IDIOPATHIC SEGMENTAL INFARCTION OF THE OMENTUM—A RARE CAUSE OF ABDOMINAL PAIN

Report of a Case

HERBERT C. KHALOUF, M.D.,* and ROBERT E. HERMANN, M.D.
Department of General Surgery

IDIOPATHIC segmental infarction of the omentum is a rare cause of acute severe abdominal pain. Various names have been given to this entity: *omental infarction*, *acute segmental omental infarction*, *spontaneous infarction of the greater omentum*, *thrombosis of omentum*, *primary omental torsion*, *acute epiploitis*, and *hemorrhagic infarction of the greater omentum*. This specific disease entity has been confused with other, more common types of omental infarction secondary to other conditions. In 1956 Wrzesinski, Firestone, and Walske[1] proposed definite criteria for diagnosing this disease: "(1) The infarction must be spontaneous and idiopathic and not preceded by trauma, infection or other etiologic factors which could, by obvious cause and effect, initiate the process; (2) it must be segmental and not associated with massive vascular occulsions involving large areas of the omentum and/or adjacent organs; (3) it must be primary in the omentum and not the result of disease in a neighboring structure; (4) it should present the typical gross and microscopic picture excluding especially the presence of a pedicle, either twisted or untwisted, which would indicate pathology secondary to torsion."

Bush[2] in 1896, and Johnson[3] in 1932, have each been credited by various authors[1,4-6] for reporting the first case. Neither of the two cases, however, *completely* fulfills the criteria proposed by Wrzesinski, Firestone, and Walske.[1] Because the above criteria for diagnosis have not been fulfilled by all cases reported before 1956, other authors who have reviewed the literature have differed in their counts of the total number of cases reported to date.[5-8] There probably are fewer than 75 cases of true idiopathic segmental infarction of the omentum which have been documented and, as Knudson[6] points out, more than half of these were reported in the last decade.

No cases of idiopathic segmental omental infarction have previously been reported as occurring in Negroes.[4] Our report concerns a case which occurred in a Negro man, treated at the Cleveland Clinic Hospital, which fulfills the diagnostic criteria as outlined above. A study of all the medical records of the Cleveland Clinic Hospital and of the University Hospitals† of Cleveland has shown this case to be the first instance of this diagnosis to be recorded at either institution.

**Fellow in the Department of General Surgery.*

†Permission to review the case records was kindly given to the authors.

Report of a Case

A 36-year-old Negro man was admitted to the Cleveland Clinic Hospital on April 2, 1963, because of the onset of abdominal pain in the right lower quadrant three days previously, and after a bout of heavy drinking; there was no nausea or vomiting. Though the pain was steady and persistent his appetite was good and he had eaten regularly. His bowels had moved regularly; he noted some frequency and nocturia over the prior few days, but no dysuria. He could recall no previous trauma. Two years previously he was hospitalized because of tuberculosis for three months, then discharged to his home, with no further sign of disease activity.

On physical examination, his temperature was 99.4 F., his pulse was 80, and blood pressure was 100/70 mm. of Hg. The results of physical examination were normal except for the abdomen, which was soft, with localized tenderness in the right lower quadrant at its maximum over McBurney's point. There was muscular guarding in that region without rebound tenderness. Bowel sounds were present. There was no tenderness on rectal examination.

Blood tests revealed the following: hemoglobin, 15.2 gm. per 100 ml.; cell volume, 44 percent; white blood cell count, 15,900 per milliliter; and differential count, 82 percent polymorphonuclear cells, 13 percent lymphocytes, and 5 percent monocytes. Urinalysis was normal except for a trace of albumin, from 4 to 6 red blood cells, and from 1 to 3 white blood cells per high-powered field on microscopic examination. A plain roentgenogram of the abdomen showed only that the lower half of the right psoas shadow was not so well defined as the left one. A roentgenogram of the chest showed evidence of a fibronodular infiltrate in the left apex, but otherwise appeared normal. An intravenous urogram was reported as showing normal function and structures.

A diagnosis of probable acute appendicitis was made. Because of the somewhat atypical disease course and findings, it was decided to postpone operation and to observe the patient closely. The next day his temperature remained at 99 F., but the tenderness was more localized than before; there was localized rebound tenderness and some tenderness on rectal examination high on the right side. Surgical treatment was advised.

A laparotomy was performed (under general anesthesia) through a low right paramedian incision. On opening the peritoneum, a small amount of serosanguinous fluid was encountered. The appendix appeared normal. A firm, purplish-red, 3 cm. by 5 cm. mass of hemorrhagic fatty tissue was present in the right border of the greater omentum. The colon, gallbladder, duodenum, and other intraabdominal organs all appeared normal. An appendectomy was performed and the involved segment of the omentum was excised. The abdomen was irrigated and closed without drainage. Pathologic study of the excised segment of omentum showed "venous congestion, severe, focal hemorrhage and focal infarction." The appendix was reported to be normal. The patient had an uneventful recovery and was discharged to his home on the sixth postoperative day.

Discussion

Etiology. The pathogenesis of idiopathic segmental omental infarction is not known. Many theories have been proposed but none of them is entirely satisfactory. The almost constant location of the infarcted segment in the right side of the greater omentum suggested to Eger and Barto,[9] that some anatomic peculiarity of the venous drainage, predisposing to thrombus formation in its lower right portion, might be a contributing factor. Harris, Diller, and Marcus[10] believed that an extremely fatty omentum, by increasing gravitational pull on the omental vessels, might act in conjunction with other factors to cause rupture of a vessel with subsequent hemorrhage and infarction. Pines and Rabinovitch[11] reported that in rabbits an experimental forceful pull of the jugular vein injured its endothelial lining and resulted in a clot at the site of the injury. They believed that in humans the similar stretching of an omental vein might occur, consequent to some trauma, and this in turn could lead to formation of a thrombus.

Totten[12] postulated that sudden increased intraabdominal pressure, such as

that caused by coughing, sneezing, or laughing, especially after a heavy meal, might cause primary rupture of some of the dependent omental veins. This could result in hemorrhagic extravasation into the omentum and secondary thrombosis.

Clinical findings. There is no characteristic clinical picture for this type of infarction of the omentum. The diagnosis is rarely made preoperatively, the most common preoperative diagnosis being that of acute appendicitis, and next in frequency, acute cholecystitis. Other reported preoperative diagnoses have included splenic infarction, perforated peptic ulcer, diverticulitis of the sigmoid, pancreatitis, and mesenteric thrombosis. The patients generally have been young or middle-aged adults, although it has been reported as occurring in all age groups and in a child as young as three years of age.[7] Abdominal pain is the only constant symptom. Usually the pain starts suddenly, in the right lower quadrant; it is generalized and mild at first, but then localizes and becomes severe. It may be persistent or remitting, is aggravated by movement, and often is diminished or relieved when the patient lies down. Often the pain has been present for two or three days before the patient seeks medical consultation.

Abdominal tenderness always is present. This is most commonly found in the right lower quadrant and may be mild or extremely severe; often rebound tenderness and moderate rigidity may be present. Hyperesthesia of the abdominal wall has been reported[7,8] but is not a frequent finding. Anorexia and nausea may be present but rarely is there vomiting. Constipation or diarrhea occasionally occur. Most patients, however, have few gastrointestinal symptoms, have continued to eat and to have normal bowel function. There may or may not be a low-grade elevation of temperature. The leukocyte count is usually increased with an increase in polymorphonuclear forms. Occasionally a tender mass may be palpated if the infarcted omental segment is large.

Pathologic findings. Serosanguinous or sanguinous fluid is commonly found on gross examination when the abdomen is opened. The typical lesion is approximately triangular and located in the right lower edge of the greater omentum. It may vary from a dark-yellow, edematous and indurated area, to a purple mass of hemorrhagic and gangrenous fat, depending on the age of the lesion. On microscopic examination there is venous congestion and thrombosis with necrosis and hemorrhagic extravasation into omental tissue. The surface may show an inflammatory exudate. In old lesions there is frequently infiltration of polymorphonuclear and mononuclear cells with proliferation of fibroblasts and foamy macrophages.

Treatment. The preferred treatment in almost all cases has been excision of the affected segment of omentum. When this has been done the results have been uniformly good. Cagney and Milroy[13] reported one of the few cases in which excision was not done; the lesion was in the gastrocolic omentum. They drained the area only; there was immediate relief of pain and the patient had an uncomplicated recovery. Hallstrand[7] suggested that leaving the infarcted area intact might

allow it to serve as a source of emboli. Eger and Barto[9] believed that failure to recognize and to remove the lesion might lead to adhesions and subsequent unexplained abdominal symptoms.

Comment. Since the diagnosis of idiopathic segmental infarction of the greater omentum can rarely be made preoperatively, its importance lies solely in recognition of the lesion, at exploratory laparotomy, as the cause of the abdominal symptoms. As it so closely mimics other causes of acute abdominal pain, laparotomy is usually mandatory. The preoperative diagnosis will most often be that of acute appendicitis or cholecystitis. When the expected pathologic lesion is not found, the surgeon should carefully inspect the omentum, especially if serosanguinous fluid is present in the abdominal cavity. It is probable that as surgeons become more aware of this entity, it will be found with greater frequency. At present, the increasing number of such cases being recognized and reported attests to the fact that it may not be so rare as was previously believed.

Summary

A case of idiopathic segmental infarction of the greater omentum is reported, the first such case reported as occurring in a Negro. A brief review of previously reported cases has disclosed that in recent years the lesion has been less rare as more cases are recognized and reported. The etiology, clinical picture, pathology, and treatment of this entity are reviewed. Resection of the omental infarct currently is the treatment of choice. When an expected pathologic lesion causing acute abdominal pain is not found at laparotomy, it is suggested that the omentum be carefully inspected for the presence of an infarction.

References

1. Wrzesinski, J. T.; Firestone, S. O., and Walske, B. R.: Primary idiopathic segmental infarction of omentum; report of two cases. Surgery **39:** 663-668, 1956.
2. Bush, P.: Case of a hemorrhage into great omentum. Lancet **1:** 286, 1896.
3. Johnson, A. H.: Great omentum and omental thrombosis. Northwest Med. **31:** 285-290, 1932.
4. Dugas, J. E., Jr.; Burke, E. L., and Oms, L.: Primary idiopathic segmental infarction of greater omentum; collective review and report of case. Am. J. Gastroenterol. **31:** 382-388, 1959.
5. Halligan, E. J., and Rabiah, F. A.: Primary idiopathic segmental infarction of great omentum; report of four cases and review of literature. A.M.A. Arch. Surg. **79:** 738-745, 1959.
6. Knudson, R. J.: Primary idiopathic segmental infarction of greater omentum. Am. Practitioner **13:** 191-194, 1962.
7. Hallstrand, D. E.: Primary omental infarction. Am. J. Surg. **87:** 563-566, 1954.
8. Leitner, M. J.; Jordan, C. G.; Spinner, M. H., and Reese, E. C.: Torsion, infarction and hemorrhage of omentum as cause of acute abdominal distress. Ann. Surg. **135:** 103-110, 1952.

9. Eger, S. A., and Barto, R. E., Jr.: Primary idiopathic segmental infarction of greater omentum. Am. J. Surg. **78:** 518-519, 1949.

10. Harris, F. I.; Diller, T., and Marcus, S. A.: Hemorrhagic infarction of greater omentum simulating acute appendicitis. Surgery **23:** 206-210, 1948.

11. Pines, B., and Rabinovitch, J.: Idiopathic segmental infarction of greater omentum. Surg. Gynec. & Obst. **71:** 80-85, 1940.

12. Totten, H. P.: Primary idiopathic segmental infarction of greater omentum. Am. J. Surg. **56:** 676-679, 1942.

13. Cagney, M. S., and Milroy, G.: Idiopathic infarction of omentum. Brit. J. Surg. **35:** 95-97, 1947-48.