Amyloidoma can occur within the brain parenchyma. Periventricular amyloidomas developed in a man aged 69 years as gadolinium-enhancing lesions on magnetic resonance imaging. The lesions were composed of amyloid AL λ with congophilia resistant to potassium permanganate. There was no evidence of systemic amyloidosis or an underlying inflammatory or neoplastic disorder.

Arch Neurol. 2005;62:477-480

**REPORT OF A CASE**

A man aged 69 years noted an occasional tendency of leaning toward the right side without experiencing falls during a 1-year period. His memory had been poor for approximately 20 years. His mother (aged 94 years) had senile dementia. At the initial evaluation, results of the examination were unremarkable save for difficulties in short-term memory. The T1-weighted magnetic resonance image (MRI) with gadolinium enhancement showed 5 lesions in the tegmentum of the upper right pons, periaqueductal gray matter, and right and left subinsular region (extreme capsule) and adjacent to the midbody of the left lateral ventricle (subependymal; Figure 1). The lesions were hyperintense on T1-weighted MRI and showed gadolinium enhancement. Findings on computed tomographic scans of the abdomen, pelvis, chest, and bone were normal. Two brain biopsies were performed. Results of the first were nondiagnostic. After the first biopsy, the patient's imbalance worsened; he was fatigued and his speech became slurred. Results of the neurological examination were remarkable for mild dysarthria. Strength and sensation, including proprioception, were normal. No dysmetria or dysdiadochokinesis was detected. His gait was wide-based, and he had slight difficulty initiating locomotion. Results of serum protein electrophoresis were normal. The cerebrospinal fluid was acellular, with levels of glucose, protein, IgG, and albumin within reference ranges, and no banding on agarose gel electrophoresis. No bacterial or fungal growth, cryptococcal antigen, or positive VDRL test results were found. Total-body positron emission tomography using fludeoxyglucose F 18 showed unremarkable findings.
During the course of 3 months, the pontine lesion grew. The second biopsy was performed 4 months after the first. The second biopsy specimen was investigated using standard protocols (hematoxylin-eosin and Congo red staining), polarized light examination, and immunohistochemistry (prealbumin and κ and λ light chains). For immunoglobulin heavy chain (IgH) gene rearrangement analysis, DNA was isolated from paraffin-embedded tissue using the Qiagen DNA Mini Kit (Qiagen, Valencia, Calif) according to the manufacturer’s protocol. Consensus primers for the complementarity-determining region III of the IgH gene were used for polymerase chain reaction analysis. The polymerase chain reaction products were separated on a 12% polyacrylamide gel.

Numerous deposits of acellular amorphous material that bound Congo red stains and showed apple green birefringence under polarized light were found within the neurophil (Figure 2). The material was immunoreactive for the κ light chain (Figure 3), with negative findings for prealbumin. Results of the IgH gene rearrangement analysis did not show any evidence of a clonal cell population of the B-lymphocyte lineage.

Investigation failed to reveal systemic amyloid. Results of urine analysis for Bence Jones protein and biopsy of abdominal fat were negative. Computed tomography of the chest and abdomen, radionucleide scan of bone, echocardiography, and nerve conduction studies had normal findings. The electrocardiogram showed no conduction anomaly.

**COMMENT**

We herein report the case of a patient with primary amyloidomas of the brain parenchyma and a clinical syndrome characterized by cognitive decline, cerebellar dys-
Results of immunohistochemical staining for β-amyloid protein and prealbumin were negative in our case. Evidence of local or systemic light chain disease was absent. Our patient underwent repetitive analysis of peripheral blood for monoclonal populations and of bone marrow for clonal expansion.

Accepted for Publication: March 19, 2004.

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Author Contributions: Study concept and design: Tabatabai, Baehring, and Hochberg. Acquisition of data: Tabatabai and Baehring. Analysis and interpretation of data: Tabatabai and Baehring. Drafting of the manuscript: Tabatabai and Baehring. Critical revision of the manuscript for important intellectual content: Hochberg. Administrative, technical, and material support: Tabatabai, Baehring, and Hochberg. Study supervision: Baehring and Hochberg.

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