A Rare Cause of Ischemic Colitis:
Polyarteritis Nodosa*

MICHAEL K. WOOD, M.D., DON R. READ, M.D., AVRAM R. KRAFT, M.D.,
TELLY M. BARRETA, M.D.

Polyarteritis nodosa has been associated with a variety of secondary intestinal complications but has not previously been reported to cause fulminating, gangrenous ischemic colitis. Recent experience with a patient who had this unusual complication led us to review the pertinent literature and to report this case.

Report of a Case

A 76-year-old black man was in good health except for mild, compensated heart failure until six weeks prior to admission when he developed the “flu.” Four weeks prior to admission he noted the onset of malaise, anorexia, nausea, dull aching pain in both calves and progressive weakness most marked in his legs. He had limited his intake to liquids and had lost 30 pounds of weight. Occasional constipation was his only other gastrointestinal complaint. Progressive weakness led to his admission to Cook County Hospital in May 1977. At the time of admission, he had a cough productive of greenish-yellow sputum.

The patient had a two-year history of congestive heart failure treated with digoxin, furosemide, and a potassium supplement. On physical examination he appeared cachectic but in no acute distress. The rectal temperature was 102.9 F, pulse 100 and regular, respiration 20, blood pressure 110/60 mm Hg, height 5 feet 10 inches and weight 120 pounds. He had patches of blood on his nasal mucosa. The lungs were clear but his breathing was shallow. The abdomen was scaphoid and soft, bowel sounds were decreased, and he had diffuse tenderness most marked in the left lower quadrant. Diffuse tenderness was present on the rectal examination and brown stool was positive for occult blood. Proc-tosigmoidoscopy showed edematous, red and ulcerated mucosa extending to the left lower sternal border with radiation to the axilla. A fourth heart sound was present and the jugular venous pressure was mildly increased. The abdomen was scaphoid, soft and nontender without organomegaly or palpable masses. Bowel sounds were normal. Rectal examination showed an enlarged prostate with gaujac-negative stool. Mild pedal edema was present to the mid-calf. On neurologic examination, he had diffuse, generalized weakness of all four extremities. Deep tendon reflexes were absent in the lower extremities and reduced in both upper extremities.

The admission hemogram showed a microcytic, hypochromic anemia and leukocytosis with a shift to the left. Urinalysis, coagulation profile, serum electrolytes, enzymes, thyroid studies, serology, and serum tests for collagen diseases were normal. The only notable abnormalities were a decreased serum albumin and magnesium. The digoxin level was within the therapeutic range; the electrocardiogram showed normal sinus rhythm and complete left bundle branch block. Chest and abdominal roentgenograms were unremarkable.

The patient’s early hospital course was marked by persistent fever (100.0–102.9 F). Urine, sputum, and blood culture results were negative. A battery of skin tests suggested that he was anergic. A therapeutic trial ofisoniazid and rifampin was initiated for suspected miliary tuberculosis but had no effect. Because of the clinical suspicion of bacterial endocarditis, he was treated with ampicillin, penicillin G, and streptomycin which eradicated an incidental urinary tract infection of Serratia marcescens. Urinalysis consistently showed 1+ proteinuria and 10 to 25 erythrocytes three weeks after admission. An extensive search was made for a collagen disease or an occult malignancy. Contrast x-ray studies, ultrasonography, scans, computerized axial tomography, angiography, and biopsies of the liver, axillary lymph node, temporal artery, and bone marrow were either normal or showed nonspecific abnormalities.

The patient’s febrile course stopped for unexplained reasons three months following admission by which time his weight had fallen another 6½ pound. On August 31, 1977, the patient had left iliofemoral venous thrombosis documented by impedance plethysmography and venous Doppler studies and was treated with heparin and warfarin. Two weeks later, he developed bloody diarrhea followed by diffuse, dull, abdominal pain. Surgical consultation was obtained. His pulse was 116, respiratory rate 22, temperature 98.6 F orally, and the blood pressure was 148/92 mm Hg. The abdomen was scaphoid and soft, bowel sounds were decreased, and he had diffuse tenderness most marked in the left lower quadrant. Diffuse tenderness was present on the rectal examination and brown stool was positive for occult blood. Proctosigmoidoscopy showed edematous, red and ulcerated mucosa with a necrotic area at 18 cm. Paracentesis was negative. Leukocyte count was 14,900, the hematocrit 31% and blood urea nitrogen 37 mg/dl. The prothrombin time and partial thromboplastin time were prolonged to twice normal. Abdominal roentgenograms showed a nonspecific gas pattern. An operation was performed with the preoperative diagnosis of gangrenous ischemic colitis secondary to a low flow state. Resuscitation with saline, packed erythrocytes and fresh frozen plasma preceded the laparotomy.

At laparotomy, the colon and focal regions of approximately 25 cm of terminal ileum appeared cyanotic. Gentle palpation of the colon and terminal ileum caused a violaceous discoloration of the bowel. The remainder of the abdominal exploration was normal. Pulsations were felt in the celiac, superior mesenteric, and inferior mesenteric arteries. Due to the patient’s recent iliofemoral venous thrombosis, a clip was placed on the inferior vena cava below the renal veins. The entire colon and terminal ileum were resected. The rectal stump was oversewn, an ileostomy created and tube gastrostomy performed.

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Address reprint requests to: Dr. Kraft, Division of General Surgery, Cook County Hospital, 1825 West Harrison Street, Chicago, Illinois 60612.
ISCHEMIC COLITIS DUE TO POLYARTERITIS NODOSA

The pathologist reported prominent, tortuous, thickened, and focally nodular blood vessels bulging into the serosal surface of the entire colon and in focal areas of the ileum (Fig. 1). The serosal and intramural blood vessels showed thickened walls and narrow or occluded lumina. The mucosa of the ileum and colon revealed large longitudinal ulcerations with ragged, granular bases and necrotic deposits on the surface. The ulcers had irregular, hyperemic margins and were located mostly at the antimesenteric border of the bowel. Microscopically, sections of the ulcerated areas of the ileum and colon showed denuded mucosa with flattening of the mucosal folds, marked vascular congestion and hemorrhage in the lamina propria. Submucosal and subserosal small and medium sized arteries showed luminal narrowing or occlusion, fibrinoid necrosis of the intima, and extensive degeneration of the medial and adventitial layers which were densely infiltrated by neutrophils and eosinophils (Figs. 2 and 3). The pathologic diagnosis was ischemic enterocolitis secondary to polyarteritis nodosa.

The patient made a slow but steady recovery and his BUN returned to normal by the fourth postoperative day. When the diagnosis of polyarteritis nodosa was established, he was started on 60 mg of prednisone daily. He was transferred to a chronic care hospital 5 months following admission. Three weeks after transfer, his condition deteriorated, he became anorectic, and his BUN increased to 96 mg/dl. His death one week later was presumed to be secondary to renal failure. A postmortem examination was refused.

Discussion

Ischemic Colitis: As described by Marston et al., ischemic colitis develops as a result of impaired colonic blood supply which is first noted at the mucosal surface. This ischemic insult is enhanced by the particular virulence of the colonic bacteria which then invade the colon wall. Marcuson has divided the causes of ischemic colitis into 1) large artery occlusion, 2) "non-occlusive" states, 3) venous occlusion, and 4) vascular insufficiency caused by increased intraluminal pressure. In a large artery occlusion, such factors as atherosclerosis, thrombosis or embolism, surgical vascular interruption, mesocolon hematoma, and Buerger's disease appear to play key roles. Small-vessel disease, vasoconstriction, and hypovolemia account for the "non-occlusive" variety. Oral contraceptives and venous thrombosis may play a role in the "venous occlusion" state associated with ischemic colitis. Increased intracolonic pressure proximal to an