INTRACAVERNOUS TRIGEMINAL GANGLION AMYLOIDOMA: CASE REPORT

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OBJECTIVE: Isolated amyloidomas rarely manifest in nervous system tissues. To the authors' knowledge, there have been 52 documented cases of primary amyloid tumors of the central nervous system and closely associated structures. The authors present a case of a woman with a history of presumptive trigeminal neuralgia who was found to have an amyloidoma of the trigeminal ganglion.

CLINICAL PRESENTATION: A 32-year-old Caucasian patient presented with a chief complaint of severe numbness and pain throughout the right side of her face. Her symptoms had been progressive over the previous 3 years. Medical management of her presumptive diseases with Zoloft (Pfizer Inc., New York, NY) and Neurontin (Pfizer Inc.) failed to improve or halt her right facial numbness and pain. Brain magnetic resonance imaging was acquired, demonstrating abnormal contrast enhancement and enlargement of the right trigeminal ganglion. The lesion abutted and indented the right internal carotid artery and extended from Meckel's cave into the inferior cavernous sinus and distally to the foramen ovale.

INTERVENTION: The patient underwent a right frontotemporal craniotomy for resection of the gasserian ganglion lesion. A delicate incision was made in the wall of the cavernous sinus, allowing confirmatory biopsy of the lesion. With the site of the tumor within the cavernous sinus verified by pathology, the remainder of the tumor was removed. A final pathological review of the resected tumor confirmed a diagnosis of amyloidoma of the trigeminal ganglion.

CONCLUSION: We present the case of a patient with a rare trigeminal ganglion amyloidoma that closely mimicked idiopathic trigeminal neuralgia. Even in the absence of systemic signs of amyloidosis, this benign protein deposition disease should be considered in the differential for atypical dysesthesias of the trigeminal dermatomes. Furthermore, central and peripheral nervous system amyloidomas respond well to surgical resection and rarely recur.

KEY WORDS: Amyloidoma, Intracavernous, Trigeminal ganglion

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Nervous system amyloidomas are rare tumors (13). To the authors' knowledge, there have been 52 documented cases of primary amyloid tumors of the central nervous system (CNS) and closely associated structures; 23 involved the cerebral parenchyma (4, 7, 8, 14, 17, 19, 23, 27, 28, 30, 33, 37, 46, 47,50, 52, 53), eight involved the spinal cord or spinal column (3, 13, 24, 29, 32, 35, 40, 41, 44), three involved the pituitary gland (2, 26, 38), four involved the cranial base (9, 18, 27, 54), and 13 involved the gasserian ganglion (5, 6, 11, 12, 27, 34, 39, 56, 57). We report a case of primary trigeminal nerve ganglion amyloidoma in a 32-year-old woman who presented with trigeminal neuralgia.

Patient Presentation

A 32-year-old Caucasian patient presented with severe numbness and pain throughout the right side of her face. Her symptoms had been progressive over the previous 3 years; she had visited multiple physicians during this time period. After extensive examination and tests, including lumbar puncture and magnetic resonance imaging (MRI) scans, had failed to expose any abnormalities, she was diagnosed with fibromyalgia and trigeminal neuralgia.

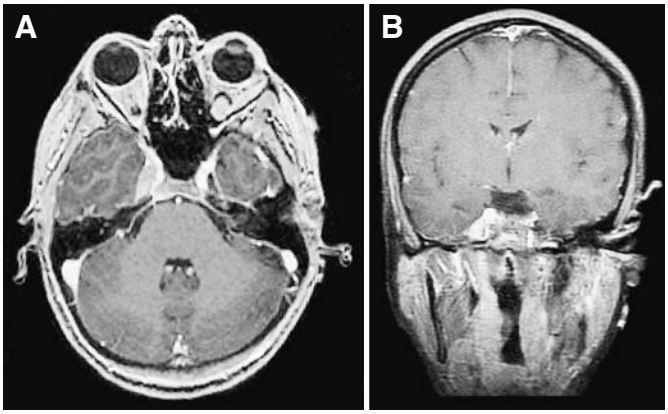

FIGURE 1. *Axial (*A*) and coronal (*B*) postcontrast T1-weighted MRI scans demonstrating the enhancing lesion in the right cavernous sinus.*

However, medical management of her presumptive diseases with Zoloft (Pfizer Inc., New York, NY) and Neurontin (Pfizer Inc.) failed to improve or halt her right facial numbness and pain. Her past medical history was otherwise unremarkable.

At the time of her initial presentation, the patient reported complete loss of sensation over the right side of her face, with periods of severe, diffuse right-sided facial tingling not associated with any recognizable triggers. The patient also noted having persistent headaches. No further symptoms were reported.

Examination

On examination, the patient had perturbed appreciation of light touch throughout all three trigeminal distributions on the right side. Cranial Nerves II through XII, with the above exception, were normal. She displayed no motor weakness, cerebellar tests were normal, and there was no evidence of gross cognitive deficits. Cardiopulmonary examination was normal, and there was no evidence of macroglossia or hepatosplenomegaly.

Imaging

Brain MRI scans were acquired, despite past reports of normal brain imaging. These scans showed abnormal contrast enhancement and enlargement of the right trigeminal ganglion (*Fig. 1*). The lesion abutted and indented the right internal carotid artery and extended from Meckel's cave into the inferior cavernous sinus and distally to the foramen ovale. Given the new lesion, the patient's previous MRI scans were also reviewed. Careful examination identified the same trigeminal ganglion lesion on multiple previous images. No abnormalities were identified in the contralateral ganglion.

Operation

The patient underwent a right frontotemporal craniotomy for resection of the gasserian ganglion lesion. After the bone flap was elevated, the dura was incised in a curvilinear fashion and the frontal and temporal lobes were gently elevated with Leila self-retaining retractors. The Sylvian fissure was then split using microdissectors, allowing an ample view of the right cavernous sinus, which was somewhat plump. A BrainLab frameless stereotactic navigation system (Heimstetten, Germany) was used to localize the lesion within the cavernous sinus. A delicate incision was made in the wall of the cavernous sinus at the site indicated by the navigation system. This provided a confirmatory biopsy of the lesion. With the site of the tumor within the cavernous sinus verified by pathology, the remainder of the tumor was removed using Yaşargil and Rhoton tumor forceps. The portion of the sinus containing the tumor was relatively avascular, allowing minimal blood loss during resection.

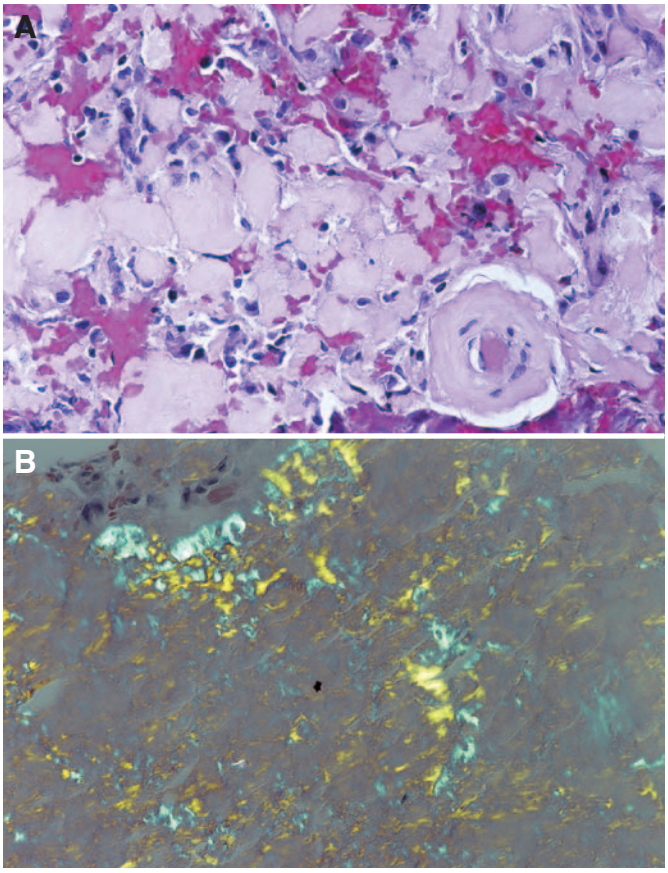

FIGURE 2. A, *hematoxylin and eosin stain of trigeminal ganglion amyloidoma with distinctive amorphous, eosinophilic, hyalinaceous material and minimal inflammatory reaction.* B, *polarized light microscopy image of Congo red-stained tumor. Note the* green *birefringence of the amyloid protein.*

Pathological Findings

A final pathological review of the resected tumor confirmed a diagnosis of amyloidoma of the trigeminal ganglion. The tissue samples consisted of amorphous material with little intervening inflammatory tissue (*Fig. 2*). Samples of the amorphous material stained positively with Congo red and displayed apple-green birefringence under polarized lighting, consistent with amyloid protein. Immunostaining for κ and λ light chains proved the amyloid to consist predominantly of λ light chains.

Postoperative Course

Given the unusual nature of the patient's diagnosis and her otherwise benign medical history, a workup for systemic causes of amyloidosis began. However, thorough blood work and urine testing for elevated serum protein failed to demonstrate a systemic source of amyloid production. Serum protein electrophoresis, serum free light chain assays, quantitative immunoglobulins, and 24-hour urine protein tests showed no evidence of M-spikes, altered κ-to-λ ratios, elevated immunoglobulin, or proteinuria. A fat pad biopsy with Congo red staining showed no amyloid deposits.

The patient was discharged in good health 4 days after her operation. She had a slight right Cranial Nerve III palsy immediately after the operation, which steadily improved over the subsequent weeks. At the time of her 2-month follow-up examination, the patient reported no pain or numbness across all trigeminal divisions.

DISCUSSION

Amyloid is an amorphous and heterogenous protein deposit associated with a variety of inflammatory and noninflammatory conditions, such as plasmacytomas, chronic renal failure, rheumatoid arthritis, and familial transthyretin mutations (25, 31, 40, 45, 49, 55). In rare instances, amyloidomas, consisting of β-pleated proteins with variable amounts of N-terminal digestion, may arise in the absence of any obvious sources of exuberant protein production (14, 30, 51). Instances of localized amyloid formation associated with systemic plasma cell dyscrasias occur in the context of amyloid light-chain amyloidoses, which may produce systemic or local disease, usually involving the kidney (48). A fluorescent in situ hybridization study of patients with systemic amyloid light-chain amyloidosis has demonstrated frequent chromosomal abnormalities that enhance plasma cell light-chain production. However, patients with localized amyloid tumors in the same study did not show similar chromosomal abnormalities (20). Most commonly, these isolated amyloid tumors arise in the respiratory tracts of afflicted patients; however, they may also be found in the skin and genitourinary tract (22).

In contrast, nervous tissues, both peripheral and central, are rare locations for amyloidoma formation. Laeng et al. (27) have theorized that amyloidomas may arise by way of monotypic λ-producing B-cells, perhaps in reaction to unique antigens. Cohen et al. (8) have put forth a similar theory, suggesting that genetically predisposed microglia may be sources of amyloid when challenged by certain antigens. Such mechanisms might explain why CNS amyloidomas are so rare, as the CNS is an immunologically protected site and would only be exposed to triggering antigens in rare instances (27).

However these amyloid tumors form, they are distinct from the much more common amyloid cores found in Alzheimer's disease and some elderly patients. Unlike the conjectured mechanisms of protein production in amyloidomas, amyloid cores found in the neuritic plaques of patients with Alzheimer's disease derive from a specific chain of processing events involving the amyloid precursor protein (15).

Pathological Characteristics

Although the appearance of amyloid-infiltrated tissues is not consistent on gross inspection, some generalizations can be made. Affected tissues will often appear bulging and waxy on gross inspection, and palpation sometimes finds these tumors gritty with imbedded osseous pockets (1, 54).

A definitive diagnosis must be made by microscopic examination and Congo red staining. Serological tests for elevated serum proteins can hint at the diagnosis of systemic amyloidosis. However, elevated light chain serum proteins are rarely detected in cases of amyloid tumors, and their absence cannot rule out the presence of an amyloid pathology (22). A biopsy sample of suspect tumors or of adipose tissue provides the best opportunity for diagnosis. The classic microscopic appearance of amyloid impregnated tissues stained with hemotoxylin and eosin is an amorphous, eosinphilic, extracellular hyaline substance. Occasionally, inflammatory infiltrates may be seen amid amyloid pockets but this is an unreliable finding (1). Foci of calcification have also been reported in several instances of intracranial amyloidomas, likely forming when calcium, released from bone eroded by these osteolytic protein deposits, binds to fibrils in the amyloidoma (11, 13, 27, 34, 54). The most striking microscopic feature of amyloidomas, however, is the apple-green birefringence that amyloid's β-pleated proteins give off under polarized light and Congo red staining. This appearance is consistent for all forms of amyloid (1). Immunohistochemical studies for λ and κ light chains in nervous tissue amyloidomas have nearly universally been positive for λ light chains, including the case reported here (4, 6, 8, 17, 27, 30, 34, 47, 50, 52, 57).

Imaging

Amyloid deposits on brain imaging can be quite variable in their appearance, depending on the density of protein deposition and the presence or absence of edema (27). Most commonly, these lesions involve the cerebral parenchyma, particularly the periependymal white matter of the lateral ventricles (4, 7, 14, 17, 27, 28, 52). On computed tomographic scans, amyloidomas appear hyperintense and readily enhance with contrast medium. Depending on their protein concentration, amyloidomas can appear hyper- to hypointense on T1-weighted MRI scans, whereas T2-weighted images tend to demonstrate intermediate intensities owing to varied protein content (17, 28). Gasserian ganglion amyloidomas, in particular, appear as brightly enhancing and swollen segments of Cranial Nerve V within Meckel's cave (27, 34, 39).

Presentation

Reported cases of amyloidomas of the brain and cranial nerves have generally occurred in middle-aged patients (mean age, 48.4 yr), with a slight bias toward women (male-to-female

ratio, 0.85). They most often occur as single lesions, but as many as one-third of the patients in the literature had multiple amyloidomas (2, 4–9, 11, 12, 14, 16–19, 26–28, 20, 33, 34, 37, 39, 46, 47, 50, 52–54, 56, 57). Most intracranial amyloidomas occur in the white matter of the brain (22 out of 40 cases) (2, 4–9, 11, 12, 14, 16–19, 26–28, 30, 33, 37, 39, 46, 47, 50, 52–54). When peripheral nerves are involved, the gasserian ganglion is almost invariably the culprit site (13 out of 17 cases) (5, 6, 10 to 12, 21, 27, 34, 36, 39, 42, 43, 56, 57). The reason for this predilection for trigeminal nerve involvement has not been clarified; however, it is suspected that latent infections of the ganglion might provide cryptogenic antigens that stimulate amyloid-forming B-cells.

Although it is sparse, the literature on gasserian ganglion amyloidomas is consistent in describing the common symptoms associated with these tumors. In all cases, the patients had primary complaints of pain and numbness in the distribution of one or more trigeminal branches and were often presumed to be experiencing classic trigeminal neuralgia. The diagnosis of this rare intracranial lesion is often delayed, with a mean duration of symptoms of 6.3 years, highlighting the unusual and deceptive nature of these tumors (5 to 7, 11, 12, 27, 34, 39). Among other intracranial amyloidomas, the most common symptoms are seizures and cognitive decline (2, 4, 7–9, 14, 16–19, 26–28, 30, 37, 46, 47, 50, 52–54).

Prognosis and Treatment

Because diagnosis of a trigeminal ganglion amyloidoma is often illusive, short of histological examination, their symptoms are typically present for a number of years before definitive diagnosis. Even so, surgical intervention has been shown to be effective in alleviating patient symptoms. Despite 3 years of progressive pain and numbness, the patient reported in this case report quickly regained normal facial sensation after surgical resection of her amyloidoma. In total, three out of 11 cases of gasserian ganglion amyloidomas achieved total resolution of symptoms within a few months. The remaining eight patients reported elimination of their pain but not their facial numbness (5, 6, 11, 12, 27, 39, 56).

Overall, cerebral and trigeminal nerve amyloidomas respond well to surgical resection, including cases with multiple foci. In the literature, 24% of patients with intracranial nervous tissue amyloidomas experience total symptomatic recovery after resection, whereas another 71% report only partial relief from surgical excision of their amyloidomas (4, 5, 6, 8, 11, 12, 14, 16, 26, 27, 28, 39, 47, 50, 53). There are three documented cases of death in patients with cerebral amyloidomas. Two patients died of seemingly unrelated cardiopulmonary events (2, 27). Another patient died 15 years after surgery owing to a massive cerebral hemorrhage at the site of a recurrent amyloidoma of the occipital lobe. This was the only instance in the literature of a CNS or PNS amyloidoma recurring (14).

While surgery is a frequent and successful treatment option for peripheral and CNS amyloidomas, non-invasive treatments have occasionally been used with little effect. Reports of the use of focused radiotherapy, steroids, or colchicine have not detailed any positive results (2, 27, 39). In the case of the patient treated with 4500 rads of local radiation, the patient died after his radiation treatments (2).

CONCLUSION

We present the case of a patient with a rare trigeminal ganglion amyloidoma that closely mimicked idiopathic trigeminal neuralgia. Even in the absence of systemic signs of amyloidosis, this benign protein deposition disease should be considered in the differential for atypical dysesthesias of the trigeminal dermatomes. Furthermore, peripheral and CNS amyloidomas respond well to surgical resection and rarely recur.

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COMMENTS

This is a nicely written, concise case report and review of intracranial amyloidomas. These are very rare tumors that can be missed easily, particularly in the trigeminal ganglion, because it is so common to assume enhancing cavernous sinus lesions are either meningiomas or schwannomas. They are, therefore, treated with stereotactic radiosurgery.

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This report of an amyloidoma of the gasserian ganglion and cavernous sinus is instructive in warning against irradiation of cavernous sinus lesions without first obtaining histological confirmation of presumed tumors such as meningioma or schwannoma. Stereotactic radiosurgery at tumor doses to this mass would likely not have controlled the patient's pain and certainly would not have restored sensation. The authors should be congratulated for their successful management of this case and for a highly informative review of the literature and discussion.

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