

# NEUROIMAGES

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HOPITAL NEUROLOGIQUE DE MONTREAL  
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## Milano Due

Settembre 4-8, 1990

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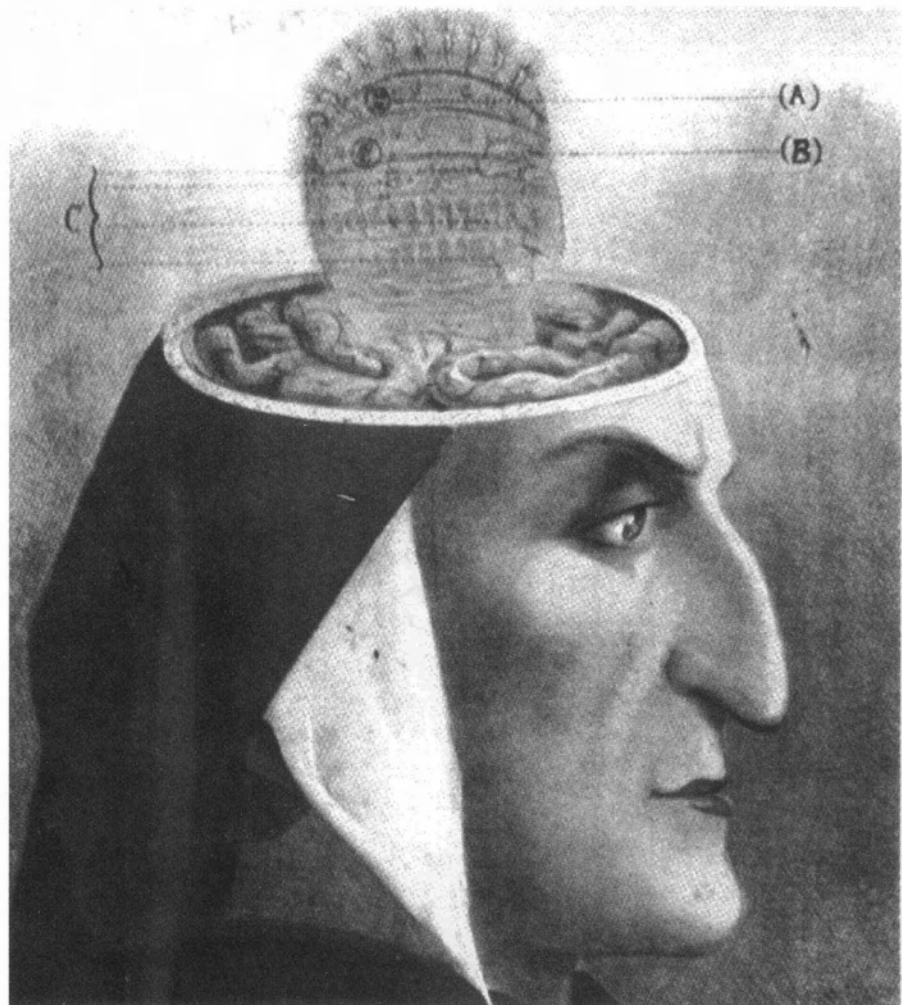
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# Meningioma Cysts

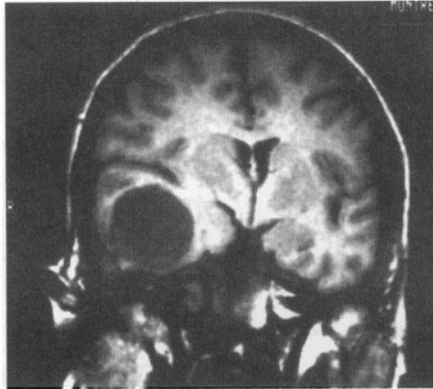
D. Melançon

Cysts are rarely found in meningiomas. Several anatomic configurations of meningiomas associated with cyst formation have been reconized on the basis of the relationships between brain, tumor, cyst and subarachnoid cyst.

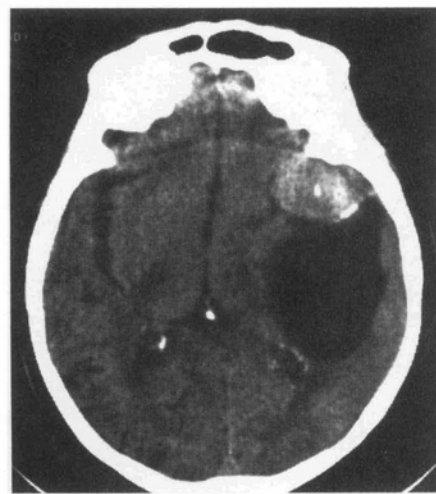
We present two examples; one (Fig1) where the cyst is in the brain, and the other (Fig 2) where the cyst is formed by a loculation of the subarachnoid space as the tumor grows inward: for that reason no enhancement is seen around the cyst.



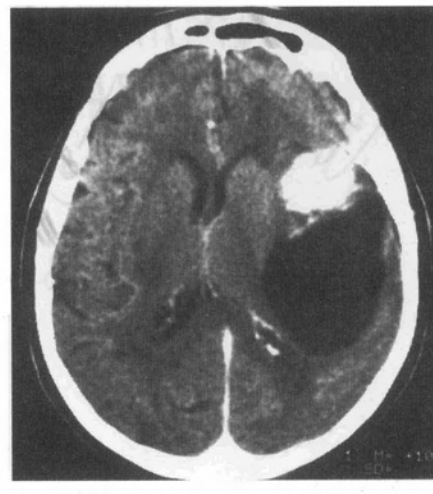
**Fig. 1a** Ct enhanced. Thin rim of enhancement around the cyst.



**Fig. 1b** MRI coronal T1. Large hypointense lesion within temporal lobe.

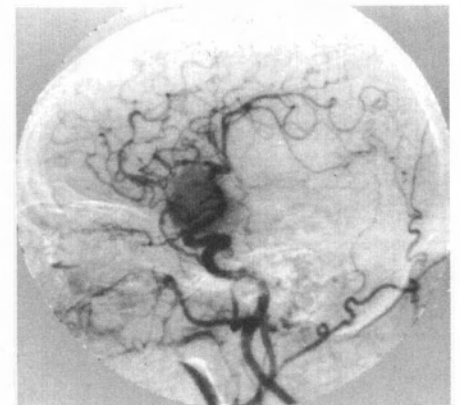


**Fig. 2a** CT plain. Hyperdense nodule along the lateral sphenoidal ridge, with a large hypodensity posterior to it in the temporal region.



**Fig. 2b** CT enhanced. The nodule enhances but not the wall of the cyst; the sylvian vessels are displaced.

*References:*  
*Worthington C. and al., Neurology 1985; 35: 1720-1724*  
*Nauta HJW. and al., J Neurol Neurosurg Psychiatry.*  
*Rengachary S. and al., Neurosurgery 1979; 4; 107-114.*



**Fig. 2c** Carotid angiogram. Homogeneous blush of nodule; avascular region behind.

# CT & Vertigo

## D. Melançon

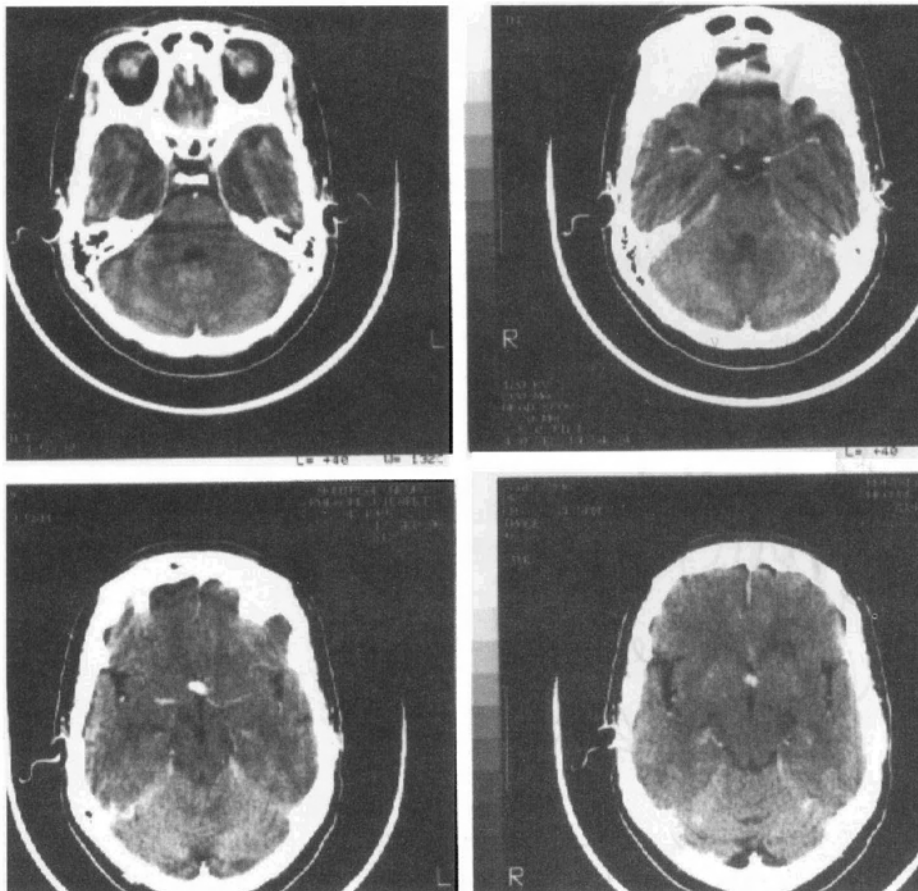
I was invited to participate in the "THIRD EUROPEAN COURSE IN NEURORAIDIOLOGY" from September 4 to 8 in Milano, Italy, and sat on the panel discussing the investigation of different neurological disorders. One of these was VERTIGO. I mentioned our policy of first investigating this disorder with CT and IV contrast, 5mm sections of the posterior fossa, soft tissue and bone windows. It was debated whether these patients should come to CT upon the

only clinical mention of VERTIGO. I bring the following examples of such a circumstance.

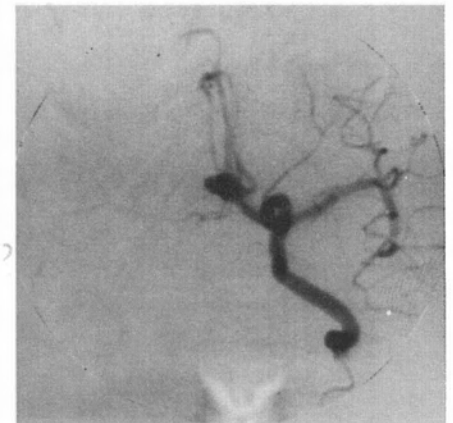
A forty-one(41) year old lady was referred to our CT unit on an out patient basis, with the clinical information of a recent episode of VERTIGO. She was scanned according to the protocol mentioned above and an anterior communicating artery aneurysm was found (Fig. 1a). She was admitted to our neurosurgical unit, an angiogram was performed and the aneurysm was

confirmed (Fig. 1c). Upon careful questioning, it was found that the episode was preceded by a violent headache and accompanied by nausea and vomiting. This occurred on August 18th. She was seen by the neurologist in the clinic only on August 31st, the scan requested that day but scheduled on September 17th. She was operated on on September 19th and left the hospital in good condition.

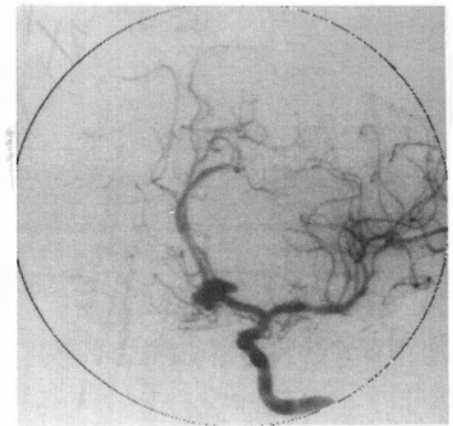
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**Figure 1a**



**Figure 1b**



**Figure 1c**

# Milano Due European Course of Neuroradiology.



## CT & Vertigo (continued)

The second patient is a young lady of twenty-eight (28) years who was followed for one year by her ENT specialist as a case of Meniere's disease before being referred for CT scanning: the scan disclosed a tumour in the 4th ventricle (Fig. 2a). At surgery, an ependymoma was removed subtotally.

Vertigo may be a non specific clinical presentation and may lack proper attention.

Definitions of dizziness and vertigo are often difficult to apply in clinical practice. Dizziness is described as a

feeling of dazed lightheadedness or a feeling of impending falling, whereas vertigo implies a sense of rotation either of the patient or of the environment, coupled with a sense of imbalance: it is a symptom of disturbance in the vestibular system; when of sudden onset, it is usually caused by acute viral labyrinthitis.

That careful medical history should have suspected the true nature of the events is no argument against the access to CT scanning for the patient: CT picked up the lesions and saved the patients, a possible disastrous second

episode for the first one, and some serious complications for the second.

A logical approach would be to first rule out the remote possibility of tumour and then proceed to the other tests for final diagnosis, and not to go through all the other tests before CT. Not too often, but often enough, the first sequence may hurt the patient.

I take this opportunity to congratulate my friend Giuseppe Scotti for the success of the Course and thank him for having invited me to participate.

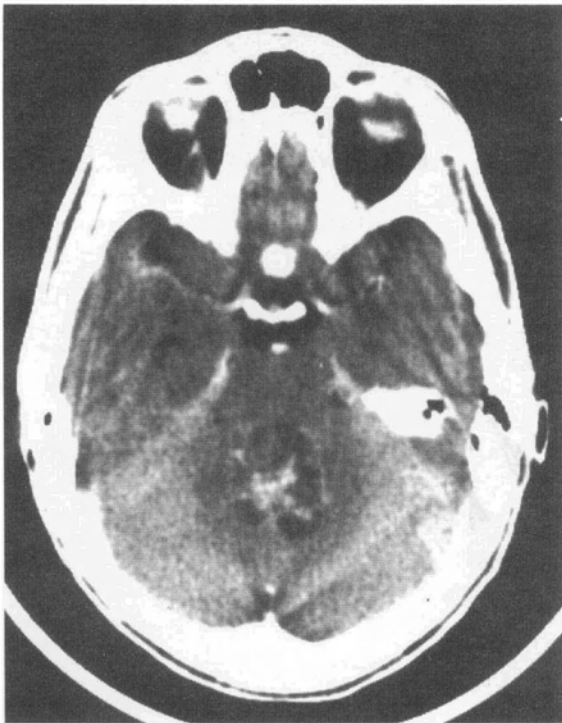
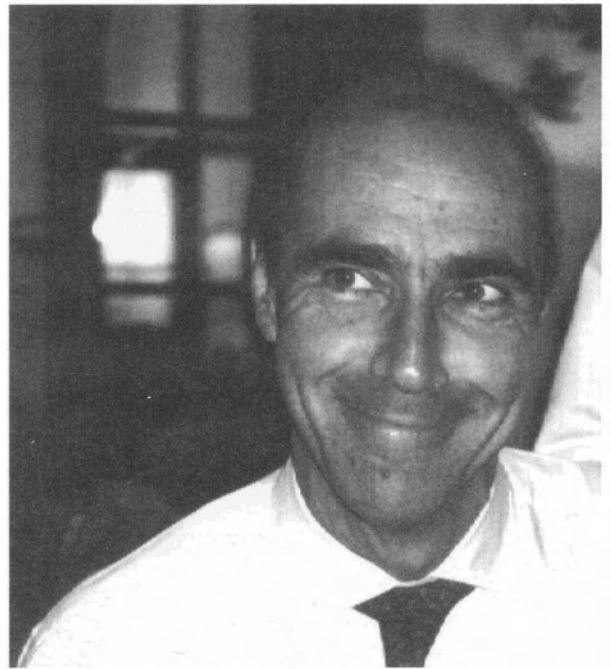


Figure 2



"Bravo Giuseppe"

# Interhemispheric Lipoma

Alvaro Zuluaga Gomez, Raquel del Carpio-O'Donovan.

## Case Report.

This thirty-nine(39) year old man had been on antiseizure medication for most of his life. In 1970, he had a frontal craniotomy, of which details are unknown. He was sent to us for MRI. Although the diagnosis of "Callosal Lipoma" was known, associated anomalies were being investigated.

CT: as in the classical cases, an area of hypodensity (low attenuation) immediately superior to the lateral ventricles, with several calcifications, was seen (fig. 1). Calcifications may be curvilinear, around the periphery of the lesion or nodular within the lipoma.

MRI: the lipoma is identified by its bright signal on T1-weighted (Fig. 2,3) and decreasing signal on T2-weighted sequences (Fig. 4).

"Callosal lipomas", more appropriately called interhemispheric lipomas, lie in the midline, in the interhemispheric cistern with variable asymmetry. The corpus callosum is always dysgenetic. In our case, it is totally absent, in others; depending on tumor location, the corpus callosum appears normal from the genu to the lipoma but callosal fibers are not identified beyond it.

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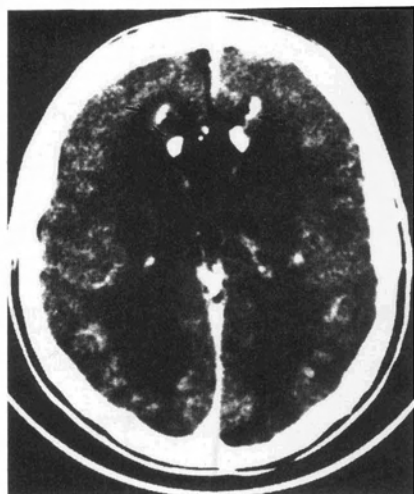
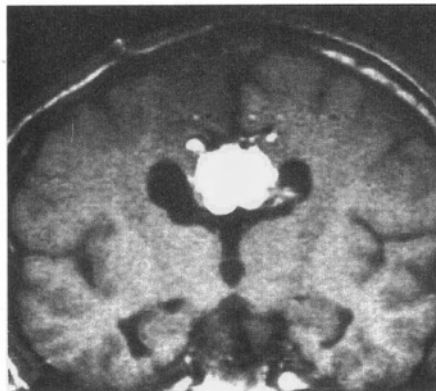
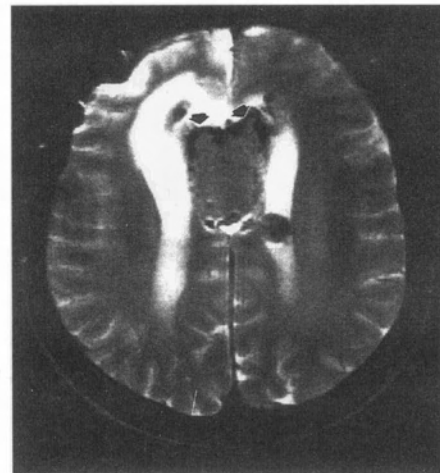


Figure 1. CT, axial, with contrast enhancement. 4x3 cm anterior sagittal, hypodense mass. Curvilinear and nodular calcifications in its anterior portion (arrows). Colpocephaly.



3. MRI, coronal SE 550/15. Callosal lipoma between third and interhemispheric fissure. Lateral ventricles have the steernhorn appearance seen in callosal agenesis.



4. MRI, axial, SE 2100/80. The CSF in the lateral ventricles is bright while the midline lipoma is isointense with the cortex: marked signal loss compared to T1 sequence. The curvilinear anterior calcifications are seen as zones of absent signal (arrows) The areas of hyperintensity in the right frontal lobe are related to remote surgery.

## References:

- 1- Barkovich. A.J. *Congenital Malformation of the Brain. In: Contemporary Neuroimaging. Pediatric Neuroimaging.* Raven Press. 1990. Vol 1:77-121.
- 2- Truwit. C.L., Barkovich A.J. *Pathogenesis of Intercranial Lipoma: An MR study in 42 patients. AJNR* 1990. 11, 4:665-674.
- 3- Kazner E, Stochdorph O, Wende S. *Intercranial Lipoma. Diagnostic and Therapeutic considerations. J. Neurosurg* 1980; 52: 234-245.



2. MRI, sagittal, SE 550/15. Comet like shape of the tumor which typically has high signal intensity on T1-weighted sequence. Absence of corpus callosum. Radiating pattern of medial gyri. Lack of convergence of calcarine and parieto-occipital sulci.

## Interhemispheric Lipoma (continued)

### Discussion.

Since 1818, when the first intracranial lipoma was described by Meckel, this lesion has kept puzzling the neuro scientists. Virchow and Chiari, in the late XIX century, proposed their theories and since then many others have appeared in the literature. Some authors favored the idea of hypertrophy of pre-existent fatty tissues, while others advanced the transformation or metaplasia theory as well as that of degeneration of glial tissue.

Lipomas have been included in the category of dermoid and epidermoid tumors. The mesodermal inclusion, or abnormal neurulation process hypothesis, explains the frequent association of lipomas to other brain and cranial anomalies and it was often quoted in case reports and related articles.

Recently, Truwit and Barkovich (2) in a study of forty-two (42) patients with intracranial lipomas, reviewed the previous theories and proposed their own, based on thorough observation and study of neuro-embryology. Theirs explains the origin of this malformation -nor a hamartoma, not a tumor- as related to persistence and mal-differentiation of the meninx. It also clarifies the concept of cisternal location of the lipoma.

As in our case and applying Truwit and Barkovich theory (2), the maldifferentiation occurs most frequently in the region dorsal to the lamina terminalis. Therefore this phenomenon may prevent formation of the massa commissuralis resulting in callosal agenesis.

Intracranial lipomas generally do not require therapy (3). In the case we present, the reason for the right frontal craniotomy (done several years ago and in another institution) is unclear.

Alvaro Zuluaga Gomez, Raquel del Carpio-O'Donovan.

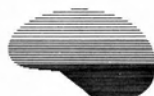
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