Letters to the Editor are welcomed. They may report new clinical or laboratory observations and new developments in medical care or may contain comments on recent contents of the Journal. *They will be published, if found suitable, as space permits. Like other material submitted for publication, letters must be typewritten, double-spaced, and submitted in duplicate. They must not exceed* ***two*** *typewritten pages in length. No more than five references and one figure or table may be used. See "Information for Authors" for format of references, tables, and figures. Editing, possible abridgment, and acceptance remain the prerogative of the Editors.*

Extraintestinal Hodgkin's Disease in a Patient with Crohn's Disease

To the Editor: The relationship between Crohn's colitis and non-Hodgkin's lymphoma is well established.[1,2] Only a few publications that associate Crohn's disease (CD) with Hodgkin's disease (HD) have been reported, however. We report an unusual case of a patient who developed HD without intestinal involvement 2 years after underlying CD was diagnosed.

In July 1998, a 26-year-old woman was diagnosed with CD. She was treated with mesalazine (500 mg tid) and low-dose methylprednisolone (5 mg/d). In March 2000, the patient presented with acute exacerbation of her symptoms, including persistent fever, lethargy, recurrent diarrhea, abdominal pain, and weight loss. A physical examination revealed palpable lymph nodes in different regions. A computed tomographic scan showed cervical, axillary, supraclavicular, and mediastinal lymph node enlargement and nodular splenic lesions. A histological examination of a node biopsy showed nodular sclerosing HD. Bone marrow aspiration and biopsy were negative for involvement. The HD was staged as III BS according to the Costwolds Staging Classification, and six courses of combination chemotherapy with alternating ABVD (doxorubicin, bleomycin, vinblastine, and dacarbazine) were administered. During complete restaging, the patient experienced complete remission of both diseases. At her last follow-up examination in November 2002, she was well and asymptomatic with no evidence of HD, and she needed no treatment for CD.

A statistically significant higher probability of developing gastrointestinal lymphoid tumors has been noted in patients with CD.[1,2] Most lymphomas reported in this setting are commonly a high-grade non-Hodgkin's type of B- or T-cell linage, whereas cases of primary HD are extremely rare. Patients with CD also may be at increased risk for developing extraintestinal lymphoma and leukemia without intestinal involvement.[2] To our knowledge, only three reports of extraintestinal HD without gastrointestinal involvement have been reported in patients with CD.[1,3,4] These reports suggested that this presentation might be evidence for a common cause of the two conditions.

Most patients with HD in Crohn's colitis are men in their 30s, and the median interval between the time of CD diagnosis and the occurrence of lymphoma is 8.9 years.[5] Our case does not entirely match these criteria, especially because only 2 years elapsed between the diagnoses of CD and HD.

The pathogenesis of lymphoma associated with CD is still unclear. Hypotheses include immunologic defects associated with CD, prolonged therapy with immunosuppressive agents, and/or exposure to x-rays.[5] Our patient had not taken any immunosuppressive drugs other than low-dose steroids before the diagnosis of HD was made. Even though the two conditions occur in similar age groups, it is our opinion that the likelihood that they will occur together by chance alone on a number of occasions seems remote.

The appropriate treatment of unusual cases of HD associated with CD follows the guidelines of lymphoma treatment primarily. Our patient received six cycles of ABVD chemotherapy, and there has been no evidence of the recurrence of either disease, and neither methylprednisone nor mesalazine was administered in the 4 years after the patient underwent chemotherapy.

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A Different Therapeutic Approach in Patients with Severe Ulcerative Colitis: Hyperbaric Oxygen Treatment

To the Editor: Hyperbaric oxygen treatment (HBOT) has been used in patients with some diseases of the gastrointestinal system, such as radiation enterocolitis, CD, and experimental colitis; however, the experience with HBOT in patients with ulcerative colitis (UC) is limited.[1,2] We present a case of clinical remission after HBOT in a

patient with UC who did not respond to treatment with 5-amino salicylic acid, methylprednisolone, and azathioprine.

A 52-year-old woman with relapsing UC was admitted because of a 3-month history of bloody diarrhea with mucus, abdominal pain, fatigue, and loss of appetite. She had diarrhea (16–18 bowel movements/d), tenesmus, and macroscopic bleeding. Her first laboratory workup revealed anemia and thrombopenia. Albumin was 2.9 g/dl, serum iron was 26 μg/dl, and ferritin was 5 ng/ml. A colonoscopic examination revealed left-sided UC with Grade IV endoscopic activity (Truelove scale).[3] The histopathologic findings supported the clinical and endoscopic findings. The stool cultures, stool microscopy, and tests for *Clostridium difficile* were negative. Oral mesalamine 4 g/d and methyl prednisolone 40 mg/d were started. The patient became asymptomatic within 2 weeks. Corticosteroids were gradually tapered and then stopped within 4 weeks.

A colonoscopy performed 4 months after the cessation of prednisolone was completely normal. The mesalamine dose was decreased gradually to 1.5 mg/d thereafter. Within 4 weeks, the patient's symptoms recurred with the same degree of severity as the first attack. The mesalamine dose was increased to 4 mg/d. Methylprednisolone 40 mg/d was reinitiated. There was no response to this treatment at 3 weeks. Azathioprine 50 mg tid was added while the steroids were tapered. At the end of the 31st week, the patient's symptoms were the same, with almost no response.

At this stage, HBOT with 100% oxygen at 2.0 atm absolute for 120 min/d was initiated. HBOT was administered in a double-lock multiplace chamber for 35 days. In the second week of HBOT, the patient's diarrhea and rectal bleeding decreased and her abdominal pain was remarkably improved. Twenty days after the initiation of HBOT, the patient was asymptomatic, and she was discharged to home to continue HBOT on an outpatient basis. Mesalamine and azathioprine at the same dosages were continued during and after HBOT. Despite clinical remission, the patient's endoscopic activity in a colonoscopy performed after the cessation of HBOT was Grade II. More than 6 months after the cessation of HBOT, the patient was still in remission.

In recent years, there have been reports about heparin as a therapeutic option in patients with severe UC in accordance with suggestions that microthromboembolism may play a role in the etiopathogenesis of UC.[4,5] In light of the fact that the success of heparin has been attributed to its effects on colonic microcirculation, the application of HBOT in these patients seems reasonable. With this therapeutic concept in mind, HBOT was tried previously with successful results in a patient with toxic megacolon.[1] Clinical remission that lasted more than 2 months with the use of HBOT also was reported recently in a patient with severe UC refractory to medical treatment.[2] Although clinical remission was achieved in the patient, there was a relatively small decrease in the endoscopic severity of the disease. We also were able to induce clinical remission in our case, but the decrease in endoscopic activity was smaller. The difference in our case is that the clinical remission was longer than that in the previously reported case. The two cases reported previously and our case point out that HBOT can be effective in inducing remission in patients with severe UC.

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Arsenic and Heavy Metal Poisons

To the Editor: In April 1997, shortly after opening an antique automobile restoration business, I gradually began to experience several debilitating illnesses. My symptoms included severe burning sensations in my hands and feet, which ultimately resulted in what is known as peripheral neuropathy, or numbness in the extremities. In addition, I experienced gastrointestinal burning and severe indigestion. Walking became difficult for me, because heavy metal poisons cause nerve damage in the spine and legs. I also experienced chest pains.

I saw several doctors, and they were all admittedly baffled. The cost of my doctors' bills and hospital outpatient testing grew to more than $20,000. After a few months, one of my doctors performed a heavy metals test and discovered that I had been poisoned with arsenic. The doctor was required by law to notify police investigators and a toxicologist. An in-depth investigation ensued, and it was determined that my business partner was responsible for the poisoning. The investigation revealed that my partner secretly had obtained a $100,000 insurance policy on my life, listing himself as the sole owner and

lone beneficiary of the policy. A surprise search of my partner's home and premises was conducted, and arsenic was found in a makeshift laboratory in one of his outbuildings. Arsenic and other poisons also were found hidden behind an electrical panel at our place of business.

In January 2000, a 1-week trial with a sequestered jury was held, and my partner was found guilty on all counts of first-degree attempted murder, theft, and tax violations. He was sentenced to 31 years in prison. The discovery of the poison in my body was made just in time to save my life. My doctors were looking only for normal illnesses. Had the heavy metals test not been run, no further testing would have taken place, and my business partner would have walked away, having committed the perfect murder.

I am confined to a wheelchair because of nerve damage in my spine and legs that was caused by the poisoning. Although I have not regained my ability to walk, some feeling has returned to my arms and hands, and I am able to type again. I am 63 years old, and I wanted to tell this story before I died. Even more important, I would like physicians everywhere to read this story so that they will look for and recognize the signs and symptoms of heavy metal poisoning. I think that it will help to save the lives of others in the future.

My story was recently featured in an episode of *Forensic Files* ("The Metal Business"; http://www.forensicfiles.com/Ffiles.asp?Fid=1230) on the Court TV cable television channel, but many facts were omitted because of the limited time allowed for the program. I have not written this letter for the purpose of personal gain. I have newspaper articles, personal writings, and other related documents scanned into my computer if anyone would like to learn more about my case. I also am willing to share a videocassette tape of the *Forensic Files* episode if anyone is interested in viewing it.

The expert toxicologist, who was also the expert witness for the prosecution in my case, was the head of forensic reviews at the University of Tennessee Medical Group in Memphis. He stated that he was amazed that I had lived to take the witness stand and that I did not have to "speak from the grave." I will be glad to answer any questions that *Southern Medical Journal* readers may have about my case.

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Every great advance in science has issued from a new audacity of imagination.

—John Dewey (1859–1952)