5 × 1.5 cm., respectively. On histologic examination there was hemorrhage throughout the larger specimen, extensive intravascular thrombosis, and some infiltration by acute inflammatory cells (Fig. 2). In the smaller specimen there was more marked inflammatory reaction than in the former, necrosis of fat cells, and distention of blood vessels (Fig. 3). The appendix had edema of the subserosa with fibrinous exudate on the serosal surface. The diagnoses were hemorrhagic infarction and nonspecific inflammation of the omentum, and acute periappendicitis.

Discussion

With the addition of the present case 88 instances of primary idiopathic segmental infarction of the greater omentum have been reported, 52 of which are in addition to those reviewed by Shea *et al.*[27] in 1956. Fifty-one patients were men and 27 were women. Ages ranged from 3 to 72 years with the distribution shown in Figure 4. The lesion occurs at all ages but apparently with the lowest frequency in the second decade of life. In all cases, the presenting symptom was abdominal pain in the locations listed in Table 1. This was in the right half of the abdomen in 84 per cent of instances. Figure 5 shows the duration of symptoms prior to hospitalization. Eighty-eight per cent of patients for whom data were available sought medical attention on or before the fifth day. On this evidence the severity of the disease appears to be of the same order as acute appendicitis or cholecystitis. Most patients had pain as the only symptom. However, in a significant minority there were associated problems. Among 79 patients in whom the presence or absence of symptoms referable to the upper gastrointestinal tract was noted in the case history, 20 patients (26%) had nausea only; 15 (20%) had emesis; and 44 (54%) had neither. Bowel habits were recorded in 48 case reports. Among these diarrhea was present in eight (17%), constipation in 12 (25%), and neither in 28 cases (58%). There were no reports of associated urinary dysfunction.

Physical findings were variable, but tenderness and peritoneal irritation were usually localized. Frequently, the site of maximum discomfort was lower than usually found in acute cholecystitis and slightly higher than in acute appendicitis. On admission, 33 per cent of patients were afebrile, while another 57 per cent had temperature elevations of 2 degrees or less, and the remaining 11 per cent had elevations between 2 and 4 degrees.

Laboratory studies in the cases reported were limited. The white blood cell count

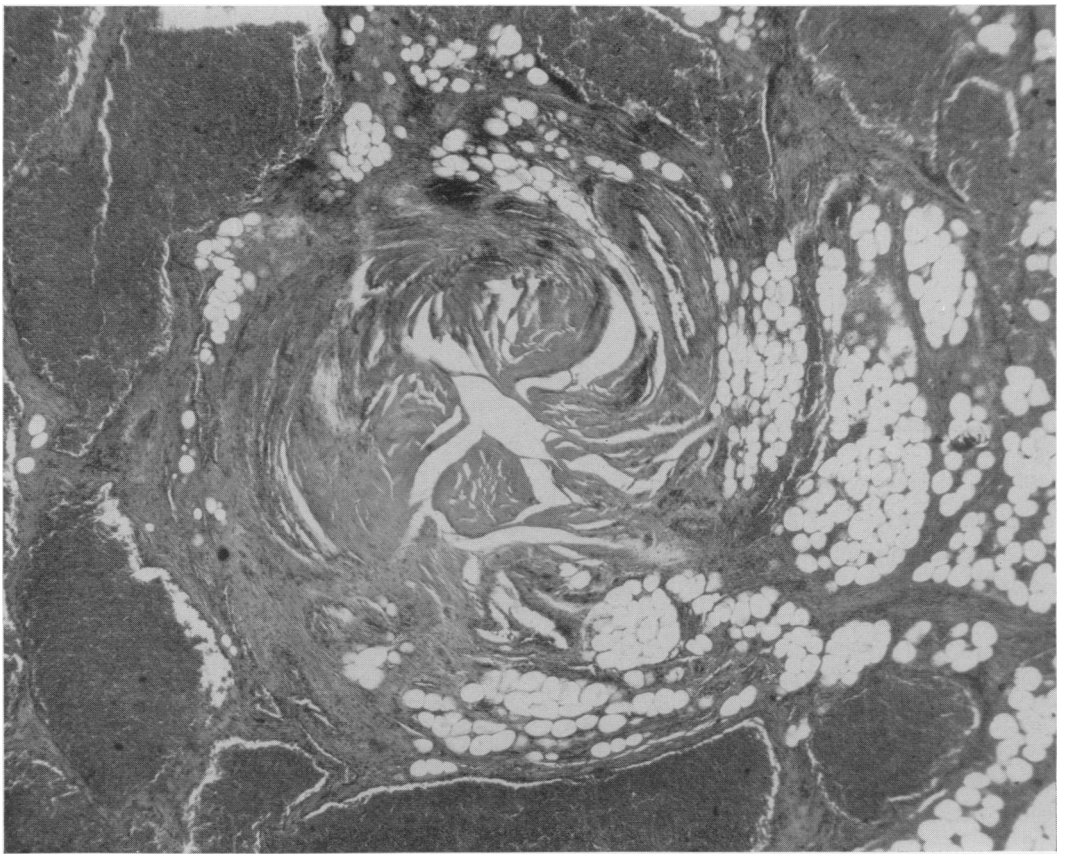

FIG. 2. Photomicrograph of the larger specimen showing a vein occluded by thrombus, extravasated red blood cells, and some infiltration by acute inflammatory cells (×32).

was normal in 37 per cent; elevated but not in excess of 14,000/cubic mm. in 47 per cent of patients. The remaining 16 per cent had elevations between 14,000 and 18,000 white blood cells/cubic mm.

The diagnosis was established in all patients by operation. Table 2 lists the preoperative diagnoses made in 83 patients. The most common misdiagnoses were acute appendicitis and acute cholecystitis which were considered in 64 per cent and 22 per cent of the cases, respectively. With increasing awareness of primary idiopathic segmental infarction of the greater omentum, three patients were correctly diagnosed preoperatively.[2, 10, 14]

At the time of operation serosanguinous fluid was found in the peritoneal cavity of 25 patients. The infarcted omental mass was attached to the ascending colon, cecum, or anterior peritoneum. All patients responded well to surgical exclusion without further complications. Although it has been speculated that failure to remove the infarcted omentum would not result in any great harm, it is likely that without excision the morbidity rate would be considerably greater.

The pathophysiology of primary idiopathic segmental infarction of the greater omentum has been postulated to involve venous congestion and thrombosis. Pines

TABLE 1. *Location of Abdominal Pain at Onset*

Location	No.	%
Right Lower Quadrant	46	52.3
Right Upper Quadrant	19	21.6
Right Side	9	10.2
Lower	3	3.4
Left Upper Quadrant	3	3.4
Left Side	2	2.3
Epigastric	2	2.3
Paraumbilical	1	1.1
Left Side	1	1.1
Generalized	1	1.1
Not Specified	1	1.1
Total	88	100.0

TABLE 2. *Preoperative Diagnosis*

Diagnosis	Number	%
Acute Appendicitis	53	63.9
Cholecystitis	18	21.7
Omental Infarction	3	3.6
Perforated Ulcer	2	2.4
Carcinoma of Cecum	2	2.4
Diverticulitis	2	2.4
Carcinoma or Diverticulitis of Sigmoid	1	1.2
Splenic Infarction	1	1.2
Retroperitoneal Tumor	1	1.2
Total	83	100.0

and Rabinovitch [21] demonstrated that venous congestion is most marked in the marginal veins of the dependent border of the omentum. A temporary retardation of flow occurs during digestion, and injury to the endothelium during this time predisposes to clotting in that vessel. Totten [30] believed the small veins of the omentum were markedly distended after a full meal and that during this postprandial period any strain resulting from coughing, sneezing, or lifting a heavy weight might cause rupture and extravasation. In his report of a 29-year-old man the episode of abdominal pain occurred after the patient had lifted several 120 pound boxes following a heavy noon meal. MacKenzie [16] reported a 29-year-old man whose pain occurred after having ingested one pound of cheese for lunch. Harris and associates [11] believe that an extremely fatty omentum may increase gravitational pull on the vessels, thereby predisposing the veins to easy clotting. Several authors believe that this entity more commonly occurred in obese people, but only 21 of the 88 patients described were reported as obese.

The above mentioned explanations are based on the frequency of postprandial onset and obesity or heaviness of the greater omentum. They do not explain the high incidence of occurrence on the right side, 84 per cent as determined by the location of pain, nor the consistency with which the infarcted omentum is found adherent to the right colon, nor the apparent sequestration of the omentum observed in the present case.

A review of the developmental anatomy of the omentum suggested that it may contribute to the pathogenesis of the lesion. Beginning in the embryo of 12 to 14 mm.,[2] the stomach rotates on an axis in two planes to attain its final, approximately transverse position. During this maneuver, the dorsal mesogastrium is converted into a pouch-like double fold consisting of two layers of flattened endothelium, the greater omentum. As the omentum descends the transverse colon ascends to its adult position, so that after the fourth month of fetal life the posterior layer of the omentum lies over and adheres to the transverse colon. The ventral mesogastrium subsequently gives rise to the lesser omentum, and from its right border, known as the hepatoduodenal ligament, there occasionally arises an abnormal extension which may become incorporated with the greater omentum.[6] This secondary portion of omentum may become attached to the ascending colon and sometimes to the terminal ileum. The greater omentum may also extend beyond

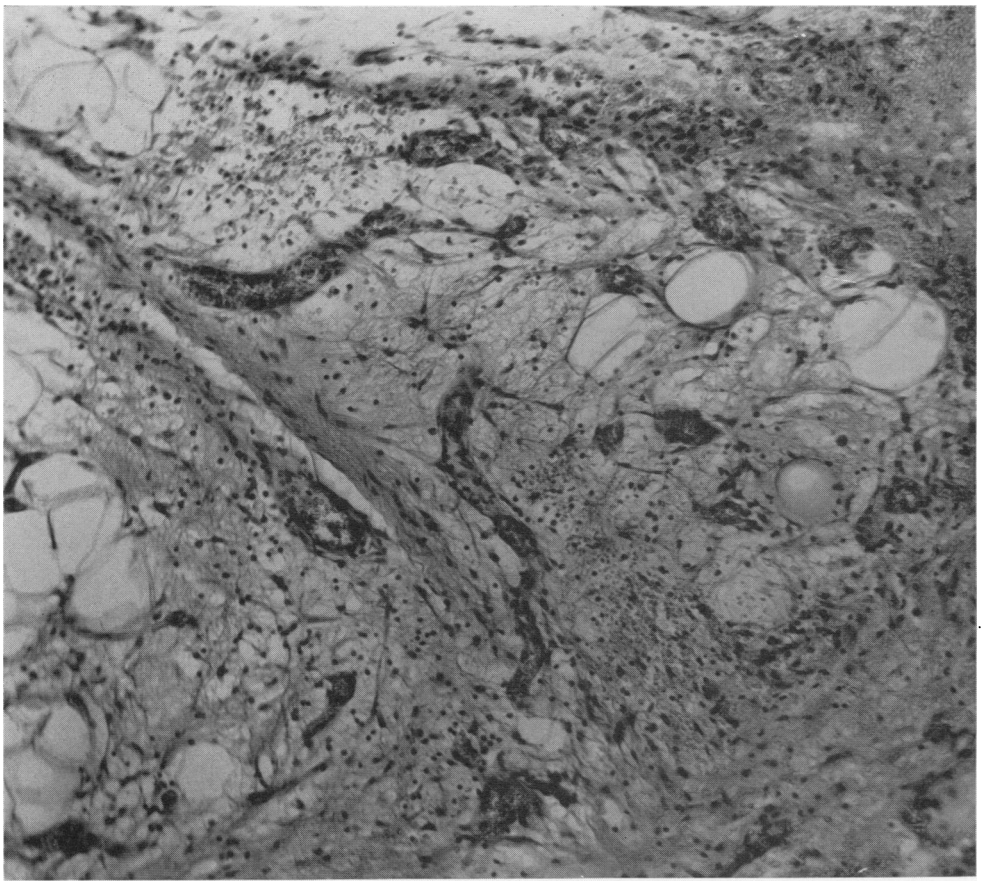

FIG. 3. Photomicrograph of the smaller specimen showing necrosis of fat cells, some extravasation of red blood cells, and infiltration by acute inflammatory cells (×100).

the right colon and attach to the anterior peritoneum. This latter attachment, which is known as the ligament of Haller, is particularly likely to be ascribed to some antecedent inflammatory process when discovered at the time of celiotomy.[13] The circulation of the omentum is derived from the epiploic vessels. The size, shape, weight, and position of the omentum varies markedly.[25] In obese individuals the greater omentum may attain huge proportions while in thin individuals, as a rule, it is relatively devoid of fat.

The areas of infarction are those portions of the greater omentum that have arisen from the ventral mesogastrium and are attached to the greater omentum by weak bands of fusion; the blood supply to these areas, coming from the lesser omentum and secondarily via parasitic vessels from the greater omentum, is tenuous. Kinking of these veins due to position or vascular congestion following large meals and an abrupt increase of intra-abdominal pressure, may produce thrombosis and extravasation. This would account for the fact that although all the sites of infarction were in the so-called dependent free border of the omentum, the infarcted area was

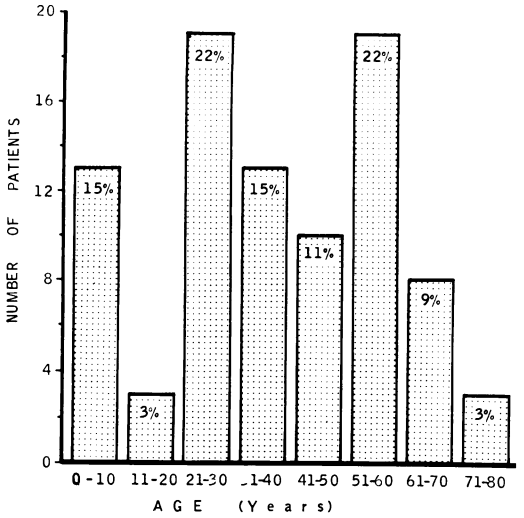

FIG. 4. Distribution by age of patients with primary idiopathic segmental infarction of the greater omentum.

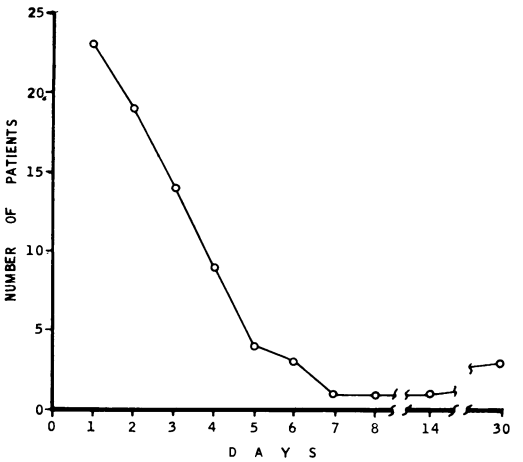

FIG. 5. Duration of symptoms prior to hospitalization of patients with primary idiopathic segmental infarction of the greater omentum.

always adherent to either the colon or the anterior peritoneum.

Summary

Reported cases of primary idiopathic segmental infarction of the greater omentum are reviewed and one case added, bringing the total to 88. This is a rare acute abdominal lesion characterized primarily by pain and the physical findings of peritoneal irritation in the right side of the abdomen. It is frequently mistaken for acute appendicitis or cholecystitis. Although excision of the infarcted omentum may not be necessary, operative therapy is recommended to decrease morbidity and to exclude other more common abdominal conditions which require surgical intervention.

The observations that the infarcted segment of omentum is charasteristically on the right side, adherent to the right colon, cecum and anterior peritoneum, and in the present case, at least, possesses only the most tenuous connection to the remainder of the greater omentum, suggest that it is an embryologic variant, a secondary portion of the greater omentum actually derived from the ventral mesogastrium.

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