



Get Clarity On Generics

Cost-Effective CT & MRI Contrast Agents



FRESENIUS
KABI

WATCH VIDEO

AJNR

Intracerebral schwannoma: CT and MR findings.

C Di Biasi, G Trasimeni, M Iannilli, E Poletti and G F Gualdi

AJNR Am J Neuroradiol 1994, 15 (10) 1956-1958

<http://www.ajnr.org/content/15/10/1956>

This information is current as
of August 22, 2025.

Intracerebral Schwannoma: CT and MR Findings

Claudio Di Biasi, Guido Trasimeni, Massimo Iannilli, Elisabetta Polettini, and GianFranco Gualdi

Summary: A case of intracerebral schwannoma in a 19-year-old man is reported. CT and MR findings were nonspecific, suggesting a primary glioma. The histologic features were of schwannoma.

Index terms: Neuroma; Brain, computed tomography; Brain, magnetic resonance; Brain, neoplasms

Intracranial schwannomas account for 8% of all primary brain tumors, usually occur during the first three decades of life, and are about twice as common in male as in female patients (1), although some authors refer to a prevalence in middle-age women (2). The most common site of origin for schwannomas is the vestibular portion of the eighth cranial nerve; in the spinal canal they have a marked predilection for sensory nerve roots (3).

Although uncommon, these tumors have been reported to occur on each of the cranial nerves (with the exception of the optic nerve) and in the sympathetic chain and adrenal medulla (4, 5). Parenchymal schwannomas of the brain and spinal cord are rare lesions.

In 1966 Gibson et al published what seems to be the first recorded case of an intracerebral Schwann cell tumor (6). This developed in the temporal lobe of a 6-year-old boy. The present report records a prerolandic intracerebral Schwann cell tumor occurring in a 19-year-old man.

Case Report

The patient was a 19-year-old man who had had two focal motor seizures in the last year; the focal seizures took the form of clonic contractions of the left side of the face and the left arm, without secondary generalization. No family history of von Recklinghausen disease or any other neurologic disease was found in this patient.

The findings at neurologic examination were normal. The patient did not show pigmentary changes of the skin or any other sign or symptom of neurofibromatosis.

Magnetic resonance (MR) imaging of the brain showed a round, intraparenchymal mass, located in the right precentral region, which appeared of low signal intensity relative to gray matter on the T1-weighted precontrast images (Fig 1A), isointense on the proton-density images, and hyperintense on the T2-weighted images (Fig 1B). On postcontrast scans the lesion showed a marked homogeneous enhancement (Fig 1C and D). Computed tomography (CT) showed a round slightly hypodense lesion (Fig 2), with uniform enhancement after administration of contrast medium. Cerebral angiography demonstrated a slight venous blush.

The clinical presentation and CT and MR findings suggested the diagnosis of a prerolandic, intracerebral glioma. At surgery a well-encapsulated intraaxial mass was found. It was smooth and slightly yellow in color, and the adjacent parenchyma did not seem to be infiltrated by the tumor. The tumor was completely removed microscopically.

Histologic examination revealed that the tumor was composed predominantly of interwoven bundles of oval or spindle cells, containing elongated nuclei and eosinophilic cytoplasm. In many areas of the tumor mass, the stroma was hyalinized and surrounded by tumor cells, and the nuclei were aligned in a palisade arrangement. The blood vessels showed thickening and hyalinization of their walls.

The histologic features demonstrated the characteristics of schwannoma. The follow-up over the next year indicated no symptoms or neurologic deficits.

Discussion

We found no stigmata of neurofibromatosis or family history of this disease in our patient. Previous literature reports also did not show a significant association between von Recklinghausen disease and intraparenchymal neurofibroma both in spinal cord and cerebral locations. Bruni et al described a case of schwannoma of the frontal lobe in a 39-year-old man affected by von Recklinghausen disease (7).

Received November 24, 1992; accepted after revision March 8, 1993.

From the MR-CT Unit, Policlinico Umberto I^o, Università degli Studi di Roma "La Sapienza," Rome, Italy.

Address reprint requests to Dr Claudio Di Biasi, Unità di Risonanza Magnetica, I Clinica Medica, Università degli Studi di Roma "La Sapienza," Viale del Policlinico, 00161 Rome, Italy.

AJNR 15:1956-1958, Nov 1994 0195-6108/94/1510-1956 © American Society of Neuroradiology

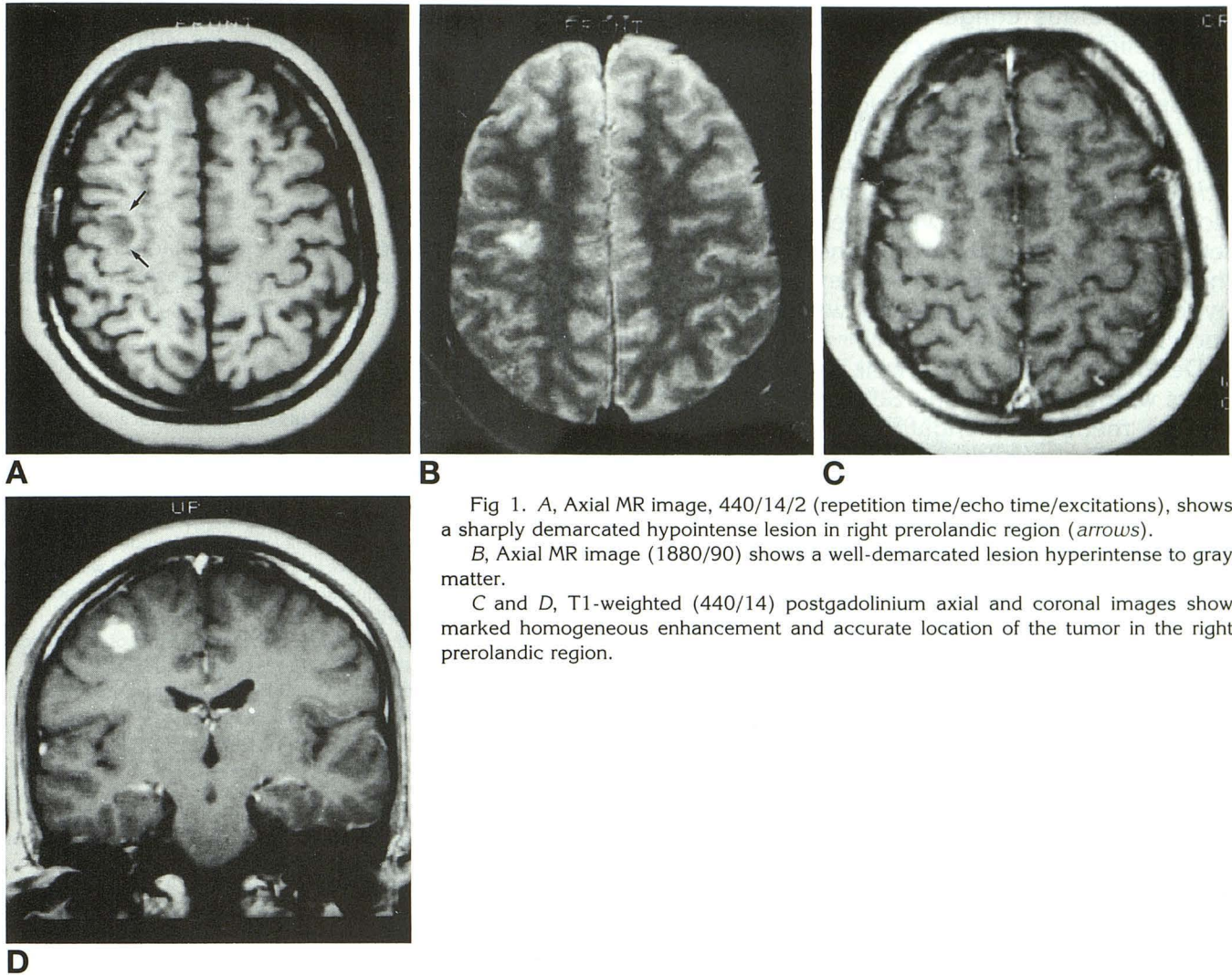


Fig 1. A, Axial MR image, 440/14/2 (repetition time/echo time/excitations), shows a sharply demarcated hypointense lesion in right prerolandic region (arrows).

B, Axial MR image (1880/90) shows a well-demarcated lesion hyperintense to gray matter.

C and D, T1-weighted (440/14) postgadolinium axial and coronal images show marked homogeneous enhancement and accurate location of the tumor in the right prerolandic region.

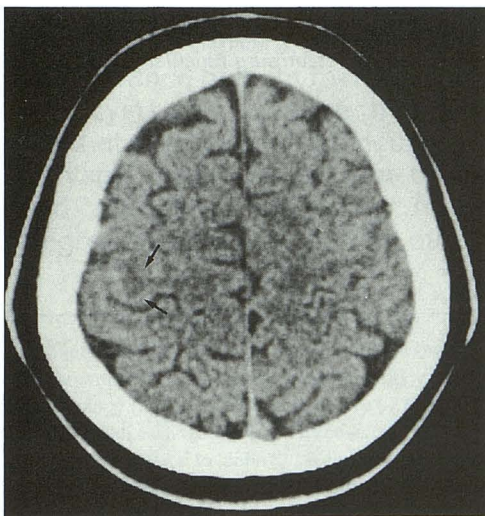


Fig 2. Axial precontrast CT scan shows a round slightly hypodense lesion in the right prerolandic region (arrows).

The site of origin of intracerebral schwannomas remains uncertain. Theories regarding the origin of intramedullary schwannomas have been proposed by several authors (1, 2, 8–12). The most generally accepted theory is that these tumors arise from proliferation of Schwann cells of the perivascular nerve plexus (1–8). According to Riggs and Clary, hyperplasia of the perivascular nerve plexus may be developmental in origin or caused by a chronic disease (8). Feigin and Ogata proposed that Schwann cells may originate from differentiation of multipotential mesenchymal cells within the central nervous system (9). In a review of previous case reports, Stefanko et al underlined the prevalence of supratentorial location and the preponderance in male patients in the first three decades (1). On the basis of these considerations, they suggested the possibility of a

hamartomatous congenital origin of Schwann cells, which can undergo conversion to neoplastic cells.

Russell and Rubinstein suggested later that pial cells sometimes can undergo conversion to Schwann cells. They proposed the term *schwannosis* to indicate a hamartomatous lesion consisting of Schwann cells and related reticulin fibers located within the substance of the spinal cord and brain. They stressed the hypothesis of the origin of schwannomas from such foci (2).

De Meyer reported aberrant peripheral nerve fibers with accompanying Schwann cells entering the medulla oblongata with vagal rootlets (10), and Prakash hypothesized that they may form a nidus for the development of an intramedullary schwannoma (11). Ramamurthi et al suggested, on hypothetical grounds, that displaced neural crest cells may be the source of the tumors; they postulated that such cells may possess increased neoplastic potential (12).

Therefore, the origin of these tumors remains unclear, despite the theories that have been proposed; because schwannomas occur in the brain and in the spinal cord, the hypothesis of an ectopic origin of a central nervous system schwannoma is certainly the most attractive one.

We could find only one case reported in literature of a malignant schwannoma, with three recurrences in 9 months after surgical treatment (1). All the other cases reported in the literature and our case referred to benign schwannomas.

In our experience, and in all cases we found in the literature, neuroradiologic findings were not specific for the diagnosis of intracerebral schwannoma. We excluded a metastatic lesion

because of the lack of a primary extracerebral tumor and the absence of surrounding edema. The diagnosis on the basis of CT and MR was primary glioma.

Acknowledgments

We thank the radiology technicians, Dino D'Amico and Stephano Caprasecca, for their precious assistance in the preparation of images and figures.

References

1. Stefanko SZ, Vuzevski VD, Maas AIR, et al. Intracerebral malignant schwannoma. *Acta Neuropathol* 1986;71:321-325
2. Russell DS, Rubinstein LJ. *Pathology of Tumours of the Nervous System*. 5th ed. London: Edward Arnold, 1989:100-109
3. Goldberg HI, Spagnoli MV, Grossman RI. High field MRI evaluation of acoustic neuroma. *Acta Radiol* 1986;369 (suppl):173-175
4. Miettinen M, Saari A. Pheochromocytoma combined with malignant schwannoma: unusual neoplasm of the adrenal medulla. *Ultrastruct Pathol* 1988;12:513-537
5. Myssiorek DJ, Silver CE, Valdes ME. Schwannoma of the cervical sympathetic chain. *J Laryngol Otol* 1988;102:962-965
6. Gibson AAM, Hendrick EB, Conen PE. Intracerebral schwannoma: report of a case. *J Neurosurg* 1966;24:552-557
7. Bruni P, Esposito V, Greco R, et al. Solitary intracerebral schwannoma in von Recklinghausen disease. *Surg Neurol* 1984;22:360-364
8. Riggs HE, Clary WU. A case of intramedullary sheath cell tumor of the spinal cord: consideration of vascular nerves as a source of origin. *J Neuropathol Exp Neurol* 1957;16:332-336
9. Feigin logata J. Schwann cells and peripheral myelin within human central nervous tissues: the mesenchymal character of Schwann cells. *J Neuropathol Exp Neurol* 1971;30:603-612
10. De Meyer W. Aberrant peripheral nerve fibres in the medulla oblongata of man. *J Neurol Neurosurg Psychiatry* 1965;28:121-123
11. Prakash B, Roy S, Tandon PN. Schwannoma of the brain stem: case report. *J Neurosurg* 1980;53:121-123
12. Ramamurthi B, Anguli VC, Iyer CGS. A case of intramedullary neurinoma. *J Neurol Neurosurg Psychiatry* 1958;21:92-94