## CASE REPORTS

# Massive Hemorrhage Following Small Bowel Biopsy

M. VIDINLI, M.D., M.S.* and
J. M. FINLAY, M.D., F.R.C.P.[C], F.A.C.P.,† *Toronto*

INCREASING interest in the study of gastrointestinal problems, both physiological and pathological, has led to the development of a considerable number of techniques which have found practical use in the understanding of gastrointestinal disease and its treatment. Histological studies of small bowel were not practical clinically before the introduction of oral intubation techniques for obtaining duodenal and jejunal biopsies in 1956.[1, 2] Since then, this contribution has become established technique, a variety of small bowel biopsy tubes have become available and a considerable experience with normal and abnormal small bowel histology has accumulated.

In the earliest reports, this procedure seemed free of any serious complications.[1-3] Subsequently, a few instances of moderate gastrointestinal bleeding following biopsy were reported.[4-6] More recently, massive gastrointestinal hemorrhage has been described after jejunal biopsy with the Shiner tube.[7, 8] The present report describes another instance of massive gastrointestinal hemorrhage following small bowel biopsy with the Rubin multipurpose suction biopsy tube. It serves to emphasize one possible risk of the procedure, the need for care in its performance, and the importance of clinical selection of patients.

In September 1962, a 55-year-old man was admitted to the Toronto General Hospital complaining of weakness and failure to gain weight. He had undergone a subtotal Polya-type gastrectomy for peptic ulcer complicated by recurrent vomiting and three episodes of hemorrhage, 14 years previously. Two years later, a benign chest lesion was removed at thoracotomy in another hospital. No record of the pathology of this chest "tumour" was available. In 1958, he began to suffer moderate upper gastrointestinal discomfort relieved by vomiting, which was recurrent thereafter. By July 1962, the discomfort had localized to the epigastrium and was suggestive of peptic ulcer. In July 1962, he was hospitalized because of an episode of rectal bleeding. Hemorrhage from a stomal ulcer was suspected, but complete clinical and radiological studies failed to reveal the site of bleeding. In hospital he was treated conservatively and transfused with four units of blood. No further bleeding occurred and he was discharged to be followed up in clinic.

He continued to complain of weakness and weight loss, as well as chronic fatigue, and he was readmitted in September 1962 for investigation of his symptoms and his poor nutritional state. At this time his nutritional status suggested either inadequate caloric intake due to a small gastric reservoir or post-gastrectomy malabsorption syndrome without diarrhea. He appeared thin, chronically ill and apathetic. There was no superficial lymphadenopathy and no evidence of bleeding tendency or of skin rash. Glossitis was absent and the oral cavity was normal. There was no evidence of tetany or of bone pain. He did not appear anemic and there was no dependent edema. Except for a right thoracotomy scar, no abnormalities were found during chest examination. The blood pressure was 120/80 mm. Hg and the heart rate was 76 per minute. The heart rhythm was regular. There was no evidence of heart enlargement or of congestive failure. An old midline abdominal incisional scar was present, and a large ventral hernia occupied the epigastrium and right upper quadrant of the abdomen. A normal liver edge was palpable 2 cm. below the right costal margin in the midclavicular line during inspiration. No other abdominal masses or organs were palpable and there was no abdominal tenderness. The remainder of the physical examination was not remarkable.

Urinalysis revealed no abnormality. The hemoglobin was 12.3 g. %, white count, 7700 and the differential count, normal. The total serum protein was 7.0 g. % and the albumin:globulin ratio was normal. Serum calcium was 9.9 mg. % and the serum phosphorus, 4.0 mg. %. Serum cholesterol was 230 mg. %. The prothrombin time was normal. Serum glutamic oxaloacetic transaminase (SGOT), serum bilirubin and alkaline phosphatase values were all normal. The blood urea nitrogen level was normal. The serum carotene was 58.2 μg. % (normal range: 70-250 μg. %). The Schilling test was abnormal and revealed a 24-hour urinary excretion of 6.8% of the oral dose of vitamin $B_{12}$. A chemical fat balance study revealed a fecal fat excretion of 8.9% of daily fat intake. The D-xylose tolerance test showed a normal peak blood level of xylose (44.8 mg. % at the first hour) with a low renal xylose excretion (2.1 g.) during the five-hour interval. The gastric aspirate contained no free hydrochloric acid and cultured "many *B. coli*". The laboratory data were felt to be compatible with the diagnosis of intestinal malabsorption following gastrectomy, possibly related to abnormal upper gastrointestinal tract bacterial flora.[9-11] A peroral small bowel biopsy was performed with the Rubin tube. A satisfactory biopsy was obtained from the efferent jejunal loop about 10 cm. distal to the gastrojejunal stoma, without difficulties. Histologically, the jejunal mucosa was normal (Fig. 1). The depth of the biopsy was not unusual and included the usual small fragment of muscularis mucosae. No vascular abnormality was present in the biopsy.

Except for slight nausea and anorexia, the patient was asymptomatic until nine hours after the biopsy, when he had a hematemesis of about 500 c.c. volume. Hemoglobin, repeated at this time, was 7.5 g. %. Cardiovascular collapse did not ensue and although the cardiac

*Medical Research Council Fellow in Medicine.
†Associate, University of Toronto and Toronto General Hospital.

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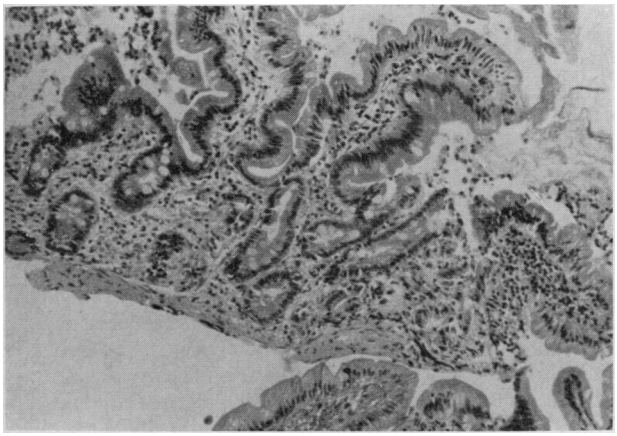

Fig. 1.—Normal jejunal biopsy.

rate was 140 per minute and the blood pressure 90/60, his pulse was strong and regular, his extremities were warm, and examination of the abdomen revealed no abnormalities.

During the next 12 hours, he was transfused with six units of blood and, although his clinical state remained satisfactory, his hemoglobin level failed to rise beyond 8 g. %. At this time he was examined with the gastroduodenal fibrescope in an effort to visualize the site of continued bleeding. During this examination a questionable ulcer crater was visualized on the jejunal side of the gastrojejunal stoma, but there was no evidence of free bleeding from this source. Conservative management was continued for 16 hours after gastroscopy, during which he required a further nine units of blood intravenously to maintain his hemoglobin at about 8 g. %.

Laparotomy was advised 30 hours after the jejunal biopsy. At operation, the gastric remnant and gastrojejunal stoma were normal. No stomal ulcer was present. About 10 cm. distal to the gastrojejunal stoma, a hematoma was present on the serosal surface of the efferent jejunal loop. This portion of the jejunum was inverted through a gastrotomy incision. On the mucosal surface of the efferent loop, at the site of the serosal hematoma, there was a small ulcer covered with fresh blood clot. Pulsatile bleeding issued from a small artery in the ulcer base. After ligation of this vessel, a vagotomy was performed and the abdomen was closed.

In the postoperative period, the patient developed a right lower lobe atelectasis and bronchopneumonia, both of which responded satisfactorily to treatment. The remainder of his course in hospital was uneventful and he was discharged on the nineteenth postoperative day.

## DISCUSSION

All of the various tubes now employed for small bowel biopsy use suction to draw intestinal mucosa through a small hole in the capsule. A knife blade, operated at the proximal end of the tube, cuts off the portion of mucosa entrapped in the capsule. The usual resulting biopsy is superficial and consists only of mucosal structures. The small blood vessels in such a biopsy have not been felt to be a likely source of serious bleeding.

Two tubes, the Crosby capsule and the Rubin tube, have been used at the Toronto General Hospital for small bowel biopsies. In the past three and a half years, 159 biopsies have been obtained. In the first six months of this interval, stool benzidine tests were done regularly after biopsy and no evidence of post-biopsy bleeding was encountered. Since then, stool benzidine tests have been discontinued. Until the mishap described in this report, no previous serious complication of small bowel biopsy had been observed. The site of the mucosal lesion discovered at operation and the presence of persistent arterial bleeding in its base bear witness that the massive hemorrhage in this patient was caused by jejunal biopsy.

The clinical and investigative value of the technique is confirmed by its increasing reported use in the differential diagnoses of gastrointestinal disease. Its application to the study of jejunal histology in malabsorption syndrome has widened and now allows pathological diagnoses of lymphoma and other neoplasms of bowel, inflammatory disorders, regional enteritis, sarcoidosis, Whipple's disease and other small bowel disease. To date, the reported incidence of serious complications of the procedure is low. Small bowel biopsy is a blind procedure, and the risk of hemorrhage from biopsy must be considered. As a routine precautionary measure, historical and laboratory absence of a bleeding tendency should be established before the biopsy is undertaken. Hypoprothrombinemia, often present in malabsorption syndrome, should be corrected by parenteral vitamin K before biopsy.

Concerning the technique of small bowel biopsy, care should be taken to avoid excessive suction to obtain the mucosal specimen. The shallow depth of the usual jejunal biopsy has been attributed to the ability of the mucosa to slide over underlying submucosal structures. It is conceivable that excess suction could draw not only mucosa but also larger nutrient submucosal vessels into the capsule at the time of biopsy. To avoid this hazard, the Rubin tube is equipped with a manometer by which negative pressure can be measured. Although the recommended negative pressure has been used in the majority of biopsies, occasionally it has been necessary to use slightly greater negative pressure because of failure to obtain the biopsy in the initial attempt. During jejunal biopsy in the patient reported herein, the first attempt was unsuccessful and a slightly higher suction than usual was used in the second successful attempt. It is of interest that, in section, the resulting biopsy was of shallow depth and contained no unusual or large blood vessels. The incidence of massive hemorrhage following small bowel biopsy will likely remain low if adequate technical care is taken and clinical precautions are observed. This complication should be emphasized as a possible risk in the use of any of the currently available instruments for small intestinal biopsy.

SUMMARY

Despite adequate precautions, massive upper gastrointestinal hemorrhage complicated peroral small bowel biopsy in the patient reported herein. Laparotomy was required to effect hemostasis. This report serves to emphasize the importance of selection of patients and the need for technical care in the performance of this procedure.

Dr. J. M. Finlay,
Suite 430, Medical Arts Bldg.,
Toronto 5.

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# Abscess of the Myocardium Complicating Infarction:
## Report of Two Cases

ALLAN KATZ, M.D.,* *Toronto*

MYOCARDIAL abscesses have been encountered in pyemia and bacterial endocarditis. Flaxman[1] in 1943 reported 29 cases in 14,160 autopsies, an incidence of 0.2%. Saphir[2] reported 32 among 5626 autopsies, an incidence of 0.56%.

Abscess complicating myocardial infarction secondary to atherosclerotic coronary occlusion is uncommon. This report concerns two such cases in which purulent pericarditis was also present.

CASE 1.—A 63-year-old white man was admitted with crushing retrosternal pain radiating into both arms. He gave a history of precordial pain on effort during the two months prior to admission. An electrocardiogram (ECG) revealed recent anterior myocardial infarction. The serum glutamic oxalacetic acid transaminase (SGOT) level two days after admission was 66 units and the erythrocyte sedimentation rate was 60 mm. per hour (Westergren).

He was treated by means of bed rest and anticoagulants. During the first week, the temperature varied between 100 and 101° F. It rose to 105° F. three weeks after admission. A blood culture taken at this time revealed no growth. Sputum cultures were negative for pathogens. The patient continued to run a low-grade fever up to 101° F. He suddenly became hypotensive and died during his seventh week in hospital.

At autopsy, the pericardial cavity contained 20 ml. of greenish-yellow fluid. In the region of the apex, a loculated collection of pus measuring approximately 40 ml. was present in the pericardial space.

The heart weighed 565 g. The left ventricle appeared hypertrophied and dilated. The valves were unremarkable. The anterior wall of the left ventricle and adjacent interventricular septum at the apex revealed recent and old infarcts. The myocardium in this region was the site of an abscess adjacent to the loculated collection of pus in the pericardial space.

Fig. 1.—Case 1. The myocardial fibres are necrotic. There is an acute inflammatory-cell infiltrate with abscess formation. (HPS; original magnification, × 80.)

The coronary arteries showed marked atherosclerosis, with organizing thrombi in the left anterior descending and right coronary arteries and a recent thrombus in the left circumflex artery.

Microscopic examination confirmed the recent and old infarcts in the anteroseptal region of the left ventricle. The myocardium in this area revealed the presence of an abscess with massive necrosis and acute inflammatory-cell infiltration (Fig. 1). The pericardium showed acute fibrinopurulent inflammation (Fig. 2).

Other significant findings were bilateral pleural effusions (750 and 800 ml.), pulmonary congestion and edema and congestion of liver and spleen.

Smears of the abscess revealed a moderate number of Gram-negative bacilli which proved to be *E. coli* on culture. No source of infection was found on microscopic examination.

*Assistant Pathologist, St. Michael's Hospital.