CASE REPORT

Idiopathic Segmental Infarction of the Greater Omentum

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IDIOPATHIC segmental infarction of the greater omentum is a rather rare condition with symptoms simulating those of acute appendicitis, acute cholecystitis, and other acute abdominal conditions. Because of its rarity and the absence of diagnostic features, it is almost impossible to make a correct diagnosis preoperatively.

A report of a case of obscure cause was made by Bush[1] in 1896. Johnson[2] in 1932, however, seems to have been the first one to report a case due to thrombosis of the vessels of the omentum, and he proposed that this condition be regarded as a definite pathological entity.

Various classifications of infarction of the omentum have been proposed,[3-5] and a simplified version is given in Table I.

TABLE I.—CLASSIFICATION OF INFARCTION OF THE GREATER OMENTUM

Omental infarction due to thrombosis
Hernia or adhesion
Inflammatory
Traumatic
Idiopathic
Omental infarction due to torsion
Primary
Secondary

Torsion infarction of the omentum had been thoroughly discussed by the mid 1940's,[6-11] and the subdivisions of this type, as suggested by Anton, Jennings and Spiegel[6] are recorded in Table II.

TABLE II.—CLASSIFICATION OF INFARCTION OF THE GREATER OMENTUM DUE TO TORSION
(After Anton, Jennings and Spiegel[6])

Primary (synonyms: idiopathic, cryptogenic, pure intra-abdominal)
Secondary
Hernial (external, in sac)
Abdominal
(a) Intrinsic (cysts and tumours)
(b) Extrinsic (associated with pathology of abdominal and pelvic organs and peritoneum)

This communication reports a case of idiopathic infarction of the greater omentum, together with a description of four cases drawn from the records of the same hospital and a review of the literature.

A 32-year-old white man, an engineer, was admitted to the Beverly Hospital, Beverly, Mass., on March 20, 1964, with right upper abdominal pain. He was well until four days before admission, when he was awakened because of the pain. It increased steadily over this period and was associated with nausea but there was no vomiting. The pain did not radiate. There was no diarrhea or melena. The past history was noncontributory.

At the time of admission the patient was a well-developed and moderately malnourished white male in acute distress due to the abdominal pain. Temperature was 99.4° F., pulse rate 90, respirations 24, and blood pressure 140/100 mm. Hg. Tenderness, spasm and rebound tenderness were present in the right upper abdominal quadrant.

The white blood cell count was 16,300 per c.mm., with 83% neutrophils, 14% lymphocytes and 3% monocytes. The hemoglobin value was 15 g. %, urinary diastase was within normal limits.

On the 3rd hospital day, the patient still complained of severe pain. There was no vomiting or radiation of the pain. On the 6th day the pain was much less. He was afebrile, and a repeat white blood cell count was 7500 per c.mm. Meanwhile radiographic studies of the gastrointestinal tract were carried out. By cholecystography there was faint visualization of the gallbladder. A barium meal examination revealed a slightly deformed duodenal bulb, but an active ulcer was not demonstrated.

A barium enema examination suggested the possible presence of a small polyp in the ascending colon.

The patient continued to complain of dull right upper quadrant abdominal pain, and because of its persistence on the 8th hospital day laparotomy was advised, although examination of the abdomen was unremarkable except for slight tenderness in the right upper quadrant.

The laparotomy was performed on the 9th hospital day, the preoperative diagnosis being cholecystitis. A right subcostal incision was made. An inflammatory necrotic mass of omentum, 3 x 7 cm. in size, was discovered. This was closely attached by recent adhesions to the gallbladder, the small bowel and the transverse colon. The appendix was also involved in these adhesions.

The gallbladder, the small and large bowel, and the appendix did not appear grossly to be inflamed. An excision of the infarcted omentum and an appendectomy were performed.

Microscopic examination of the excised portion of the omentum revealed marked edema, hemorrhage and necrosis of fatty tissue. At the periphery, fibroblastic proliferation was noted, with vacuolated macrophages. These findings were consistent with infarction of the omentum. As the omentum was not twisted, the gallbladder and small and large bowels were grossly not inflamed, and as the appendix showed microscopically only a few perivascular lymphocytes in the muscularis, the final diagnosis was idiopathic segmental infarction

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of the greater omentum. Postoperatively the patient did well and was returned home eight days after operation.

ETIOLOGY

Various hypotheses have been advanced as to the etiology of idiopathic segmental infarction of the omentum.[2-4, 10, 12-17] Only Rabinovitch attempted to prove his hypothesis, in a series of experiments using rabbits.[15] Following a forceful pull on the jugular vein, causing injury to its endothelial lining, he observed formation of thrombus in the jugular vein of the rabbit. From such experimental results he concluded that stretching of an omental vein, which might be due to gravity or to physical exercise, could lead to thrombus formation.

Hood and Geraci[14] have reported a case of a 25-year-old man who developed abdominal pain while lifting a 50-lb. box. An infarcted portion of the omentum was located in a triangular recess bounded by the superior surface of the right lobe of the liver, the right side of the falciform ligament, and the adjacent anterior abdominal wall. The sudden onset of abdominal pain at this site during vigorous exertion suggested that this segment of the omentum was suddenly impinged against the liver by contraction of the abdominal wall after the omentum had been forced into this site by flexion of the trunk.

Totten[17] reported a case in which a patient developed pain following the noon meal while moving boxes weighing approximately 120 lb. Totten concluded that in view of the close communication between the blood supply of the stomach and omentum, it is evident that the strain on a thin-walled vein would definitely be increased at the time of vascular congestion following a full meal. During this period, any additional strain from increased intra-abdominal tension brought on by straining or coughing could be sufficient to cause primary rupture of the dependent veins of the omentum, with hemorrhagic extravasation and secondary thrombosis.

In a case of idiopathic infarction of the omentum described by Seley[16] the patient had polycythemia, and a possible causal relation is suggested.

CLINICAL FEATURES

Since Halligan and Rabiah's excellent review[3] of primary idiopathic segmental infarction of the great omentum published in 1959, nine additional cases have been reported in the English literature,[18-23] and the data on a further five cases at the Beverly Hospital are recorded in Table III; this gives a total of 78 cases.

Of these 78, 52 are male and 26 female. The age distribution is shown in Table IV; it seems to be most common in middle age. The literature suggests that it is more common among obese or stocky persons, but in our own five cases there was no apparent correlation with body build.

TABLE III.—FIVE CASES OF IDIOPATHIC SEGMENTAL INFARCTION OF THE GREATER OMENTUM OBSERVED AT THE BEVERLY HOSPITAL

Case	*1*	*2*	*3*	*4*	*5*
Sex	M	M	F	M	M
Age (years)	32	7	46	43	26
Body structure	Medium	Not recorded	Medium	Muscular, large	Obese
Onset of pain	Gradual	Gradual	Gradual	Gradual	Gradual
Location of pain	RUQ	R. mid	Epigastrium	R. mid	R. mid
Nausea	+	+	—	+	—
Vomiting	—	+	—	—	—
Radiation, pain	—	—	—	—	—
Location of tenderness	RUQ	R. mid	Epigastrium	R. mid	R. mid
Spasm	+	—	+	—	+
Temperature (°F)	99.4	100.2	99	98.4	98.2
WBC (per c.mm.)	16,300	14,000	11,000	15,000	11,000
Neutrophils (%)	83	59	51	64	60
Preop. diagnosis	Cholecystitis	Appendicitis	Cholecystitis	Appendicitis	Appendicitis
Location of necrosis	RU	RL	RU	RL	RL

TABLE IV.—AGE DISTRIBUTION OF IDIOPATHIC SEGMENTAL INFARCTION OF THE GREATER OMENTUM

Age	*Cases*
3 - 9	8
10 - 19	3
20 - 29	17
30 - 39	12
40 - 49	11
50 - 59	14
60 - 69	9
70 —	3
Not recorded	1
Total	78

SYMPTOMS

According to the literature the onset of abdominal pain is usually sudden, and often is related to a physical exercise. In our series of five patients, however, onset of pain was gradual, and it was not related to physical exercise. The pain is continuous in character, and usually located on the right side either in the upper or lower quadrant. Radiation of the pain is absent. Patients complain of nausea, but vomiting was observed in only one patient in our series. Physical examination reveals tenderness in the area overlying the infarcted segment of omentum; spasm of the overlying abdominal musculature may or may not be present. Temperature elevation in our patients was rather mild; the temperatures ranged from 98.2 to 100.2° F. The white blood cell count was between 11,000 and 16,300; only a moderate degree of neutrophilic

TABLE V.—PREOPERATIVE DIAGNOSIS IN 78 CASES OF IDIOPATHIC SEGMENTAL INFARCTION OF THE GREATER OMENTUM

Preoperative diagnosis	*No. of cases*
Appendicitis	48
Cholecystitis	17
Perforated ulcer	3
Carcinoma of colon	3
Pancreatitis	1
Mesenteric thrombosis	1
Diverticulitis	1
Retroperitoneal tumour	1
Infarction of omentum	2
"Something rare, as an infarct of omentum"	1
Total	78

leukocytosis was observed. The symptoms usually gradually subside with elapse of time. These findings correspond with those of other authors.

The preoperative diagnoses in the 78 reported cases are shown in Table V. The commonest was acute appendicitis (48 cases). Cholecystitis, perforated ulcer, and carcinoma of the colon were also frequently diagnosed. A correct diagnosis was made preoperatively in only two cases.[3, 22] Site of infarction of the omentum is usually the right side. In all of our five cases, site of infarction was on the right side (right upper in three cases, and right lower in two).

TREATMENT

Treatment of choice is excision of the lesion. The prognosis is almost always good, no recurrence having been reported.

SUMMARY

Five cases of idiopathic segmental infarction of the greater omentum are described, one in detail, making a total of 78 cases reported to date in the English literature.

This condition is commonest among males of middle age. Onset of abdominal pain is sometimes related to ingestion of a heavy meal or physical exercise. The pain is usually continuous and does not radiate. Nausea occurs but vomiting seldom. Mild leukocytosis and mild temperature elevation are usually present.

Idiopathic segmental infarction of the greater omentum is difficult to diagnose preoperatively but should always be included in the differential diagnosis of acute abdominal pain.

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PAGES OUT OF THE PAST: FROM THE JOURNAL OF FIFTY YEARS AGO

TUBERCULOSIS IN CHILDREN

Of late years the suggestion that pulmonary tuberculosis in adult life is a recrudescence or lighting up, after years of quiescence, of an infection acquired during childhood, has been gaining more adherents. Belief in the possibility of the sudden outbreak of an old acquired focus of disease, years after it had ceased to manifest any clinical evidence of its presence, coupled with the almost universally accepted statement that as many as 90 per cent of children at the age of puberty were infected with tubercle, tended to give force to this hypothesis. Hence, the recently published statistics of Veeder and Johnston, of St. Louis, which appear in the June number of the *American Journal of Diseases of Children*, are of interest, especially as they show a wide divergence from the results obtained by von Pirquet and Hamburger in Vienna.

The frequency of primary pulmonary tuberculosis in children under five years old has been a much debated question owing mainly to the evidence having to rest upon clinical signs and skiagrams. The presence of tubercle bacilli in the sputum cannot be determined for obvious reasons, and the interpretation of the physical signs and of the Roentgen picture is necessarily biassed by the fixed belief of the observer in the frequency with which this form of tuberculosis occurs in early life. In the diagnosis of doubtful cases one must be able to exclude the more common disease before one can conclude definitely that one is dealing with the less common. It is true that in those cases where the disease has become dessiminated throughout the body, and has taken the miliary form, at autopsy the pulmonary lesions can often be shown to antedate those in other organs; but autopsies, except in such generalized cases, are not commonly obtainable. Clinicians agree that the utmost reliance can be placed upon the von Pirquet and tuberculin tests for determining the presence of a tuberculous lesion; but whether it is active, latent, or healed can only be decided by clinical examination. Any enquiry then into the proportion of children, other than those with known tuberculous disease, who give a positive reaction to these tests proves of value in determining the frequency of tuberculosis early in life in the cities of this continent as compared with those of the old world.

Working amongst the same class of patient as those from which the Vienna statistics were compiled, namely the poorer classes of society, Veeder and Johnston obtained a very much smaller percentage of positive results. Of 1321 children from a few months up to fourteen years of age, the percentage rises gradually from 1.5 in children under twelve months to 36 per cent, for the period of ten to fourteen years, when recognized clinical cases of tuberculosis are excluded; and to 44 per cent when cases of clinical tuberculosis are included. Hamburger and Monti obtained a positive result in 94 per cent of children between these ages and von Pirquet 80 per cent though the latter used only the test which bears his name. As in both the Vienna and St. Louis cases all the children on whom the tests were made were in hospital for some pathological condition, the percentage of children in the whole community infected with tuberculosis is probably much less than these figures indicate.—Editorial, *Canad. Med. Ass. J.*, 5: 615, 1915.