AJNR

Intracranial chondroma.

R D Brownlee, R J Sevick, N B Rewcastle and B I Tranmer

AJNR Am J Neuroradiol 1997, 18 (5) 889-893
http://www.ajnr.org/content/18/5/889.citation

Radiologic-Pathologic Correlation
Intracranial Chondroma

Richard D. Brownlee, Robert J. Sevick, N. Barry Rewcastle, and Bruce I. Tranmer

From the Department of Clinical Neurosciences (R.D.B., R.J.S., N.B.R., B.I.T.), Radiology (R.J.S.), and Pathology (N.B.R.), Foothills Hospital, University of Calgary (Alberta, Canada)

History

Clinical

A 42-year-old woman had a change in the character of her migraine headaches. Her usual pattern consisted of throbbing frontoparietal headaches that occurred at biweekly intervals for many years. The new headaches had begun several months before and she described them as "ice pick" headaches occurring three to four times per day in the right frontoparietal region. She also experienced the new onset of light scintillations in her left eye and an intermittent feeling of a "bandlike" tightness around her head. The patient had no history of seizures and no focal neurologic symptoms. Findings of neurologic and general physical examinations were normal. Laboratory investigations revealed a mild iron-deficiency anemia that responded to iron therapy.

Imaging

Radiologic investigations included computed tomography (CT), magnetic resonance (MR) imaging, and cerebral angiography.

The CT scan (Fig 1A) revealed an oval-shaped extraaxial mass in the left frontoparietal parasagittal region. The mass was heterogeneous with a thick hyperdense peripheral rim, an isodense to hypodense central core, and a thin peripheral rim of contrast enhancement. The underlying brain was compressed but there was no intraaxial abnormality.

Coronal T1-weighted MR images showed an extraaxial mass with a thick rim hypointense relative to brain and a more hypointense core (Fig 1B). A small focus of hyperintensity was also evident between the tumor and the brain. There was a thin peripheral rim of contrast enhancement and slight enhancement of the peripheral aspect of the lesion (Fig 1C). Axial T2-weighted images (Fig 1D) showed a heterogeneous, hypointense mass over the convexity with punctate areas of hyperintensity. The brain parenchyma was normal.

Cerebral angiography (Fig 1E) showed the lesion to be avascular with displacement of adjacent cortical arteries and veins. The superior sagittal sinus was patent with mild irregularity at the site of the lesion, suggesting compression or invasion by the tumor. Although the imaging findings were atypical, meningioma was considered the most likely preoperative diagnosis.

Surgery

The lesion was approached via a left frontoparietal craniotomy. The exposed dura appeared grossly normal. A hard mass in the

Address reprint requests to Dr Robert J. Sevick, Department of Radiology, Foothills Hospital, 1403 29th St NW, Calgary, Alberta, Canada T2N 2T9.

Index terms: Brain neoplasms; Chordoma; Radiologic-pathologic correlations

AJNR 18:889–893, May 1997 0195-6108/97/1805–0889

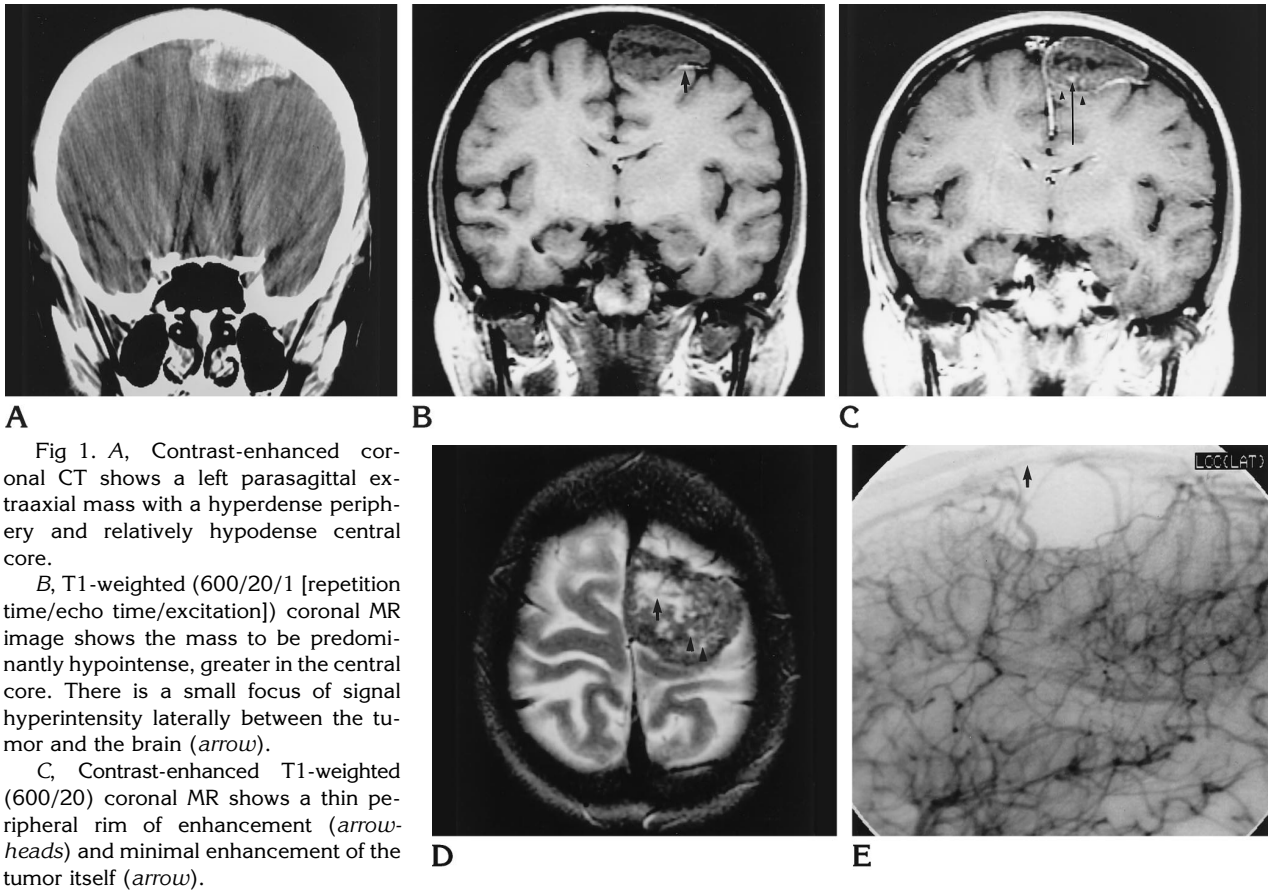

Fig 1. *A*, Contrast-enhanced coronal CT shows a left parasagittal extraaxial mass with a hyperdense periphery and relatively hypodense central core.

B, T1-weighted (600/20/1 [repetition time/echo time/excitation]) coronal MR image shows the mass to be predominantly hypointense, greater in the central core. There is a small focus of signal hyperintensity laterally between the tumor and the brain (*arrow*).

C, Contrast-enhanced T1-weighted (600/20) coronal MR shows a thin peripheral rim of enhancement (*arrowheads*) and minimal enhancement of the tumor itself (*arrow*).

D, Axial T2-weighted (3200/80) MR image shows hyperintense signal centrally (*arrow*), with a hypointense periphery containing punctate foci of hyperintensity (*arrowheads*).

E, Lateral view of a digital subtraction angiogram, left common carotid artery injection. The lesion is avascular and displaces adjacent cortical vessels. There is some effacement of the superior sagittal sinus adjacent to the lesion (*arrow*). *Figure continues.*

left parasagittal region was identified. The dura was incised in a U-shaped fashion around the mass and reflected toward the superior sagittal sinus. The mass was not attached to the overlying dura. It had a multinodular gray-pink appearance and was well circumscribed and indented the underlying brain. The tumor was weakly adherent to the falx cerebri and superior sagittal sinus, from which it was easily freed with blunt dissection. Removal of the mass produced some bleeding from the superior sagittal sinus, which was easily controlled with absorbable gelatin sponge and gentle pressure. There was a clear arachnoidal plane between the mass and the brain, locating it in the subdural space and allowing the entire tumor to be removed with minimal dissection. The patient tolerated the procedure well and was discharged from hospital on the third postoperative day.

Pathology

A firm, slightly lobulated, gray-white mass measuring 9.0 × 5.0 × 3.0 cm and weighing 12.5 g was submitted in toto (Fig 1F). Although firm, the mass was easy to cut, suggesting that it was not calcified. Sections of the tumor, cut in the coronal plane (Fig 1G), revealed a dense, white, thick outer rim of tissue and a delicate, nearly transparent inner core.

Microscopically, the tumor consisted of two distinct regions. The majority of the tumor was composed of a very dense outer zone of mature hyaline cartilage (Fig 1H). A central core of much less dense extracellular material contained thin-walled, endothelium-lined vessels that were not present in the dense outer zone of hyaline cartilage. The central material did not stain with hematoxalin-eosin, periodic acid–Schiff, or alcian blue.

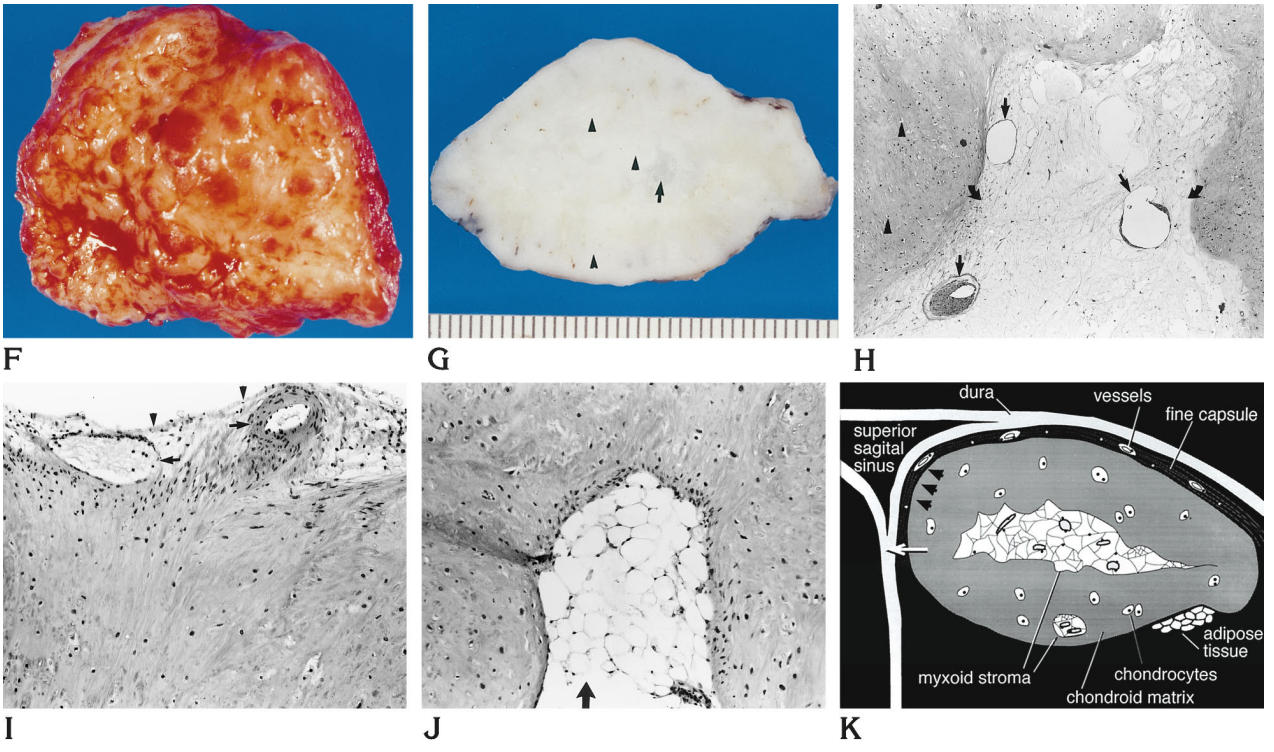

Fig 1, *continued.*

F, The fresh specimen measured 9.0 × 5.0 × 3.0 cm, weighed 12.5 g, and was firm with a granular irregular appearance. The surface adjacent to the brain is illustrated in an axial plane.

G, Gross specimen cut in the coronal plane appears white with a thick, dense outer rim of mature hyaline cartilage (*arrowheads*) and a delicate central core (*arrow*) of less dense extracellular material.

H, Microscopic section shows mature hyaline cartilage with scattered lacunes containing single chondrocytes (*arrowheads*) surrounding a core of less dense extracellular material (*curved arrow*). Several thin-walled vessels lined by endothelium (*arrows*) were present within the extracellular material in the core of the tumor (hematoxylin-eosin, original magnification ×40).

I, Microscopic section shows the fibrous capsule (*arrowheads*) containing small vessels of different caliber and wall thickness (*arrows*) surrounding the tumor (hematoxylin-eosin, original magnification ×40).

J, Microscopic section shows a small focus of adipose tissue (*arrow*) that was present on the surface of the tumor adjacent to the brain (hematoxylin-eosin, magnification ×100).

K, Anatomic drawing of the location and microscopic features of the intracranial chondroma. The tumor was located between the dura and the brain, adjacent to the falx cerebri (*arrow*). It minimally compressed the surface of the brain (not depicted) and was weakly adherent to the superior sagittal sinus (*arrowheads*).

The outer zone of mature hyaline cartilage consisted of scattered lacunes containing single chondrocytes within a dense chondroid extracellular matrix. In a few locations there were four or five chondrocytes within a single lacune. These cells exhibited small, round, regular nuclei but no pleomorphic features and no mitotic figures. There was no evidence of osteoid formation or calcification.

A delicate fibrous membrane (Fig 1I) containing small vessels and arachnoid granulations surrounded the tumor. A single minute focus of mature adipose tissue (Fig 1J) was present at one site on the outer surface of the tumor adjacent to the brain. An anatomic drawing of the tumor is presented in Figure 1K.

Discussion

Clinical Presentation

The clinical presentation of intracranial chondromas have included headache (1, 2), seizures (1, 3, 4), focal neurologic deficits (2, 5), and papilledema (3), depending on their location. The “ice pick” headaches and light scintillations experienced by this patient are difficult to explain physiologically. Intracranial chondromas have been reported in pa-

tients ranging from 15 months to 60 years of age, with a peak incidence in the third decade (4, 6) and no preference for either sex (6).

Etiology

Intracranial chondromas are slowly growing tumors that can arise from the skull base, from the dura (7, 8), from the brain parenchyma (7, 5, 9), or within the ventricles. The true cause is unknown. They have been reported to arise at sites of previous trauma (9, 10) and in association with systemic chondromatoses such as Ollier disease (11) and Mafucci syndrome (12). Our patient had no history of head trauma and no obvious evidence of systemic chondromas, although a detailed search was not performed.

Skull base chondromas usually remain extradural and are presumed to arise from embryonic cartilage rests along the basilar synchondroses (1, 5). Those arising from the dura can be totally ensheathed by the layers of the dura (13) or attached to the subdural surface of the dura or falx. In both instances an origin is postulated from embryonal cartilaginous rests (9) or multipotential mesenchymal cells that undergo metaplasia and chondroid formation (1). Of the small number of intraparenchymal chondromas, only a few are truly parenchymal and reportedly arise by metaplasia in an existing glioma (5, 14). The others are intraventricular and are usually associated with choroid plexus (14). Chondromas of dural and choroid plexus origin may arise as a result of metaplasia of arachnoid elements in that their location within the cranium is similar to meningiomas.

A single isolated focus of mature adipose tissue was attached to the surface of the chondroma. Mature adipose cells were not admixed with the cartilage in the chondroma. Because we could not exclude the possibility that this focus might represent a small rest of mature adipose tissue (commonly encountered on the surface of the falx cerebri) displaced by the enlarging chondroma, this lesion was not designated as a benign mixed mesenchymoma.

Prognosis

Intracranial chondromas are circumscribed masses of well-differentiated, cytologically benign hyaline cartilage (8). They tend not to invade or destroy the surrounding parenchyma and there is little tendency for sarcomatous change (4). Total excision is frequently possible (1–5, 7, 9, 13, 15, 16) and is curative, with no recurrence reported on long-term follow-up (2–4, 7).

Radiologic-Pathologic Correlation

CT

On CT, chondromas can be hyperdense (1) or hypodense (4) relative to brain. In the present case, as in previous reports (15, 16), the lesion had mixed attenuation with a thick hyperdense rim and hypodense core. Contrast enhancement is usually minimal and can be evident within the substance of the tumor (3, 13, 15). In our patient, enhancement was restricted to a thin peripheral rim around the tumor. On pathologic examination, the thick outer rim of hyperdensity consisted of mature hyaline cartilage with normal chondrocytes scattered throughout a dense chondroid matrix (Fig 1H). There was no evidence of osteoid formation or calcification. The hypodense core was acellular and consisted of a delicate extracellular matrix containing thin-walled vessels. The thin rim of contrast enhancement corresponded to the fibrous capsule (Fig 1I) containing small vessels.

MR

On MR imaging, the tumor showed hypointense signal on T1-weighted images and mixed hypointense and hyperintense signal on T2-weighted images. A rim of enhancement as well as minimal enhancement within the lesion were shown with infusion of contrast material (Fig 1C). These features are similar to those in a previously reported case (13). The thick peripheral rim of mature hyaline cartilage was slightly hypointense relative to brain on T1-weighted (1B) and T2-weighted (Fig 1D) images. The central zone of loose extracellular material containing thin-walled vessels appeared even more hypointense on T1-weighted images and hyperintense on T2-weighted images. On T1-weighted images there was a small hyperintense focus between the tumor and

the brain (Fig 1B, arrow), which we believe represented the small focus of adipose tissue (Fig 1J, arrow). The peripheral zone of enhancement (Fig 1C, arrowheads) corresponded to a fibrous capsule containing small vessels (Fig 1I). Punctate areas of signal hyperintensity on T2-weighted images (Fig 1D, arrowheads) corresponded to islands of the less dense extracellular material present in the core of the tumor (Fig 1H, curved arrow).

Angiography

Cerebral angiography (Fig 1E) revealed an avascular mass, as previously reported (3–5). On pathologic examination, the dense cartilagenous component of the tumor appeared avascular, while the core of extracellular matrix contained several thin-walled vessels of venous caliber (Fig 1H, arrows). These vessels were not evident on angiography, suggesting that flow through them was very slow. The fibrous capsule (Fig 1I, arrowheads), which enhanced with contrast on CT (Fig 1A) and MR (Fig 1C), also contained small vessels (Fig 1I, arrows) which were not evident on angiography (Fig 1E).

Summary

We have presented a case of intracranial chondroma with radiologic-pathologic correlation. MR imaging of intracranial chondromas reveals a peripheral zone of hypointensity relative to brain on T1- and T2-weighted images that represents mature hyaline cartilage. A second, more central region of hypointensity on T1-weighted images and of hyperintensity on T2-weighted images must reflect the less dense extracellular material. These lesions can be misdiagnosed as meningiomas. Imaging features that may help to distinguish chondromas from meningiomas include a heterogeneous appearance on MR imaging, a lack of or minimal contrast enhancement on CT and MR imaging, and an avascular appearance on cerebral angiography.

Acknowledgments

We thank Marilyn Mora for her artistic assistance in creating the anatomic drawing of the chondroma (Fig 1K).

References

1. Acampora S, Troisi F, Fusco G, Del Gaizo S. Voluminous intracranial chondroma. *Surg Neurol* 1982;18:254–257
2. Hardy RW, Benjamin SP, Gardner WJ. Prolonged survival following excision of dural chondroma: case report. *J Neurosurg* 1978;48:125–127
3. Ozgen T, Pamir N, Akalan N, Bertan V, Onol B. Intracranial solitary chondroma. *J Neurosurg* 1984;61:399–401
4. Mapstone TB, Wongmongkolrit T, Roessman U, Ratcheson RA. Intradural chondroma: a case report and review of the literature. *Neurosurgery* 1983;12:111–114
5. Ahyai A, Spoerri O. Intracerebral chondroma. *Surg Neurol* 1979;11:431–433
6. Dutton J. Intracranial solitary chondroma: case report. *J Neurosurg* 1978;49:460–463
7. Burger PC, Scheithauer BW, Vogel FS. *Surgical Pathology of the Nervous System and Its Coverings*. 3rd ed. New York, NY: Churchill Livingstone Inc, 1991;96–98
8. Burger PC, Scheithauer BW. *Tumors of the Central Nervous System: Atlas of Tumor Pathology*. 3rd series, fascicle 10. Washington DC: Armed Forces Institute of Pathology; 1994; 302
9. Chorobski J, Jarzymski J, Ferens E. Intracranial solitary chondroma. *Surg Gynecol Obstet* 1939;68:677–686
10. Dukes HT, Odom GL. Discrete intradural osteoma: report of a case. *J Neurosurg* 1962;19:251–253
11. Traflet RF, Babaria AR, Barolat G, Doan HT, Gonzalez C, Mishkin MM. Intracranial chondroma in a patient with Ollier's disease: case report. *J Neurosurg* 1989;70:274–276
12. Chakrabortty S, Tamaki N, Kondoh T, Kojima N, Kamikawa H, Matsumoto S. Maffucci's syndrome associated with intracranial enchondroma and aneurysm: case report. *Surg Neurol* 1991;36:216–220
13. Nakazawa T, Inoue T, Suzuki F, Nakasu S, Handa J. Solitary intracranial chondroma of the convexity dura: case report. *Surg Neurol* 1993;40:495–498
14. Hirwonen J, Heikinheimo H. A case of intracerebral chondroma. *Acta Pathol Microbiol Scand* 1969;76:19–24
15. Yang PJ, Seeger JF, Carmody RF, Fleischer AS. Chondroma of falx: CT findings. *J Comput Assist Tomogr* 1986;10:1075–1076
16. Hadadian K, Abtahii H, Asil ZT, Rakhshan M, Vessal P. Cystic falcine chondroma: case report and review of the literature. *Neurosurgery* 1991;29:909–912
17. Dretzschmar HA, Eggert HR, Beck U, Furmaier R. Intracranial chondroma: case report. *Surg Neurol* 1989;32:121–125