RECTAL ULCERATION DUE TO ISCHEMIC NECROSIS*

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RECTAL ULCERS MAY be associated with many diseases. They may occur with benign or malignant neoplasms; they may be secondary to infectious diseases such as tuberculosis, amebic and bacillary dysentery, schistosomiasis, fungi, syphilis, and lymphogranuloma venereum. They are present in ulcerative colitis, and may result from accidental or self-imposed trauma, fecal impaction, as a complication of the injection treatment of hemorrhoids, or as the result of irradiation of adjacent neoplasms. Even aberrant gastric mucosa has been a reported[4] cause of rectal ulceration. In terminal uremia diffuse rectal ulceration may develop. The following case is one of rectal ulceration due to ischemic necrosis as a result of arteriosclerosis in the small hemorrhoidal arteries.

CASE REPORT

A. T., a 36-year-old Norwegian male, was first admitted to the Lankenau Hospital on the Medical Service, March 8, 1949, with the complaint of intermittent frontal and orbital headaches of 2 months' duration. He had a past history of a "bladder operation" in 1936, following an injury to his left flank, with resulting gross hematuria. He had been told in 1947, after a routine examination, that he had high blood pressure.

Complete studies were performed with the following pertinent findings: The blood pressure was 230/140. Fundoscopic examination revealed a Grade IV hypertensive retinitis. An intravenous urogram revealed an atrophic non-functioning left kidney. On the basis of unilateral renal disease as the cause of the hypertension, a left nephrectomy was performed by the Urological Service. The kidney weighed 40 Gm. and microscopically revealed chronic pyelonephritis. The blood pressure was not lowered by this procedure. The patient was discharged and 3 weeks later was readmitted to Surgical Service "A" and underwent, in two stages, bilateral thoracolumbar sympathectomy and splanchnicectomy, with exploration of the adrenal glands. Recovery was uneventful. There was a marked reduction in the blood pressure, which, however, gradually returned to the previous levels after 6 months. He was seen twice yearly in our Follow-Up Clinic, and during the ensuing 38 months gained weight, was symptom free, and was actively employed. The blood pressure remained at the previous hypertensive levels.

On December 3, 1952, 44 months after sympathectomy, the patient was readmitted to the Lankenau Hospital with the chief complaint of rectal bleeding and tenesmus for 6 months. He had lost 34 pounds in weight. His blood pressure was 260/120. Fundoscopic examination showed marked attenuation of all arteries; there were no hemorrhages or exudates and there was no papilledema. On rectal examination there was normal sphincter tone with tenderness and induration in the rectum. Proctoscopy the day prior to admission had not revealed any pathologic condition. There was a marked secondary anemia. Studies for blood dyscrasias were negative. A barium enema was normal. Proctoscopy was repeated six days later and showed an ulceration 2 cm. above the mucocutaneous line on the right anterolateral surface of the rectum. The ulcer had a gray base with smooth edges. There was no inflammation of the adjacent mucous membrane and no induration on palpation of the ulcer base. Repeated stool examination for amebae, ova and parasites were negative. Serology and Frei tests were negative. Biopsies of the ulcer were reported as "interstitial and ulcerative proctitis." Fifteen days after admission, a third biopsy was obtained under anesthesia, as previous examinations of this area had been most painful and distressing to the patient.

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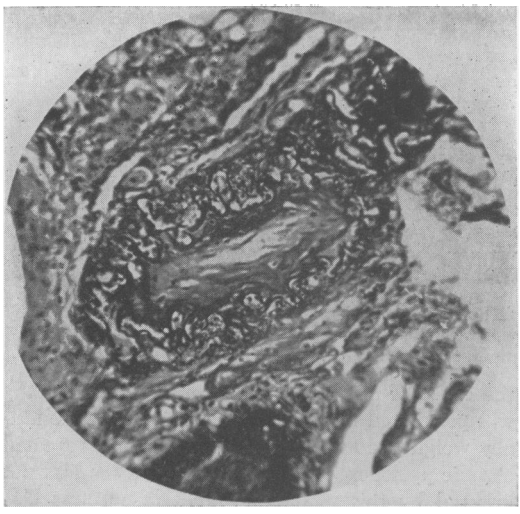

FIG. 1. Surgical biopsy of rectal ulcer showing a small subserosal artery near the ulcer base. Occlusive fibrohyaline thickening with reduplication of the elastica is evident.

Curettage, dark field examination for spirochetes, and Papanicolaou smears were all negative. This biopsy was reported as "ulcer of the rectum, probably arteriosclerotic in etiology" (Fig. 1). Muscle biopsies subsequently obtained showed no evidence of periarteritis nodosa.

During this hospitalization the patient was given courses of terramycin, aureomycin, streptomycin, cortisone and diodoquin. Despite this, rectal pain, bleeding, tenesmus, and lower abdominal discomfort persisted. The patient was discharged on the 36th day of hospitalization but little improved.

The patient's final admission was 3 weeks later, on January 23, 1953, because of a continuation of symptoms. He appeared emaciated and pale and in much distress due to continuous rectal pain. The blood pressure was 198/132. Again a marked secondary anemia was present. Urinalysis revealed a 3+ albumin, but the blood urea nitrogen was normal. Two days after admission, the anemia having been corrected by blood transfusions, proctoscopy was repeated and disclosed persistence of the ulceration. At laparotomy the mesenteric vessels were grossly normal to inspection and palpation. There was no pathologic change apparent upon exploration of the pelvic and abdominal contents. A Lahey type of colostomy was performed, placing the distal mucous fistula in the suprapubic area by means of a stab incision.

Postoperatively, within 24 hours, necrosis of the functioning colostomy was apparent. The patient was weak, pale, and perspired profusely. His blood urea nitrogen had risen on the sixth postoperative day to 78.8 and his urinary output was diminishing daily despite oral, intravenous and subcutaneous fluids and electrolytes. The patient had become mentally confused. On the eight postoperative day, gas and watery fecal-colored material was coming from the anus—the colostomy was non-functioning. The abdomen was soft with active peristalsis. Despite charcoal given orally for three days an ileo-rectal fistula was not confirmed. The patient gradually failed, with acidosis and uremia increasing despite calculated fluid and electrolyte replacement. He expired on the 14th postoperative day.

The pertinent autopsy findings revealed a slightly hypertrophied heart with scattered atheromatous desposits in the coronary arteries; the lungs and upper gastro-intestinal tract were normal. Compensatory hypertrophy of the right kidney had occurred.

The terminal ileum was dull gray and distended by gas. It was densely adherent to the peritoneal reflexion posterior to the bladder and communicated with a retroperitoneal abscess. This abscess, in turn, communicated with a large perforation in the anterior wall of the rectum. Both colostomies

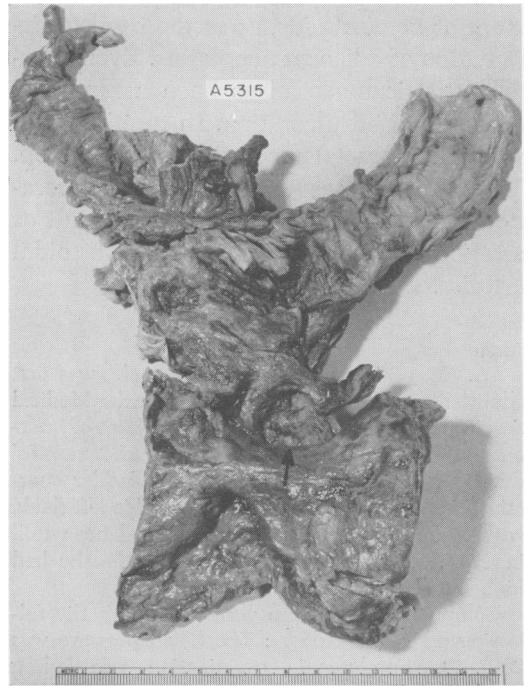

FIG. 2. Gross specimen as removed at autopsy. The upper right segment of bowel is sigmoid the mucosa of which is intact but pale. Numerous large ulcers are apparent, with the site of perforation marked by an arrow. The terminal ileum is in the upper left and has become adherent to the pelvic peritoneum to form the ileo-rectal fistula.

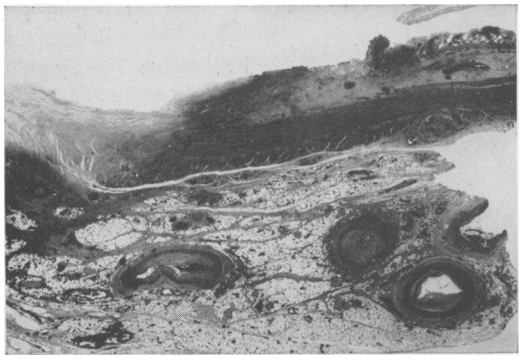

FIG. 3. Low power magnification showing necrosis of rectal mucosa and muscularis on the left. The pararectal small arteries are in various stages of occlusion caused by intimal proliferation.

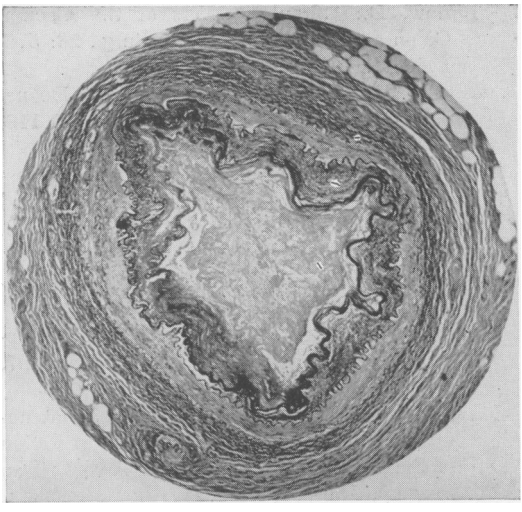

FIG. 4. Small hemorrhoidal artery totally occluded by fibrohyaline intimal proliferation.

were gangrenous terminally. The ascending, transverse, and descending colon were grossly normal and contained inspissated fecal material. The rectum was dilated, the walls were thickened and necrotic. Multiple ulcers involved the rectal mucosa and one of the larger of these, the site of the preoperative ischemic ulcer, had perforated anteriorly to form the retroperitoneal abscess (Figs. 2 and 3). The inferior mesenteric artery was patent. Thickening was present in the walls of the superior and middle hemorrhoidal arteries, the smaller branches of which were grossly obstructed.

Sections of the rectal ulcers revealed that the mucosa had largely been replaced by an exudate of fibrin, leukocytes and necrotic debris. The muscularis was necrotic and the veins were engorged. In the outer layers of the rectum the arteries were occluded by intimal fibrohyaline changes. Sections of the inferior hemorrhoidal artery showed complete occlusion by fibrohyaline thickening (Fig. 4).

DISCUSSION

A survey of the literature has failed to reveal any case of rectal ulceration secondary to small vessel arteriosclerosis. This is not surprising in view of the plentiful blood supply of this area.

Vague gastro-intestinal symptoms have been attributed to diffuse arteriosclerosis, and in cases of hypertension and renal disease.[5, 9, 10] Abdominal apoplexy[3] and rectal bleeding[6, 8, 11] have been associated similarly with diffuse arteriosclerotic changes. Postmortem evaluation of the degree of involvement of the mesenteric vessels in arteriosclerosis[13] have shown marked freedom of these vessels from such degenerative changes. Spontaneous rupture of the rectum[1, 7] is a rare occurrence and no relationship to arteriosclerosis has been cited in previous reports. Simple benign ulcers of the large bowel[2, 12] have been infrequently encountered and their occurrence in the rectum is rare. In no such instances has vascular insufficiency been proposed or found as an etiological factor. Zeek and Phair[14] in 1931 presented three cases of gangrene and ulceration of large segments of the gastrointestinal tract which they attributed to atherosclerosis of the smaller vessels. Critical analysis of their cases, however, indicates the possible presence of other contributing factors in the pathological picture.

SUMMARY

A case is reported of death from perforation of a rectal ulcer due to ischemia resulting from arteriosclerosis of the small vessels, and associated with hypertension. No other similar case has been found in the literature.

BIBLIOGRAPHY

1 Allen, C. P.: Spontaneous Rupture of the Rectum. Lancet, 2: 378, 1948.

[2] Barlow, D.: "Simple" Ulcers of the Cecum, Colon and Rectum. British J. Surg., **28**: 575, 1940-41.

[3] Berk, J. E., N. S. Rothschild and J. C. Doane: Intra-Abdominal Apoplexy. Ann. Surg., **113**: 513, 1941.

[4] Ewell, G. H., and R. H. Jackson: Aberrant Gastric Mucosa in Rectum with Ulceration and Hemorrhage. Wisconsin M. J., **38**: 641, 1939.

[5] Jablons, B.: Gastrointestinal Symptoms in Hypertension and Renal Disease. Review of Gastroenterology, **11**: 246, 1944.

[6] Lockhart-Mummery, J. P.: Causes of Hemorrhage from the Rectum. British M. J., **1**: 997, 1938.

[7] McLanahan, S., and M. L. Johnson: Spontaneous Rupture of Lower Colon with Evisceration of Small Intestine Through the Anal Orifice. Surgery, **18**: 478, 1945.

[8] Odessky, L.: Arteriosclerosis: Its Role in the Pathogenesis of Rectal Hemorrhage (Case Report with Autopsy Findings). Ann. Int. Med., **36**: 1121, 1952.

[9] Riseman, J. E. F., and S. Weiss: The Symptomatology of Arterial Hypertension. American J. Med. Sci., **180**: 47, 1930.

[10] Shapiro, S., and J. E. Astrachan: Proctological Manifestations in Systemic Disease. Review of Gastroenterology, **14**: 786, 1947.

[11] Stone, H. B.: Large Melena of Obscure Origin. Ann. Surg., **120**: 582, 1944.

[12] Turell, R., J. S. Krakauer and A. deL. Maynard: Colonic and Anorectal Function and Disease, Collective Review. Internal Abstracts of Surgery, **96**: 313, 1953.

[13] Wartman, W. B.: The Incidence and Severity of Arteriosclerosis in the Organs from 500 Autopsies. Am. J. Med. Sci., **186**: 27, 1933.

[14] Zeek, P., and J. Phair: Mesenteric Small Vessel Sclerosis with Ulceration and Gangrene of the Enteric Tract. Am. J. Med. Sci., **181**: 548. 1931.