

MESENTERIC VENOUS THROMBOSIS WITH OPERATION AND CURE

Report of a Case

WILLIAM E. LOWER, M.D. and McCLEERY GLAZIER, M.D.

Mesenteric vascular occlusion is considered by most authorities to be a relatively rare disease. This condition was first described by Triedman in 1843 and Virchow described its pathology in 1847 but not until 1895 when Elliott reported his case was operation first carried out successfully. Since 1895 there have accumulated numerous case reports, classifications, and theories regarding its etiology, frequency, and symptomatology. The purpose of this paper, however, is not to review the literature but to report a case which presented an interesting problem in diagnosis and treatment.

REPORT OF CASE

The patient was a white, married man, 39 years of age, whose occupation was inspector of golf clubs. He entered the Clinic on Dr. William J. Engel's service complaining chiefly that rather severe, constant pain in the epigastrium had been present for 20 hours.

Present Illness: About 20 hours previously, while working, this patient had a sudden attack of acute pain in the upper part of the abdomen; the pain did not radiate but remained localized. It was not colicky in nature and within a few minutes subsided to a dull ache. Since the onset, he had experienced acute exacerbations which necessitated morphine for its relief. There was no immediate associated nausea or vomiting but a few hours after the onset the patient stated that a small amount of the contents of the stomach had been vomited. There was no hematemesis. There was no previous history of stomach trouble or disease of the gallbladder. He had never been jaundiced and there had never been acholic stools. He gave no history of attacks of appendicitis previously and this pain was not associated with diarrhea, constipation, or blood in the stools. The bowels had moved the afternoon of the day of onset but had not moved the day of entrance to the Clinic.

Past History: The past history and history by systems revealed no significant findings. As stated previously, the patient denied having had disease of the stomach or gallbladder, and there was no history of cardiac pathology or blood dyscrasia. He absolutely denied previous illness in any form.

Physical Examination: The patient was a well developed, fairly well nourished, white man, of about the stated age, who appeared to be

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experiencing quite severe abdominal pain. The skin was somewhat cold and clammy, the temperature was 98.4° F., the pulse rate 72, respirations 20, and blood pressure 145 systolic, 100 diastolic. The pupils were pinpointed and did not react to either light or accommodation. The patient had received one-fourth grain of morphine about one hour before entering the Clinic. Otherwise the eyes were entirely normal. The findings of the remainder of the examination of the head and neck were entirely within normal limits.

The chest was symmetrical and expansion was equal; the lungs were clear to auscultation and percussion; there was no dullness or tubular breathing; the mediastinum was negative. The heart was not enlarged and there were no murmurs; the sounds were of good quality, the rate was 72, and the rhythm was normal. The peripheral vessels were apparently normal.

The abdomen was moderately distended throughout and mildly spastic above the umbilicus. There was no rigidity and no definite point of maximum tenderness to palpation, but the patient complained of generalized soreness while being examined. No scars and no masses were present. The liver edge could not be felt; the gallbladder and spleen were not palpated.

Rectal examination gave entirely normal findings; the prostate was normal in size, shape, and consistency. Neurological findings were all within normal limits; no pathological reflexes could be elicited.

The clinical impression of the examiner was: (1) subacute perforated peptic ulcer; (2) subacute pancreatitis; (3) volvulus; and (4) Meckel's diverticulum.

Laboratory Studies: Examination of the urine showed the reaction to be 5.5; there was four plus albumin with numerous hyaline and granular casts, 10 to 12 red blood cells, and 3 to 5 white blood cells per high power field. Examination of the blood showed 5,700,000 red cells, 23,150 white cells, 100 per cent hemoglobin, 85 per cent neutrophils, 13 per cent lymphocytes, and 2 per cent monocytes. The level of the blood sugar was 152 mg. and of the blood urea 54 mg. per 100 cc. two and one-half hours postprandial. The Wassermann and Kahn tests gave negative reactions.

It was the impression of the referring physician, Dr. N. Kiefer, Geneva, Ohio, that this was possibly an atypical attack of renal colic and he requested complete urological examination which was carried out immediately by Dr. Engel. A routine plain film of the kidneys, ureters, and bladder was entirely normal except that the roentgenologist stated that there was considerable gas in the upper portion of the intestines. Cystoscopic examination gave entirely normal findings.

Catheters were passed to both kidney pelves and a bilateral pyelogram showed both pelves to be normal.

About four o'clock in the afternoon the patient was admitted to the hospital for observation. He was made comfortable with hot stupes to the abdomen and a rectal tube to relieve the distention. No morphine was given at this time. About three hours after admission, the temperature rose to 101.4° F., the pulse rate was 128, and respirations 24. At this time, the patient was in quite severe pain, the pain being confined particularly to the upper part of the abdomen and epigastrium. As before, the pain did not radiate but remained localized and was not associated with nausea, vomiting, or diarrhea. The abdomen was definitely spastic in its upper half but quite soft and distended in the lower half. Percussion revealed free fluid in the right flank. No board-like rigidity was present and no masses were palpable. It was our impression that, although the findings were certainly not typical of a perforated viscus, this was the most probable intra-abdominal pathology.

At 8:30 in the evening, about four and one-half hours after admission, Dr. William E. Lower performed an exploratory laparotomy, using a right paramedian incision. Upon opening the peritoneum, there was a gush of approximately 2000 cc. of blood-stained abdominal fluid which had the consistency of a transudate. Upon removal of this fluid, the presenting loop of bowel was seen to be definitely injected and distended, but no evidence of an exudative peritonitis could be seen. The mesentery of this loop was found to be apparently normal. This loop of bowel was examined proximally where it was found to be entirely normal. The stomach, gallbladder, and duodenum were likewise normal. The liver was palpated and was found to be normal. The presenting loop of bowel was then examined distally where, about midway in the ileum, it was found to merge with a loop of bowel approximately 18 inches long which was reddish-purple in color with petechial hemorrhages over the serosa of the bowel. It was moderately distended and the mesentery of this segment was of a purplish-blue color with dilated veins; the mesentery was definitely thickened and edematous. No arterial pulsations were palpated and although the veins were dilated they were not indurated. There was no evidence of torsion of this involved segment of gut. Distal to this involved segment the small intestine again became apparently normal with the exception of moderate injection of the serosa; the bowel was collapsed and the mesentery apparently was normal. The large bowel was entirely normal and collapsed.

It was the impression of the surgeon that this lesion was one of beginning intestinal gangrene due to vascular occlusion; however, it was impossible to state whether the occlusion was arterial or venous in

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origin. The operation consisted of resection of the involved segment of bowel, care being taken to remove all the involved mesentery and bowel. The resection was carried out into normal tissue. The mesentery was then approximated by means of continuous chromic catgut sutures and an end-to-end anastomosis of the cut ends of the bowel was performed by the closed method. About eight inches above the anastomosis an ileostomy was performed for the purpose of decompressing the bowel and to relieve tension on the suture lines. The abdomen was closed without drains.

The patient was placed immediately upon a strict Alonzo Clark routine with continuous stomach suction. Adequate fluids were given parenterally. The following morning his condition appeared to be fair, his temperature was 101° F., pulse rate 140, and respirations 28. The abdomen was flat and the Wangensteen was functioning properly as was the ileostomy. During the course of the day he remained in fairly good condition but that afternoon his temperature rose to 104° F., pulse rate to 160, and the respirations were 32. Examination of the abdomen at this time revealed mild distention but no rigidity. It was felt that this reaction was probably due to a beginning peritonitis so the parenteral administration of sulfanilamide with equal amounts of sodium bicarbonate intravenously was prescribed. The initial dose was 90 grains daily for the first 48 hours which was followed by 60 grains every 24 hours thereafter for the following three days and then it was discontinued. Mild acidosis developed and this condition was combatted by means of the intravenous administration of sodium bicarbonate. The number of white blood cells dropped to 9000 with 85 per cent neutrophils and remained at about this level for the following five days.

During the first three postoperative days, the evening temperature rose to about 104° F. with tachycardia in proportion to this elevation; however, the temperature began to approximate normal thereafter and on the fifteenth postoperative day the highest elevation was 99.5° F. and on the nineteenth postoperative day it became normal and has remained so.

On the fourteenth postoperative day, because of the immense loss of fluid through the ileostomy, it was closed by means of four interrupted chromic catgut sutures.

The patient was discharged from the hospital on the thirty-first postoperative day. The pulse, temperature, and respirations were normal, his abdomen was flat, and he was gaining in weight and strength. The bowels moved daily, aided occasionally by small enemas. He showed a mild secondary type of anemia and this was being corrected by

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means of ferrous sulphate given orally and Parke-Davis A-B-D capsules. Chemical study of the blood gave entirely normal findings.

In conclusion, we believe that this case is one of early mesenteric venous thrombosis because:

1. Microscopic sections through the involved bowel and mesentery showed extensive edema, extravasation of blood in the tissues, and venous engorgement. There was no evidence of ulceration of the mucous membrane of the bowel, a condition which is manifested relatively early in arterial occlusion.

2. The presence of 2000 cc. of blood-tinged abdominal fluid.

3. The realization that wet gangrene occurs with arterial occlusion but always following a primary ischemic gangrene and that, if this condition existed in this case, we should expect to find ulcerative lesions in the mucosa of the bowel.