

flexion deformities of the fingers as a result of loss of extensor tendons and subsequent openings of the interphalangeal joints in burns can often be avoided by early débridement of the eschar and placing the fingers, or fingers and hand, under a flap on the abdomen or thigh.

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170 St. George St.

## CASE REPORTS

### PRIMARY IDIOPATHIC SEGMENTAL INFARCTION OF THE GREATER OMENTUM

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Venous thrombosis is frequently encountered in cases of torsion of the great omentum. Such torsion may be primary (idiopathic) or secondary to hernia or other intra-abdominal lesion. To those cases in which no torsion can be demonstrated and no pre-existing etiological factor can be found, Totten has given the name primary idiopathic segmental infarction.

Our search of the literature has revealed only eight previously reported cases. Pines and Rabinovitch reported six cases in 1940 and Totten in 1942 reported two. Hines described a fatal case of hæmorrhagic infarction of the great omentum secondary to portal vein thrombosis and Berger recorded a case in which cardiac insufficiency was a predisposing factor in hæmorrhagic infarction of the omentum. The latter two cases cannot be considered primary lesions because pre-existing disease undoubtedly influenced their occurrence.

The etiology of the condition is not known. Pines and Rabinovitch are of the opinion that "a possible stretching of an omental vein may occur consequent to some trauma, and this in turn leads to the formation of a thrombus". Totten points out that the blood supply of the stomach and omentum is in close intercommuni-

cation, and he feels that the strain on these thin-walled veins is definitely increased at the time of vascular congestion incident to a full meal. During this period additional strain from increased intra-abdominal tension due to straining, coughing, sneezing or lifting, may be sufficient to cause a primary rupture of the dependent veins of the omentum, with hæmorrhagic extravasation and secondary thrombosis. Our patient gave a history of eating a pound of cheese immediately prior to the onset of his pain, so it would appear that the vascular congestion occurring when the stomach is full does play a part in the etiology of this condition.

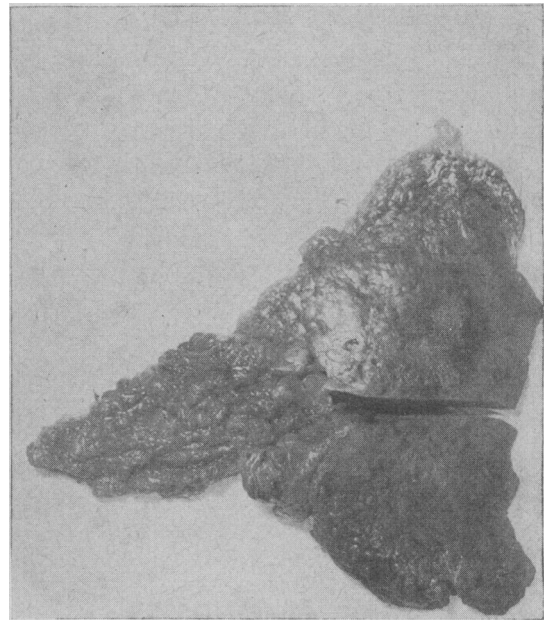


Fig. 1.—Showing the infarcted omental mass. The broad pedicle can be seen showing no evidence of torsion.

Transient torsion must be borne in mind as an etiological possibility, but when one remembers that in true torsion six or more turns of the pedicle are usually seen, this possibility seems unlikely. No evidence of torsion was demonstrated in any cases discussed in the literature or in the one herein presented.

C.J.R., a 29-year old white male was admitted to R.C.N. Hospital, Halifax, the afternoon of December 18, 1944, complaining of severe abdominal pain of three days' duration.

The patient stated he was perfectly well until the night of December 15 when he developed mild crampy generalized abdominal pain following the ingestion of one pound of cheese. He slept fairly well that night only being awakened once with pain. He ate his breakfast the following morning but ate no lunch because of a marked feeling of distension which was not relieved by alkali; the passing of flatus gave some relief.

On the evening of December 16, his pain increased in severity and there was profuse diarrhoea. The latter provided some relief of symptoms. Following the attack of diarrhoea the pain settled in the right lower quadrant.

On December 17 the pain in the right lower quadrant was increasing in severity and he took 1½ ounces of castor oil. On that night he slept very little due to progressing severity of pain. Castor oil was ineffectual and he took epsom salts and ex-lax. Neither of these was effectual. By morning the pain was so severe that he was unable to straighten up in bed.

At no time did he vomit or feel nauseated during the three day siege of pain. He worried constantly about the fact that his bowels were not regular.

The past history was essentially negative.

*Physical examination.*—Temperature 100° F.; pulse 80; respirations 20; blood pressure 135/70; white blood cells 17,620; sedimentation rate 10; urinalysis negative.

The patient was a well developed, well nourished, moderately obese male, quite distressed, unable to lie quietly in bed.

The essential findings were a distended and tympanic abdomen. There was moderate rigidity of the right rectus; he was acutely tender in the right upper quadrant, somewhat less tender in the right lower quadrant. The left side of the abdomen showed nothing abnormal, and rectal examination was essentially negative.

A tentative diagnosis of acute appendicitis was made and a laparotomy performed.

On opening the peritoneum a moderate amount of sero-sanguineous fluid was found in the abdominal cavity. The appendix was essentially negative and certainly could not be considered the cause of his symptoms. Further exploration of the abdomen revealed a large hæmorrhagic necrotic mass involving the right terminal portion of the great omentum. This mass was dark red to purple in colour and firm in consistency. It was attached to the anterior parietal peritoneum by fine adhesions which were easily separated with blunt finger dissection. The mass measured 11 x 7 x 4 cm. and was attached to the main body of the omentum by a broad 6 cm. pedicle which showed no evidence of torsion (Fig. 1). The pedicle was doubly clamped and removed. Thorough examination of the peritoneal cavity revealed no other disease. The abdominal incision was closed in layers.

The pathological report\* read as follows: "Re the portions of omental mass taken from C.R., I am quite in agreement with the diagnosis of 'segmental infarction of the greater omentum'.

"From the sections it is obvious that there is a venous blockage somewhere. Whether it is a thrombosis or tumour condition I cannot tell, but the veins are all tremendously engorged and there is extravasation of blood into the surrounding fat tissue. There is some acute inflammatory infiltration, particularly over the peritoneal surface, with proliferative reaction in the stroma. I also found some deposits of hæmosiderin in the histiocytes. This could make it look as though it had been there for a reasonable length of time, possibly a week or so. Probably if more blocks were taken proximal to the infarction you might be able to demonstrate thrombosis or some other factor causing this condition, but, from these sections of course, it is impossible to say what the etiology is. I have never seen such a case in my experience."

#### DISCUSSION

*Diagnosis.*—In seven of the previously reported cases the preoperative diagnosis was acute appendicitis, while in the other cases subacute

perforation of a peptic ulcer was suspected, with appendicitis as an alternative diagnosis. Our case was considered acute appendicitis before operation and it is felt that clinically the preoperative diagnosis of primary idiopathic segmental infarction will rarely be made. However, the condition must be borne in mind when the findings in the appendix will not explain the symptoms present. In the symptomatology constipation has been a frequent finding. These patients have been "bowel conscious" and have usually resorted to cathartics and enemata before hospitalization.

*Pathology.*—At operation three patients of the Pines and Rabinovitch series exhibited sero-sanguineous fluid in the peritoneal cavity and in four instances the omental infarct was adherent to the anterior parietal peritoneum by a fine exudate. These findings were present in the case presented here. All the reported cases including the present one have exhibited a hæmorrhagic necrotic infarct in the terminal portion of the right or distal free border of the omentum. This mass has usually been red to purplish-black in colour and firm in consistency. No evidence of torsion of the pedicle has been present in any case.

#### SUMMARY

1. A case of primary segmental infarction of the omentum is presented.
2. The etiology of this condition is unknown.
3. The preoperative diagnosis is extremely difficult.
4. The treatment is relatively simple and consists of removal of the infarcted mass. When operation is performed early the results are universally satisfactory.

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\* Courtesy of Dr. W. L. Robinson, Professor of Pathology, University of Toronto.