there are different LZM isoenzymes in man with different lytic activities in the two assay systems. These isoenzymes might be derived from different tissues, eg, leukocytes and cartilage, in patients with RA. Furthermore the presence of possible LZM inhibitors, (ie, circulating glycosaminoglycans) in RA may cause charge interactions with cationic LZM and subsequent interference, particularly in the lysosyme (diffusion) assay.

We emphasize that these methodologic considerations must be recognized when LZM determinations of pleural fluid are used in the diagnostic procedures to discriminate between tuberculous and rheumatoid pleural effusions.

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REFERENCES

Charcot-Marie-Tooth Disease and Respiratory Failure

To the Editor:

We would like to report our experience with a patient with Charcot-Marie-Tooth disease and respiratory failure. Our observations support those of Chan et al (Chest 1987; 91:567-70) that Charcot-Marie-Tooth disease can involve respiratory muscles. Unlike the two cases reported by Chan et al, however, this patient did not have diabetes mellitus, eliminating the possibility of diabetic phrenic neuropathy as a factor contributing to respiratory failure.

A 79-year-old woman presented to the local emergency room in April, 1985 with dyspnea, confusion, and lethargy. Arterial blood gases on FIO₂ (100 percent) taken shortly after tracheal intubation were pH 7.17, PCO₂ 110 mm Hg and PO₂ 324 mm Hg. Measured serum bicarbonate level was 40 mEq/L. She was transferred to our hospital four days later after several attempts to wean her from the ventilator were unsuccessful because of persistent hypoventilation. She had a 30-yr history of progressive bilateral foot and leg weakness, and had spent the last ten years in a wheelchair. In recent years she had become weak in the hands and arms. She had no previous history of pulmonary disease and had never smoked cigarettes. Examination revealed bilateral atrophic paralysis of gastrocnemius and peroneal muscles, and pes cavus deformities. Muscle atrophy and weakness were present in the intrinsic muscles of the hands. Both shoulders were weak, the trapezius muscles were ribbon-like, and the chest was moderately kyphoscoliotic. No deep tendon reflex was present. No fasciculations, cranial nerve abnormalities or peripheral nerve enlargement was present. Spontaneous tidal volume was 60 to 50 ml, vital capacity was 200 to 250 ml and minute ventilation was 1.5 to 2.0 L/min. Maximum negative inspiratory force could she generate varied from negative to 12 cm H₂O and did not improve with correction of mild hypokalemia and hypophosphatemia, the only identified serum chemical perturbations. Thyroid function studies, aldolase, magnesium, anti-nuclear antibody, rheumatoid factor, and creatinine phosphokinase levels were normal. An RPR test was non-reactive. Chest radiographic film showed reduced lung volume, subsegmental basal atelectasis and a calcified mitral annulus. Compensated mitral insufficiency was confirmed by physical examination and echocardiography. Cerebrospinal fluid was benign. A computed tomogram of the head showed only mild cerebral atrophy. Electromyographic and nerve conduction velocity studies of the lower extremities showed no motor response in the lower limbs and conduction velocities of 14 m/sec in the upper extremities. Her only son was examined and was found to have a significant peroneal atrophy, high arches and leg weakness. A cousin was examined and found to have absent ankle jerks, peroneal weakness, atrophy of the hands, and conduction velocities of 27 m/sec in the arms. Additional family history obtained revealed that the patient’s mother had reported hand atrophy. Two siblings of the patient had high arches, gait disturbance and atrophy of the hands. Over four generations of the family are affected in a pattern of autosomal dominant inheritance. She could not be weaned from the respirator because of persistent hypoventilation and lived two years at home with positive pressure ventilation therapy before dying of urosepsis and cerebrovascular accident.

We believe this woman had Charcot-Marie-Tooth disease because of the progressive atrophic paralysis beginning in the feet, nerve conduction velocity studies compatible with a chronic demyelinating neuropathy, absence of deep tendon reflexes, and findings in her family typical of this disease. We were not able to study this patient’s diaphragm function as Chen et al were able to do with their patients. We cannot prove that Charcot-Marie-Tooth disease directly involved any of her respiratory muscles. Kyphoscoliosis was no doubt a factor in this woman’s hypoventilation, but our judgment is that her respiratory failure far exceeded that which might be expected with her degree of scoliosis. We would like simply to draw further attention to a possible cause-effect relationship of Charcot-Marie-Tooth disease and respiratory failure so that more observation might be made.

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Long-term Tuberculosis Care

To the Editor:

In their recent clinical dialog, "Tuberculosis Long Term Care Beds: Have We Thrown Out the Baby With the Bath Water?" Drs. Yeager and Medinger point out that “most cities do not have the resources to achieve the high success rate in ambulatory chemother-