tilation may also produce many of the dysesthesias reported for fish poisoning and should be considered in the differential diagnosis.

Prevention

The highest incidence of poisoning generally occurs around coral reefs that have been recently disturbed.[1,2] This can occur by natural means such as storms or hurricanes or by man-made events such as dredging operations, blasting or waste disposal.[1,2] Reef fish from these areas are especially prone to carry the toxin. In tropical and subtropical island areas one should avoid eating the larger predacious reef fish or the tropical moray eel. Finally, one should never eat the liver, viscera or roe from any tropical fish.[1,2]

As illustrated by these two cases, the problem might be seen by physicians in any part of the world who care for travelers who have been to endemic areas. In addition, with international commerce and modern fish-processing techniques, potentially toxic fish may be sold in inland markets far away from their source.

Addendum

Since this report was written, Engleberg and coworkers[23] described the effects of ciguatera poisoning in a population defined by exposure to toxic fish.

REFERENCES

1. Withers NW: Ciguatera fish poisoning. Annu Rev Med 1982; 33:97-111
2. Halstead BW: Poisonous and Venomous Marine Animals of the World—Vol 2. Princeton, NJ, Darwin Press, 1978, pp 325-400
3. Hughes J, Merson M: Fish and shellfish poisoning. N Engl J Med 1976 Nov 11; 295:1117-1120
4. Lawrence DL, Enriquez MB, Lumish RM, et al: Ciguatera fish poisoning in Miami. JAMA 1980 Jul 18; 244:254-280
5. Gudger EW: Poisonous fishes and fish poisonings, with special reference to ciguatera in the West Indies. Am J Trop Med 1930 Jan; 10: 43-55
6. Bagnis R: Clinical aspects of ciguatera (fish poisoning) in French Polynesia. Hawaii Med J 1968 Sep-Oct; 28:25-27
7. Bagnis R, Chanteau S, Chungue E, et al: Origins of ciguatera fish poisoning: A new dinoflagellate, *Gambierdiscus toxicus* Adachi and Fukuyo, definitely involved as a causal agent. Toxicon 1980; 18(2):199-208
8. Helfrich P, Banner AH: Experimental induction of ciguatera toxicity in fish through diet. Nature 1963 Mar 9; 197:1025-1026
9. Hessel DW, Halstead BW, Peckman NH: Marine biotoxin I. Ciguatera poison: Some biological and chemical aspects. NY Acad Sci Ann 1960 Nov; 90(3):788-794
10. Scheuer PJ, Takahashi W, Tsutumi J, et al: Ciguatoxin: Isolation and chemical nature. Science 1967 Mar 10; 155:1267-1268
11. Li K-M: Ciguatera fish poison: A cholinesterase inhibitor. Science 1965 Mar 26; 45:1581
12. Rayner MD, Kosaki TI, Felneth EL: Ciguatoxin: More than an anticholinesterase. Science 1968 Apr; 160:70-71
13. Setliff J, Rayner M, Hong SK: Effect of ciguatoxin on sodium transport across the frog skin. Toxicol Appl Pharmacol 1971 Mar; 18: 676-684
14. Rayner MD, Szekerczes J: Ciguatoxin: Effects on the sodium-potassium activated adenosine triphosphatase of human erythrocyte ghosts. Toxicol Appl Pharmacol 1973 Mar; 24:489-496
15. Bagnis R, Kuberski T, Laugier S: Clinical observations on 3,009 cases of ciguatera (fish poisoning) in the South Pacific. Am J Trop Med Hyg 1979 Nov; 28:1067-1073
16. Russell FE: Ciguatera poisoning: A report of 35 cases. Toxicon 1975 Nov; 13:383-385
17. Halstead BW: Fish poisonings—Their diagnosis, pharmacology and treatment. Clin Pharmacol Ther 1964 Sep-Oct; 5:615-627
18. Jones HR Jr: Acute ataxia associated with ciguatera-type (grouper) tropical fish poisoning. Ann Neurol 1980 May; 7:491
19. Banner AH, Sasaki S, Helfrich P, et al: Bioassay of ciguatera toxin. Nature 1961 Jan 21; 189(4760):229-230
20. Granade HR, Cheng PC, Doorenbos NJ: Ciguatera I: Brine shrimp (*Artemia salina L.*) larval assay for ciguatera toxins. J Pharm Sci 1976 Sep; 65:1414-1415
21. Hokama Y, Banner AH, Boylan DB: A radioimmunoassay for the detection of ciguatoxin. Toxicon 1977 Jul-Aug; 15:317-325
22. Foodborne botulism. Rockville, Md, US Dept of Health and Human Services, FDA Drug Bull 1981 Nov; 11:20-22
23. Engleberg NC, Morris JG, Lewis J, et al: Ciguatera fish poisoning: A major common-source outbreak in the U.S. Virgin Islands. Ann Intern Med 1983 Mar; 98:336-337

Alcohol-Related Pain and Hodgkin's Disease

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PAIN ASSOCIATED with alcohol ingestion is a relatively specific, though uncommon, symptom of Hodgkin's disease.[1] The pain, which may develop almost immediately after an alcoholic drink is swallowed, can be very intense and has on occasion caused chronically alcoholic persons to abstain from liquor. This dramatic symptom may be the sole presentation of Hodgkin's disease and occur before there is palpable lymph node enlargement or constitutional symptoms that might otherwise alert an examining physician to the disease. In this setting the recognition of the association of alcohol-related pain with Hodgkin's disease takes on particular importance.

Report of a Case

The patient, a 30-year-old computer salesman, was referred for rheumatologic evaluation because of recurrent right arm pain. He had been in good health until four months before he consulted a physician. At that time he first noted the development of diffuse aching in his right shoulder and arm, which would occur abruptly after a few swallows of beer or hard liquor. The pain would persist for about 30 minutes and then would gradually resolve despite his continuing to imbibe. He had no accompanying swelling or color changes of his arm, weakness or incoordination. He voluntarily tried eliminating alcohol and was free of the pain, only to have it recur with reintroduction of alcohol. Several weeks after the patient's arm pain began, similar aching developed in the left side of his mandible. Despite his alcohol-related pain, the patient continued to drink 3 to 6 oz of alcohol a day. He said he did not have any constitutional symptoms such as fever, night sweats or weight loss, and felt otherwise well.

On physical examination the patient appeared well developed and in good general health. No abnormalities of his right shoulder or arm, neck or mandible were detected. There was no lymphadenopathy or hepatosplenomegaly. Routine blood tests, including an alkaline phosphatase study, showed normal values. A chest roentgenogram showed a poorly defined left mediastinal mass overlying the main pulmonary artery. Chest computerized tomographic scan confirmed the presence of a smooth-bordered mass in the left upper mediastinum. A mediastinal biopsy was diagnostic for nodular sclerosing Hodgkin's disease. A bone marrow biopsy specimen, bone scan and lymphangiogram showed no additional sites of involvement.

En bloc irradiation of the cervical, supraclavicular, axillary, mediastinal and hilar lymph nodes (Mantle

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therapy) was instituted and within two weeks the patient's alcohol-related pain had completely resolved.

Comment

The association between alcohol-related pain and Hodgkin's disease was first noted in 1950.[2] Published surveys that followed reported an incidence ranging from 15 percent to 30 percent.[3,4] More recent observations, however, suggest a much lower frequency in the range of 1.5 percent to 5 percent.[1,5] The decreasing incidence of this symptom has been ascribed either to a possible change in the natural history of the disease or to the fact that patients with Hodgkin's disease tend to be identified earlier.[1,6]

The pain induced by alcohol has been described as either sharp and stabbing or dull and aching. A patient frequently links the discomfort with alcohol ingestion and may voluntarily discontinue its use. Characteristically, the pain begins within minutes after a patient swallows some of an alcoholic drink and subsides within 30 minutes to a few hours. In our patient, the pain subsided despite his continuing to drink.

Retrosternal pain has been noted with mediastinal lymph node involvement whereas with diseased axillary or cervical nodes, pain may be felt in the shoulder, neck or arm. The abdomen and groin are also sites of alcohol-related pain, and back pain with or without radiation down the leg has been described. When disease is present at several sites pain may not be felt in all of them. An important observation is that tumor can invariably be identified at or near the site of the alcohol-related pain, if not at the time of initial presentation, then when relapse occurs.[5]

Several features seem to characterize patients with Hodgkin's disease who have alcohol-related pain. Nodular sclerosing Hodgkin's disease is the predominant histologic type, and a higher than expected incidence of mediastinal involvement has been reported.[3,5] More women than men tend to have this symptom despite a two-to-one male predominance for the disease.[5]

The pathogenesis of alcohol-related pain is not well understood, though it is believed to result from localized swelling with stretching of the capsule of the affected lymph nodes or increased pressure in diseased marrow that expands and produces bone pain. Visible swelling of involved lymph nodes has been observed after ingestion of alcohol in patients with alcohol-related pain and, in at least one instance, thermography recorded a rise in lymph node temperature.

Although alcohol-related pain is uncommon, this report is a reminder that this symptom may precede any other recognizable feature of Hodgkin's disease and consequently may be helpful in the early diagnosis of this potentially curable disease.

REFERENCES

1. Kaplan HS: Hodgkin's Disease. Cambridge, Harvard University Press, 1980, pp 120-121
2. Hoster HA: Hodgkin's disease. AJR 1950; 64:913-918
3. James AH: Hodgkin's disease with and without alcohol-induced pain. Q J Med 1960; 29:47-66
4. Brewin TB: Alcohol intolerance in neoplastic disease. Br Med J 1966; 2:437-441
5. Atkinson K, Austin DE, McElwain TJ, et al: Alcohol pain in Hodgkin's disease. Cancer 1976 Feb; 37:895-899
6. Bichel J: Is the alcohol-intolerance syndrome in Hodgkin's disease disappearing? Lancet 1972 May 13; 1:1069

Secondary Syphilis With Pulmonary Involvement

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REPORTS OF CASES of primary and secondary syphilis have been increasing in number in the United States for the past several years,[1] and because of this physicians are rediscovering the protean manifestations of this disease. Pulmonary involvement in acquired secondary syphilis is uncommon.[2] We report the case of a 39-year-old man who had secondary syphilis and bilateral infiltrates on a chest roentgenogram. Following penicillin therapy for syphilis, the pulmonary abnormalities cleared over several months. Clinical criteria for the diagnosis of acquired secondary syphilis of the lung are suggested.

Report of a Case

A 39-year-old homosexual man was seen in August 1980 because of malaise for the past six months and a 5.9-kg (13-lb) weight loss in the previous month. He noted no fever or chills but had frequent night sweats. Four months previously, numerous nonpruritic skin lesions had developed in the groin and spread in a migratory fashion to the abdomen, chest, back, arms, legs and scalp. He smoked two packs of cigarettes per day. He said he did not abuse drugs intravenously or otherwise. He lived with 24 cats in San Francisco and had traveled recently to the Midwest. He had had multiple anonymous sexual contacts during the preceding months. He was currently unemployed but had worked in a grocery store. On review of systems there was a chronic nonproductive cough of two to three years' duration.

It was learned from the Public Health Department that the patient had been treated previously for syphilis. In 1972 his VDRL titer was positive at a 1:4 dilution and he received intramuscularly two injections of 2.4 million units of benzathine penicillin G at an interval of a week. Subsequent serologic testing, shown in Table 1, showed a VDRL titer in 1978 that was positive at a 1:2 dilution with a strongly positive result on fluorescent treponemal antibody-absorption test. In October 1979 his VDRL was positive in a titer of 1:1,024 and he was treated as in 1972. A VDRL titer in June 1980 was 1:128. In early August 1980 it rose to 1:256 and several weeks later it had risen to 1:512.

On physical examination he appeared well developed, was in no acute distress and had normal vital signs. Multiple salmon-colored, indurated, annular 0.5- to

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