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Pericarditis—An Extraintestinal Complication of Inflammatory Bowel Disease

M. ARSHAD ABID, MD NORMAN GITLIN, MD, FRCP, FRCPE Fresno, California

The occurrence of pericarditis in association with inflammatory bowel disease is rare and not widely recognized.¹ Extraintestinal manifestations of inflammatory bowel disease are well recognized, and they may be severe and unrelated to the extent or degree of activity of the colitis.² We report a case of pericarditis complicating the clinical course of ulcerative colitis.

Report of a Case

The patient, a 37-year-old man, was admitted to hospital in August 1989 because of weakness, anorexia, nausea, and lower abdominal pain. He had a diagnosis of ulcerative colitis dating 12 years, when he initially had several episodes of rectal bleeding. Over the years, he had isolated exacerbations of his ulcerative colitis, which was confined to the descending colon and rectum and which responded well to a combination of oral prednisone and sulfasalazine. He had taken the sulfasalazine irregularly over the years. His most recent flare-up was six weeks before this admission when he had cramps and diarrhea (four stools per day) with blood and mucus. Following a colonoscopy, he received a short tapering course of oral prednisone for four weeks and sulfasalazine, 2 grams per day, and he had been free of rectal bleeding for the past three weeks.

On admission to the hospital, the patient was afebrile with a pulse rate of 102 beats per minute, and he had a blood pressure of 140/70 mm of mercury. There was evidence of mild postural hypotension in keeping with his dehydrated state. There was no pulsus paradoxus, and his venous pressure was not elevated. Heart sounds were normal, and there was no gallop or pericardial friction rub. The results of the respiratory, abdominal, and neurologic examinations were within normal limits. On the third day in hospital, the patient complained of a substernal chest pain unaffected by posture or respiration. He continued to be afebrile, and his heart rate dropped to 54 beats per minute; there was no pulsus paradoxus, and his blood pressure was

(Abid MA, Gitlin N: Pericarditis—An extraintestinal complication of inflammatory bowel disease. West J Med 1990 Sep; 153:314-315)

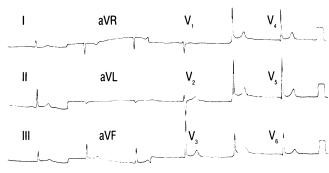


Figure 1.—An electrocardiogram taken on the first day of chest pain shows ST segment changes compatible with pericarditis.

120/80 mm of mercury. Apart from the above, the findings of the physical examination were unchanged from those on his admission. An electrocardiogram (ECG) showed sinus bradycardia with ST segment elevation in all the chest and limb leads except in leads I, aVL, aVR, and V1 (Figure 1). An echocardiogram did not reveal a pericardial effusion. The creatine kinase level was 430 units per liter; eight hours later, it was 414 units per liter, and the MB fractions were 0.06 and 0.11, respectively. Other laboratory studies included normal serum albumin and globulin levels and a leukocyte count of 16.0×10^9 per liter (with a normal differential count). A diagnosis of pericarditis associated with ulcerative colitis was made, and the patient was treated with indomethacin, 25 mg orally three times a day. He rapidly responded symptomatically to this therapy, feeling better in a day. There was no recurrence of his chest pain, and the indomethacin was discontinued after three days without a relapse of his condition. The ECG reverted to normal on the third day.

Comment

The wide spectrum of extraintestinal complications of ulcerative colitis and Crohn's disease is well known and includes inflammatory lesions of the skin (pyoderma gangrenosum and erythema nodosum), aphthous buccal ulceration, ocular lesions (uveitis and conjunctivitis), muscular skeletal disorders (arthritis, sacroiliitis, ankylosing spondylitis), vascular complications (thromboembolic disease, vasculitis, arteritis), and a variety of hepatic lesions (fatty liver, pericholangitis, sclerosing cholangitis, cholangiocarcinoma, liver abscess, drug hepatitis, immunopathic chronic active hepatitis, amyloidosis, and cholelithiasis). Pericarditis, myocarditis, and pleural effusions are uncommonly associated with inflammatory bowel disease, yet their association should not come as a surprise in view of their occurrence in other so-called autoimmune diseases such as systemic lupus erythematosus and rheumatoid arthritis.1 The pathogenesis of the extraintestinal complications of inflammatory bowel disease is unknown, and the actual frequency of their occurrence is uncertain. In large case studies, between 25% and 36% of patients with inflammatory bowel disease had at least one such associated disease.^{3,4} Most cases are associated with ulcerative colitis rather than Crohn's disease. The pericardial complications of inflammatory bowel disease run a course independent of the gut disorder.5.6 Pericarditis has been reported to antedate the diagnosis of ulcerative colitis by two years, and it can persist despite a remission of the colitis.6 A report of pericarditis manifesting weeks after a subtotal colectomy for ulcerative colitis indicates the imprecise temporal relationship between the two disorders.5 The possibility of association between the pericarditis and the medications pre-

From the Medical Services, Veterans Administration Medical Center, Fresno, California, and the Department of Medicine, University of California, San Francisco, School of Medicine.

Reprint requests to Norman Gitlin, MD, Medical Service (111), VA Medical Center, 2615 E Clinton Ave, Fresno, CA 93703.

scribed for ulcerative colitis has been raised. Sulfasalazine has been effective in resolving the pericarditis, but exacerbations of pericarditis have occurred during the drug therapy. The possibility of a rare association between 5-aminosalicylic acid enemas and pericarditis has been recently raised, and there is little to implicate the 5-aminosalicylic acid enemas as a causative factor relating to the pericarditis.

The pericarditis—and, at times, myocarditis—can range from a mild inflammation to a pericardial tamponade that may require drainage. In the milder presentations, salicy-lates or nonsteroidal anti-inflammatory agents (indomethacin) are effective therapy. In the more severe cases, the use of prednisone or an intensification of the dosage of steroids (if already prescribed for the inflammatory bowel disease) is recommended. The natural history of the pericarditis varies, and recurrent episodes of pericarditis or myocarditis have been reported. Physicians treating patients with inflammatory bowel disease should always consider pericarditis or myocarditis in the differential diagnosis of chest pain.

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Rattlesnake Capsule-Induced Salmonella arizonae Bacteremia

LIONEL A. CONE, MD WILLIAM H. BOUGHTON, PhD LAWRENCE A. CONE, MD LEVI H. LEHV, MD Rancho Mirage, California

Salmonella arizonae, described 50 years ago in colonized chuckwallas (Sauromalus obesus), horned lizards (genus Phrynosoma), and Gila monsters (Heloderma suspectum) and known as Salmonella dar-es-salaam,¹ was first identified from a human case of pyrexia in the town in east Africa by that name. The organism is part of the normal reptile intestinal flora and, although not pathogenic for snakes and turtles,² causes disease in monotremes,³ turkeys, chickens, goats, and humans.⁴ Placed in a separate group by Edwards

(Cone LA, Boughton WH, Cone LA, et al: Rattlesnake capsule-induced Salmonella arizonae bacteremia. West J Med 1990 Sep; 153:315-316)

and co-workers in 1943 and subsequently labeled *Arizona hinshawii*,⁵ it is now once again classified under a single species, *Salmonella enterica*, which is subdivided into six subspecies. Based on DNA studies and biochemical tests, *A hinshawii* and *S enterica* subspecies *arizonae* are identical.

S arizonae was recently cultured from blood specimens of two patients at the Eisenhower Medical Center, Rancho Mirage, California. Both patients had Hispanic surnames, suggesting to one of us (Li.A.C.), the District Health Officer of northern Los Angeles County, that the ingestion of rattle-snake capsules, a common practice among Mexican-Americans in the Los Angeles area, could be the cause of infection. We were able to confirm this in one of the two patients and suspect it in the other.

Report of Cases

Patient 1

The patient, a 71-year-old Mexican-American woman, while receiving corticosteroids for rheumatoid arthritis, had fever and chills develop, accompanied by left hip and leg pain. She was admitted to hospital a few days later. Culture of three blood specimens obtained on admission grew out S arizonae. A computed tomographic scan of the pelvis revealed findings consistent with an abscess of the left iliac muscle. A subsequent radionuclide bone scan yielded evidence of arthritis involving the left sacroiliac joint. Initial therapy with intravenous ceftriaxone sodium, 2 grams daily for 16 days, in this penicillin-allergic woman failed to eradicate the abscess, which subsequently completely resolved with a regimen of chloramphenicol, 4 grams daily. The patient was then queried as to her use of medications. She readily admitted ingesting several "folk remedies" while in Mexico recently but did not specifically recall the use of rattlesnake capsules. None of these medications were available for culture. S arizonae was not identified in a stool specimen.

Patient 2

The patient, a 72-year-old Mexican-American man, had metastatic melanoma develop in late 1988 that involved pleura, lungs, bone, and liver. A regimen of combination chemotherapy using dacarbazine, vinblastine sulfate, cisplatin, and carmustine was started. He was favorably responding to chemotherapy when pancytopenia and fever developed, requiring admission to hospital in January 1989. Culture of three initial blood specimens grew out S arizonae. He responded promptly to empiric therapy using vancomycin hydrochloride (later discontinued) and aztreonam. Cultures of blood were subsequently sterile. S arizonae could not be identified in stool specimens. The patient readily admitted taking capsules of vibora de cascabel (rattlesnake) to assist in the treatment of his metastatic melanoma. More than 50 of these capsules were retrieved and sent to the California State Health Department, and S arizonae was identified.

Discussion

The use of rattlesnake powder or meat to treat cancer, diabetes, arthritis, and skin disorders or other sundry ailments is common in Mexican folk remedies. With the rattles left in place for identification, the reptile is decapitated, skinned, dried in the sun, pulverized, placed in capsules, and then sold under various names: vibora de cascabel, polvo de vibora, and carne de vibora. The capsules are sold without prescription in farmacias in Hispanic neighborhoods in Los Angeles and Mexico and are readily available in several communities in California.

From the Los Angeles County Department of Health, and the Departments of Pathology and Medicine, Eisenhower Medical Center, Rancho Mirage, California

Reprint requests to Lawrence A. Cone, MD, Probst Professional Bldg, Ste 308, 39000 Bob Hope Dr, Rancho Mirage, CA 92270.