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Case#
D-576, contributed by Ellsworth C. Alvord, Jr., M. D.

This 59 yr. old farmer developed progressive weakness, weight loss, pains and cramps, with death 15 months after onset. He had been in good health until August 1956, when he awoke one morning with pain in the left hip radiating down the medial aspect of the left leg. The following week he developed numbness in the right knee, hand and face, and then abdominal pain, and constipation. Gradually numbness of both hands and dragging of the left foot developed. In December spinal fluid contained 140 mg.% protein, and chest x-ray was normal.

By February 1957 he had become more weak, unable to write or handle objects, and was hospitalized for 4 months. Examination revealed muscle strength and coordination slightly impaired in both hands. There was muscular atrophy in the quadriceps and slightly in both hands. The gait was slightly ataxic, and there was a postive Romberg. Touch, pain and position sensations were decreased bilaterally and distally, more in the arms than in than in the legs. Vibration sense was normal except in the left big toe. Biceps, triceps, and patellar reflexes were absent; the ankle jerks were normal. A left Babinski sign was recorded. X-rays revealed no significant lesion in the chest, gall bladder or gastrointestinal tract. X-rays of the spine revealed slight osteo-arthritis. Routine laboratory work was normal including total serum protein, A/G ratio, fasting blood sugar, VDRL, thymol turbidity, bilirubin, Schwartz-Watson, and LE preparations. Serum cholesterol varied from 222 to 381 mg.% on 7 occasions. The first examination of the urine for arsenic revealed 0.6 mg./L, but the next 4 examinations were all negative. Lumbar puncture on February 25 was normal except for protein 108 mg.%. Subsequent lumbar punctures revealed protein 113 mg.% on March 13 and 85 mg.% on April 26.

During his hospitalization he was treated with steroids, which afforded some improvement for a while, but then he began to complain more about paresthesias in the arm and face, and became negativistic, hypocondriacal and depressed and cried all the time. It was thought that he had a passive dependent personality with a neurotic reactive depressive reaction to his organic disease, and in September he received 10 electro-shock treatments for his psychoneurosis. His depression improved, but his weakness continued to become worse, so that he became bedfast. He was unable to swallow solids and occasionally choked on liquids and developed constant nausea. He had lost 90-100 lbs. over a 15 month period (normal weight 200 lbs). Diplopia and tremor gradually appeared. He was hospitalized for the last time on Nov. 2, 1957, when he was noted to be cachetic but alert and cooperative, moving frequently because of cramps. Neurological examination revealed marked muscular atrophy of all muscles including the shoulder girdle and small muscles of the hand. There was a coarse tremor of the hands with cogwheel rigidity. Extra-ocular movements were markedly weak with vertical nystagmus. Facial movements and sensation and hearing were normal, but he was unable to open his mouth widely, and there was slight weakness of the tongue and masseter muscles. The gag reflex was hyperactive. Bowel sounds were increased, and there was markedly increased anal sphincter tone. Sensory examination was variable and not reliable. Deep tendon reflexes were absent, but superficial abdominal reflexes were present. There was no Babinski sign.

Lumbar puncture on Nov. 5 revealed no cells and 67 mg% protein. EEG was slightly slow but within normal range. X-rays of the skull, hips, pelvis and abdomen were normal. X-rays of the chest on Nov. 4 revealed a 5 x 5 cm. tumor mass in the medial right base posteriorly. He became progressively worse and died on Dec. 10, 1957, about 15 months after the onset of symptoms.

Autopsy revealed bronchogenic carcinoma with metastases only to regional lymph nodes and left adrenal, and incidental carcinoma of thyroid and adenoma of parathyroid.