Original Investigations

Malignant Glioma and Sensorimotor Neuropathy

S. I. Mellgren*, S. Mørk, and H. Nyland
Department of Neurology and Gade's Institute, Department of Pathology, School of Medicine, University of Bergen, Norway

Summary. A case of sensorimotor neuropathy in a male with malignant glioma is reported. The symptoms of peripheral motor and sensory disturbances preceeded those of the intracranial tumor.

The history, clinical findings, electrophysiological and histopathological results are presented, as well as immunological data. A possible causal relationship between glioma and peripheral neuropathy is discussed.

Key words: Glioma, malignant – Neuropathy, sensorimotor – EMG – Histopathology – Immunology – Brain tumor – Malignancy and nervous system.

Introduction

An association between malignant neoplasia of extracranial origin and peripheral neuropathy is well known [1, 6]. The incidence of clinical peripheral neuropathy and malignant disease is greatest with carcinoma of the lung and stomach [6]. Electrophysiological [7] and histopathological [5] studies indicate that subclinical neuropathy may occur in up to 50% of cases. However, there is little information

* Present address for offprint requests: Department of Neurology, University of Trondheim, Regional Hospital, N-7000 Trondheim, Norway

0340-5354/78/0218/0001/$01.20
on the combination of glioma and peripheral pareses [1]. The present paper reports on a case of rapidly progressive sensorimotor neuropathy in a patient with a malignant glioma.

**Case Report and Methods**

The patient was a 55 year old married taxi driver. Before admission in August 1976, he had been drinking too much alcohol according to his wife. On the other hand, his job as a taxi driver obviously prevented him from drinking during working hours (cfr. very strict Norwegian law on alcohol and driving) and he had been eating normally. From September 1975 he experienced numbness and paraesthesias distally in the upper limbs, gradually accompanied by distal weakness and atrophy of muscles. Involuntary twitching (fasciculations) in the limb musculature had also been noted. The 2 months before admission he suffered from increasing morning headache. In addition, his memory failed, he felt drowsy, and sometimes while speaking he had experienced difficulty in finding the right words.

Neurological examination in August 1976 revealed a slight mental reduction and motor dysphasia. There were no clinical signs of increased intracranial pressure. A slight right central facial paresis and tongue deviation to the right was noted. There was general atrophy of the limb muscles, particularly in the hands and legs with correspondingly marked pareses. Fasciculations were observed in the shoulder girdle, arms and thighs. Glove and stocking hypalgesia and hypesthesia were also registered. The finger-nose test revealed slight ataxia on the right. Apart from a sluggish Achilles reflex, the deep tendon reflexes were normal and symmetrical. The right plantar reflex was extensor. General medical examination revealed no further signs of disease.

Routine laboratory tests (including liver enzymes) were normal, except for a slight initial hyperglycaemia with glucosuria. The EEG contained theta and delta waves in the left hemisphere, particularly in the anterior and middle temporal regions. X-ray of the cranium demonstrated a dislocation of the pineal body 5 mm to the right and cerebral angiography disclosed a left temporal expansive process with deep central extension. Other radiological examinations and liver scintigraphy did not reveal metastases. **Muscular biopsy** from the left tibialis anterior revealed scanty atrophic fibers.

In August 1976 partial resection of a tumor (histologically determined to be a glioblastoma) was performed and postoperative treatment with CCNU and radiotherapy were given. However, the patient's condition rapidly deteriorated with increasing peripheral pareses, and prior to the second admission in February 1977, he needed help with almost every activity of daily life. There was no clinical evidence of further tumor extension but there was increased atrophy of muscles and corresponding pareses. Tendon reflexes were diminished and the previously registered sensory neuropathy was confirmed. Routine laboratory tests were normal.

**EMG** was recorded in proximal and distal limb muscles and showed fibrillation as well as fasciculation potentials. **Motor conduction velocity** in the peroneal nerve was 40 m/s and in the ulnar nerve 30 m/s, thus slightly and moderately reduced, respectively. A new muscle biopsy from the right tibialis anterior showed a marked neurogenic atrophy (Fig. 1).

**Immunological Studies.** Sedimentation rate was 10 mm/h and serum-γ-globulin 15.6 mg%. Serum antibodies to peripheral nervous tissue were demonstrated in low titers by antiglobulin consumption test in which homogenized human sciatic nerve served as antigen. Peripheral blood leucocyte count was 6500/mm³ with 12% lymphocytes (980/mm³). Lymphocyte subpopulations were analyzed by identification of specific membrane markers. T lymphocytes were identified by their ability to form rosettes with unsensitized ox-erythrocytes. The percentage of T lymphocytes was 72 (normal range 55—75%) while the absolute number was 562/mm³ which is significantly reduced. The percentage of B lymphocytes was 31 (normal range 18—35%) and the absolute number was 242/mm³ (normal). The CSF contained increased total protein, 108 mg%, IgG was 6.2 mg% and agarose electrophoresis showed a normal pattern. The CSF lymphocyte count was normal, 4/mm³, but the percentage of T lymphocytes was increased, 90% as compared to a mean value of 71 ± 4.4% in a normal control group.