Letters

Optic Nerve Sarcoidosis

To the Editor: We read with interest the case report by Ing and colleagues in the January 1997 issue of the *Mayo Clinic Proceedings* (pages 38 to 43), and we would like to report a 19th case of optic nerve sarcoidosis without evidence of systemic disease.

A 42-year-old black woman came to our medical center for assessment of progressive visual loss (worse on the right side) of 7 months' duration. On examination, her visual acuity was 20/400 in the right eye, with intact pupillary reflexes. Magnetic resonance imaging revealed an enhanced infiltrative exophytic mass at the site of the optic chiasm and nerves (greater on the right than on the left). Visual evoked potentials were abnormal, with decreased amplitude and prolonged latency of the major peaks on the right. Results of cerebrospinal fluid studies were normal, as were routine blood chemistries, complete blood cell count, and thyroid-stimulating hormone, prolactin, and cortisol levels. The angiotensin-converting enzyme level was mildly increased at 88 U/L (normal, 6.0 to 64.0). A preoperative chest roentgenogram showed normal findings. A biopsy of the optic chiasm revealed a granulomatous neuritis of the optic nerve. All special stains were negative for organisms. Prednisone therapy (60 mg/day) was instituted, and her vision improved considerably.

We believe that our patient represents the 19th reported case of optic nerve sarcoidosis without systemic involvement. The clinical manifestations, biopsy findings, and response to corticosteroid therapy support this diagnosis.

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Liver Failure Due to Metastatic Small-Cell Carcinoma of the Lung

To the Editor: In their case report entitled "Small-Cell Carcinoma of the Lung Manifesting as Acute Hepatic Failure," which was published in the February 1997 issue of the *Mayo Clinic Proceedings* (pages 133 to 139), McGuire and colleagues postulated that increased uric acid levels and a substantially greater ratio of normalized serum lactate dehydrogenase to normalized serum alanine aminotransferase can facilitate the early diagnosis of small-cell carcinoma of the lung manifesting as acute hepatic failure. Although these findings raise the suspicion of lung cancer metastatic to the liver, the absence of such findings does not exclude the diagnosis. In one of my patients, a 46-year-old woman who died 9 days after hospitalization for fulminant liver failure, autopsy revealed metastatic small-cell carcinoma of the lung with diffuse infiltration of the liver, which weighed 5,200 g. At the time of her admission to the hospital, the uric acid level was normal (6.6 mg/dL; normal, 2.4 to 7.0). Additional measurements within 2 days before her death revealed increased levels—10.9 and 12.3 mg/dL. The ratio of normalized serum lactate dehydrogenase to normalized serum alanine aminotransferase remained below 1 throughout hospitalization (range, 0.56 to 0.96). The lactate dehydrogenase values ranged from 2,766 to 6,410 U/L (upper limit of normal, 240), and the alanine aminotransferase values were 879 to 1,410 U/L (upper limit of normal, 51 U/L).

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In response: We thank Dr. Galus for providing an additional case of small-cell carcinoma of the lung metastatic to the liver confirmed at autopsy. Among patients with small-cell carcinoma of the lung, 28% have liver involvement.[1] Serum lactate dehydrogenase levels, used to predict the prognosis of patients with small-cell carcinoma of the lung, correlate with the extent of the disease.[2] Lactate dehydrogenase is not specific to the liver, however, and values can be increased in patients with tumor and no liver involvement. In addition, serum alanine aminotransferase is more liver specific than lactate dehydrogenase, and levels can be substantially increased in patients with viral hepatitis, toxin-induced injury, ischemic hepatitis, autoimmune disease, and acute biliary obstruction. The considerably increased serum lactate dehydrogenase level in Dr. Galus' patient is comparable to those reported in our article. The serum alanine aminotransferase value in her patient was dramatically high in comparison with those in our patients. Because increased serum levels of alanine aminotransferase may be caused by other processes involving the liver, such as toxin-induced injury, hypotensive ischemic hepatitis, or acute biliary obstruction due to adenopathy of the porta hepatis, we agree that the absence of a high ratio of normalized lactate dehydrogenase to normalized alanine aminotransferase does not exclude metastatic small-cell carcinoma as a cause of liver failure. Without additional clinical information, we cannot speculate further on the lower than expected ratio of normalized lactate dehydrogenase to normalized alanine aminotransferase.

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REFERENCES

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