for the third person, but her anemia was microcytic and hypochromic. All three persons responded to the withdrawing of zinc or to giving copper.

The physiologic requirement for zinc is 10 to 15 mg a day. Yet the suggested pharmacologic dose of zinc is ten times this amount, a dose at which toxicity has been reported. Food faddists suggest doses from 50 mg for "basic protection" to 200 mg for infertility and osteoarthritis.[10] We recommend avoiding these huge doses in order to prevent excessive intake and its accompanying toxicity. To allow for needed dietary supplementation, we recommend that over-the-counter preparations of zinc be limited to 5-mg capsules or tablets. In using zinc as a specific medication, physicians should be extremely cautious when a dose of 150 mg a day or greater is prescribed. As noted, at this "therapeutic" dose, toxicity to the bone marrow has been described. Finally, a health care team needs to carefully review a patient's history in regard to nutritional supplements. With this increased awareness, we expect more persons with self-induced vitamin or mineral intoxication will be identified.

REFERENCES

1. Bottomley SS: Sideroblastic anemias. Clin Hematol 1982; 11:389-409
2. Bremner I: Nutritional and physiological significance of metallothionein. Experientia [Suppl] 1987; 52:81-107
3. Mills CF: Trace element interaction: Effects of dietary composition on the development of imbalance and toxicity, *In* Hoekstra WG, Suttie JW, Ganther HE, et al (Eds): Trace Element Metabolism in Animals, vol 2. Baltimore, University Park Press, 1974, pp 79-90
4. Prasad AS, Brewer GJ, Schoomaker EB, et al: Hypocupremia induced by zinc therapy in adults. JAMA 1978; 240:2166-2168
5. Brewer GJ, Hill GM, Prasad AS, et al: Oral zinc therapy for Wilson's disease. Ann Intern Med 1983; 99:314-320
6. Cordano A, Baertl JM, Graham GG: Copper deficiency in infants. Pediatrics 1964; 34:324-336
7. Patterson WP, Winkelmann M, Perry MC: Zinc-induced copper deficiency: Megamineral sideroblastic anemia. Ann Intern Med 1985; 103:385-386
8. Simon SR, Branda RF, Tindle BF, et al: Copper deficiency and sideroblastic anemia associated with zinc ingestion. Am J Hematol 1988; 28:181-183
9. Porter KG, McMaster D, Elmes ME, et al: Anaemia and low serum-copper during zinc therapy (Letter). Lancet 1977; 2:774
10. Giller RM, Matthews K: Medical Makeover. New York, Warner Books, 1986, pp 191-192

Herpes Zoster Phrenic Neuritis With Respiratory Failure

CPT WILLIAM L. MELCHER, MC, USA
MAJ ROBERT A. DIETRICH, MC, USA
MAJ WARREN L. WHITLOCK, MC, USA
San Francisco

HERPES ZOSTER is a rare cause of unilateral diaphragmatic paralysis, which manifestation was first described in 1949.[1] Since then, 17 cases have been reported.[2-5] We present a case of hypoventilatory respiratory failure due to unilateral zoster phrenic neuritis complicated by obesity.

(Melcher WL, Dietrich RA, Whitlock WL: Herpes zoster phrenic neuritis with respiratory failure. West J Med 1990 Feb; 152:192-194)

From the Pulmonary Disease Service, Department of Medicine, Letterman Army Medical Center, Presidio of San Francisco.

Reprint requests to Medical Editing HSHH-CI-ME, Department of Clinical Investigation, Letterman Army Medical Center, Presidio of San Francisco, CA 94129-6700.

Report of a Case

The patient, a 79-year-old man, had dyspnea that began in May 1987. At that time he had an aortobifemoral bypass operation complicated postoperatively by a cutaneous herpes zoster infection of the right posterior cervical region. Over the next seven months he noted progressive dyspnea, orthopnea, lower extremity edema, and daytime hypersomnolence. He said he did not have fever, cough, or chest pain. His medical history included hypertension, a right carotid endarterectomy, and a 150-pack-year tobacco history. His medications included furosemide, captopril, topical nitrates, and aspirin.

On physical examination he was obese, appeared somnolent, and was in pronounced respiratory distress with shallow respirations at 40 breaths per minute. His blood pressure was 140/90 mm of mercury. There were confluent hypopigmented scars on the right occiput, posterior neck, and shoulder (Figure 1). There was no stridor or thyromegaly. Examination of his chest revealed decreased breath sounds and thoracoabdominal asynchrony; there were no murmurs or gallops. The lower extremities showed pitting edema. There was decreased strength of the right trapezius and deltoid muscles, but otherwise muscle strength was normal. A chest roentgenogram showed a newly elevated right hemidiaphragm.

An arterial blood gas measurement done with the patient breathing room air showed a pH of 7.12, a Pco_2 of 10.8 kPa (81 mm of mercury), a Po_2 of 2.9 kPa (22 mm of mercury), and an arterioalveolar oxygen difference of 3.6 kPa (27 mm of mercury). The patient underwent emergent orotracheal intubation. There was no evidence of airflow obstruction. On fluoroscopy he had paradoxic movement of the right hemidiaphragm with a "sniff" maneuver. Computed tomography of the chest showed no mediastinal or parenchymal abnormalities. With mechanical ventilation the hypersomnolence resolved, and the patient was successfully weaned from the ventilator. He continued to manifest supine hypoventilation that was managed with nocturnal cuirass ventilation. The forced vital capacity showed a 39% decline with changing from the standing to the supine position. Table 1 shows serial changes in spirometric measurements with changes in weight. On polysomnography he had no evidence of obstructive sleep apnea, and thyroid function studies showed normal values.

During 12 months of follow-up and with a 20% weight reduction, the patient was successfully weaned from nocturnal ventilation. He has maintained normal arterial blood gas measurements despite persistent diaphragmatic palsy documented by continued right diaphragmatic elevation on repeated chest roentgenograms.

Discussion

The diagnosis of zoster phrenic neuritis was based on the findings of cutaneous lesions in the cervical dermatomes corresponding to the right phrenic nerve roots and a newly elevated hemidiaphragm that moved paradoxically under provocative fluoroscopic examination. Skeletal muscle involvement in these dermatomes was also evident. There was no evidence of causes of diaphragmatic paralysis such as

TABLE 1.—*Pulmonary Function Data and Weight in a Patient With Phrenic Neuritis*

Pulmonary Function Test	May 1987	January 1988		February 1989
		Standing	Supine	
Forced vital capacity (FVC), liters*	2.76 (71)	1.93 (50)	1.38 (36)	2.31 (60)
FEV_1, liters*	2.02 (81)	1.44 (50)	1.00 (40)	1.68 (68)
FEV_1/FVC, %	73	86	...	73
Weight, kg†	96.0	92.7	...	77.7

FEV_1 = forced expiratory volume in 1 second.

*Numbers in parentheses represent the percent (%) predicted capacity.
†Ideal body weight 69.0 kg.

mediastinal tumor, trauma, cold cardioplegia, poliomyelitis, the Guillain-Barré syndrome (acute febrile polyneuritis), Parsonage-Turner's syndrome, amyotrophic lateral sclerosis, or aortic aneurysm. Phrenic nerve conduction testing was not immediately available in our case but would have supplied additional evidence for zoster phrenic neuritis.

Herpes zoster represents a myelitis involving the dorsal root ganglion, which may occasionally extend to the adjacent anterior horn cells, leading to motor involvement.[1] In 18 previously reported cases of zoster phrenic neuritis, diaphragmatic paralysis was discovered from several days to three years after the onset of zoster.[1-5] The paralysis resolved during the period of follow-up in only three cases, 1 to 18 months after zoster infection.[3-5] This clinical course parallels the known course of axial zoster neuritis for which no effective treatment has been shown.[6] A rare syndrome of brachial and phrenic neuritis of unknown cause has been reported as the Parsonage-Turner syndrome. In contrast to that of zoster phrenic neuritis, the prognosis for recovery is excellent.[7]

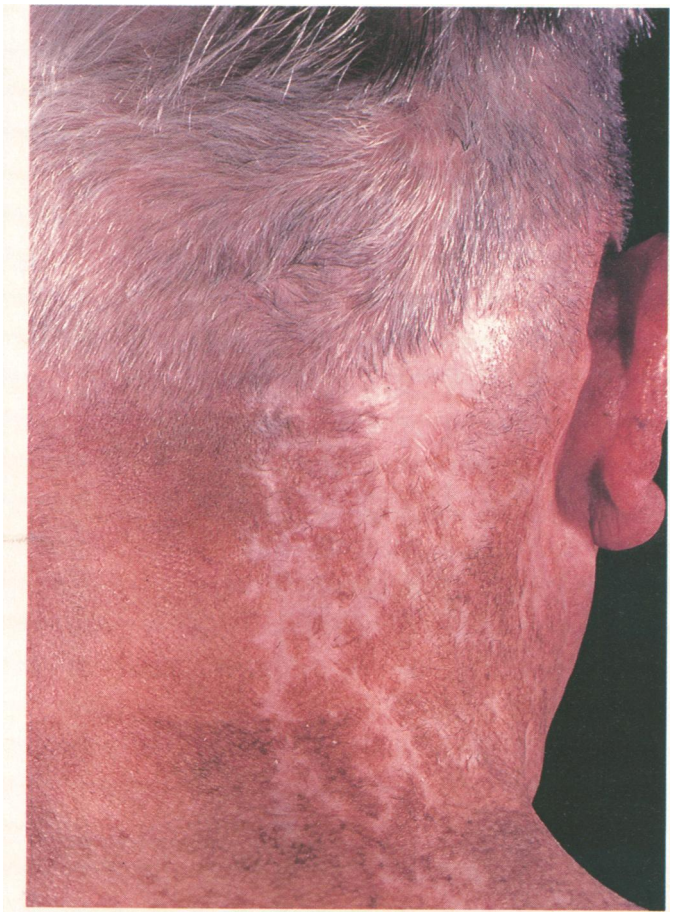

Figure 1.—Cutaneous lesions of healed herpes zoster are seen to involve the right second through fifth cervical dermatomes.

The causes of hypoventilatory respiratory failure can be divided into disorders of ventilatory drive, ventilation-perfusion, neuromuscular function, and thoracic compliance. The respiratory consequences of unilateral diaphragmatic paralysis include a substantial decrease in functional residual capacity and vital capacity; however, hypoventilation is not reported to be a feature of uncomplicated unilateral diaphragmatic palsy.[8] Similarly, the respiratory complications of obesity include a decreased functional residual capacity and decreased thoracic compliance resulting from excessive body mass.[9] As with diaphragmatic paralysis, these effects are most prominent in the supine position.

This patient experienced an excessive work of breathing because of his obesity. We postulate that the added mechanical liability of a paralyzed hemidiaphragm resulted in progressive alveolar hypoventilation. Successful management was accomplished with weight reduction to decrease the work of breathing and with interim management of positional hypoventilation with nocturnal cuirass ventilation. Negative-pressure ventilators and other forms of noninvasive mechanical ventilation have been used effectively to provide intermittent ventilatory support for patients with compromised respiratory muscle function as seen in cases of poliomyelitis and muscular dystrophy.[10]

This is the first report of unilateral zoster phrenic neuritis associated with respiratory failure complicated by obesity. The cutaneous stigmata of herpes zoster with a newly elevated hemidiaphragm was diagnostic of zoster phrenic neuritis.[3] Hypoventilation, manifested by a preserved arterio-alveolar oxygen difference, suggested a neuromuscular disorder as the cause of respiratory failure. The respiratory consequences of excessive work of breathing due to obesity and compromised neuromuscular function caused by zoster phrenic neuritis were cumulative. Therapy with short-term negative-pressure nocturnal ventilation combined with weight loss was effective.

REFERENCES

1. Halpern SL, Covner AH: Motor manifestations of herpes zoster. Arch Intern Med 1949; 84:907-916

2. Anderson JP: Paralysis in herpes zoster. Br Med J 1970; 3:587

3. Anderson JP, Keal EE: Cervical herpes zoster and diaphragmatic paralysis. Br J Dis Chest 1969; 63:222-226

4. Derveaux L, Lacquet LM: Hemidiaphragmatic paresis after herpes zoster. Thorax 1982; 37:870-871

5. Spiers ASD: Herpes zoster and its motor lesions, with a report of a case of phrenic nerve paralysis. Med J Aust 1963; 1:850-853

6. Taterka JH, O'Sullivan ME: The motor complications of herpes zoster. JAMA 1943; 122:737-741

7. Walsh NE, Dumitru D, Kalantri A, et al: Brachial neuritis involving the bilateral phrenic nerves. Arch Phys Med Rehabil 1987; 68:46-48

8. Easton PA, Fleetham JA, De La Rocha A, et al: Respiratory function after paralysis of the right hemidiaphragm. Am Rev Respir Dis 1983; 127:125-128

9. Luce JM: Respiratory complications of obesity. Chest 1980; 78:626-631

10. Garay SM, Turino GM, Goldring RN: Sustained reversal of chronic hypercapnia in patients with alveolar hypoventilation syndromes. Am J Med 1981; 70:269-274

Book Review

The Western Journal of Medicine *does not review all books sent by publishers, although information about new books received is printed elsewhere in the journal as space permits. Prices quoted are those given by the publishers.*

Preventing Emergency Medicine Malpractice

Raymond M. Fish, PhD, MD, Burnham City Hospital Trauma Center and University of Illinois, Urbana-Champaign, and Melvin E. Ehrhardt, MD, JD, Mennonite Hospital, Bloomington, Illinois. Medical Economics Books, Oradell, NJ 07649, 1989. 167 pages, $31.95.

Preventing Emergency Medicine Malpractice is a small volume full of case histories of emergency medicine malpractice and good common sense advice on how physicians practicing in emergency departments can avoid these situations. This book does a good job of classifying the potential problem areas that lead to emergency medicine malpractice and gives a reasonable overview of high-risk situations such as AMA (against medical advice) patients, psychiatric problems, abuse and assault cases, and the most common misdiagnoses that lead to malpractice claim judgments. The weakness of this book lies in its lack of strong, in-depth reviews of many of these identified problem areas. The discussions include many case reports, sometimes with legal references, but a clearly referenced explanation of the laws applicable to particular situations is often lacking. For example, in its section on "Patient Transfers and Dumping," the only reference to COBRA legislation is the sentence "COBRA legislation imposes severe penalties for some financially motivated transfers."

This book, however, is not meant to be an in-depth review of malpractice legal doctrine, and in terms of its purpose of highlighting problem areas and giving practical advice, such as the recurring emphasis on complete and specific documentation of the medical record, it has much to offer. The chapter on "Emergency Consent and Refusal of Treatment" is an excellent review, particularly of how to deal with the patient who wants to leave against medical advice. Some specific suggestions are offered on what to document in the record regarding this situation. I also found the chapter on "Medical Records" to be a good discussion of one of the most essential areas of risk management—documenting the medical record. The chapter on "Handling Psychological Problems in the Emergency Department" was a helpful discussion of a stressful and problem-prone area of emergency medicine but was somewhat weak on giving specific details and advice on psychiatric commitment, two-physician holds, police holds, and the particular rights and duties of physicians in these situations. Again, because of variances from state to state, an in-depth discussion is probably beyond the scope of a book this size.

The chapters on "Lawsuit Danger Signs" and "Common Lawsuits in Emergency Medicine" again are reasonable overviews of some common risk management pitfalls and the medical conditions that lead most frequently to emergency medicine lawsuits, such as myocardial infarctions, fractures, meningitis, wounds, and appendicitis. The discussion of these specific syndromes is filled with anecdotal case reports and common sense advice for how to avoid trouble with these diagnoses, but for the physician looking for good data, useful information from clinical trials, and strong, clinically oriented, in-depth discussions of these particular areas of medicine, this volume does not go into enough detail or give adequate references on these issues.

ROBERT EICHNER, MD
Director of Quality Assurance
Emergency Department
Emanuel Hospital and Health Center
Portland