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Metastatic Lesions Involving the Cerebellopontine Angle

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PURPOSE: To evaluate the clinical and MR findings of metastatic lesions involving the cerebellopontine angle (CPA), which may be useful in differentiating them from the more commonly occurring benign CPA lesions. **METHODS:** Clinical and MR findings of 14 patients with clinical/radiologic (seven) or pathologic (seven) diagnoses of CPA metastasis were retrospectively reviewed. **RESULTS:** Useful clinical findings included acute onset and rapid progression of cranial nerve symptoms, especially 7th and/or 8th cranial nerve deficits (92.9%). Cranial nerve symptoms could be unilateral (50%) and frequently involved multiple cranial nerves (64.3%). MR findings showed significantly more extensive disease than suggested by clinical presentation, with 100% of patients having multiple cranial nerve involvement and 85.7% bilateral. Useful MR findings included small and/or bilateral CPA-enhancing lesions with relative isointensity to brain parenchyma on precontrast MR, with associated findings of multiple and/or bilateral cranial nerve and/or leptomeningeal lesions. **CONCLUSIONS:** These associated findings suggest that cerebrospinal fluid dissemination and/or leptomeningeal extension may be an important pathway for metastatic spread to the CPA. Because the CPA metastasis may be the initial or only site of metastasis, and may occur many years after the initial diagnosis of malignancy, MR findings with clinical correlation are not only useful for the detection of CPA metastases, but also for their differentiation from the more common benign CPA tumors.

Index terms: Brain neoplasms, metastatic; Cerebellopontine angle, neoplasms

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Most neoplasms of the cerebellopontine angle (CPA) are benign, commonly of neuroectodermal origin (1, 2). Malignant tumors of the CPA including primary malignancies of the cerebral parenchyma and metastatic carcinomas are rarely reported (2–4). The availability of computed tomography (CT) and magnetic resonance (MR) has significantly improved the diagnosis of CPA lesions (1–4). Although the literature on CT and MR appearance of the more commonly observed benign CPA tumors, such as acoustic neurinomas and meningiomas, is abundant, little information is available on metastatic lesions to the CPA. This gap is important because the management of benign and malignant CPA masses differs significantly. MR has proven to be more sensitive than CT in the detection of CPA lesions, due to the lack of bony artifact. For this study we evaluated the clinical and MR findings of metastatic lesions involving the CPA, which may be useful in differentiating them from the more commonly occurring benign CPA lesions.

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Materials and Methods

Fourteen cases with MR evidence of metastases to the CPA were reviewed retrospectively. The patients were nine men and five women ranging in age from 30 to 73 years (mean, 50.9 years). Their charts were reviewed with respect to the primary cancer, systemic metastasis, interval to the

TABLE 1: Summary of patient data and MR findings

Clinical Information								MR Findings					
Patient No.	Age (yr) /Sex	Primary Site	CN	Progression of Symptoms (wk)	Time of CPA Mets (mo)	Systemic Mets	Size (mm) of CAP Mass	T1 Signal	T2 Signal	Gd Enhancement	Brain Mets	Meningeal Disease	CN
1	68/M	Prostate	7	20	72	−	10 × 10 L	iso	iso	N/A	−	N/A	5–11
2	35/F	Breast	2,8[a]	2	20	+	3 × 8 R 3 × 12 L	iso	iso	+	+	+	3[a],5[a],8[a]
3	50/M	Lymphoma	3,7[a]	1	34	+	3 × 10 R 4 × 10 L	iso	iso-high	+	+	+	7[a],8[a]
4	67/M	Lung	8	1	16	+	3 × 5 R[b] 10 × 15 L	iso	iso	+	+	+	5,6,7[a],8
5	45/M	Ocular melanoma	8[a]	16	6	+	4 × 10 R 3 × 3 L	hyper	iso-high	+	+	−	8[a]
6	39/M	Nasopharynx (squamous)	5,6,7,8, 11,12	50	0[c]	+	3 × 8 R	iso	iso	+	−	+	5–12
7	30/M	Melanoma	5	1	10	+	5 × 12 R 5 × 14 L	iso	iso	+	−	+	5[a],7[a],8[a]
8	73/F	Lung	5,8[a]	6	0[c]	−	5 × 10 R 6 × 10 L	iso	iso	+	+	+	3,5[a],7[a],8[a], 9,10, 11
9	31/M	Lung	8	2	0[c]	+	4 × 4 R 4 × 4 L	iso	iso	+	+	+	5,8[a]
10	47/F	Breast	8[a]	4	26	+	5 × 10 R 5 × 10 L	iso	high	+	+	+	2,5,8[a]
11	46/M	Lung	2,7,8[a]	2	0[c]	−	4 × 5 R 4 × 5 L	iso	iso	+	+	+	2,3[a],4,5[a], 7[a],8[a]
12	75/M	Lung	8	2	23	+	4 × 8 R 5 × 15 L	iso	iso	+	+	+	7[a],8[a]
13	52/F	Breast	6[a],7	1	19	+	5 × 15 R	iso	iso	+	+	+	3,5[a],7–11
14	65/F	Breast	3,4,5,7, 8	3	15	+	5 × 6 R 5 × 9 L	iso	iso	+	+	+	3,4,5[a],7, 8[a]

Note—CN = cranial nerve involvement; Mets = metastases; Gd = gadolinium.

[a] Bilateral involvement.

[b] No IAC involvement.

[c] Initial diagnosis of primary tumor.

development of symptomatic CPA metastases, initial symptoms (including cranial nerve dysfunction), and the time of progression of symptoms (defined as the time at which significant initial clinical changes occurred, including the complete loss of cranial nerve function and/or development of new symptoms).

Thirteen of the 14 patients had undergone both noncontrast and gadolinium-enhanced MR studies. MR studies included T1-weighted, 350–650/20/2 (TR/TE/excitations), and T2-weighted, 2000–2500/90–120/1–2, spin-echo images with section thickness ranging from 3 to 10 mm. Postcontrast MR studies were obtained immediately after the intravenous injection of 0.1 mmol/kg of gadopentetate dimeglumine, using the same parameters as those of the precontrast T1-weighted images. All MR examinations were reviewed retrospectively with special attention to the size of the CPA lesions and MR characteristics, including pattern of contrast enhancement, brain metastasis, and involvement of the leptomeninges and cranial nerves.

Results

The clinical and MR findings of the 14 patients studied are summarized in Table 1. The primary tumors were lung carcinoma (five patients), breast carcinoma (four patients), melanoma (two patients), prostatic carcinoma, squamous cell carcinoma of the nasopharynx, and non-Hodgkin lymphoma (one patient each). Five patients had lumbar punctures for cerebrospinal fluid (CSF) cytology, and two had meningeal biopsies; all seven patients were positive for malignancy. Two of these seven patients with pathologic proof of leptomeningeal disease also had evidence of drop metastasis, demonstrated by the postcontrast lumbar MR studies. In 10 patients, a diagnosis of primary malignancy had been established 6–72 months (mean, 17.2 months) before the clinical or MR features of CPA metastases became evi-

dent. In the other four patients (patients 6, 8, 9, and 11), CPA metastasis was the initial evidence of somatic malignancy; the primary tumor was later proven to be adenocarcinoma of the lung in three patients and squamous cell carcinoma of the nasopharynx in the fourth. In the patient with prostatic cancer (patient 1), the CPA metastasis was the first sign and only subsequently proven site of metastatic disease 6 years after the initial diagnosis.

Clinical Findings

In general, the rate of progression of the clinical symptoms (cranial nerve dysfunction) was rapid. Eleven patients had fulminant and rapidly progressive neurologic symptoms (eight within 2 weeks and three within 6 weeks). In the other three patients (prostatic carcinoma, nasopharyngeal carcinoma, and ocular melanoma), symptoms developed over a period of 16–50 weeks (mean, 29 weeks) (Table 1). The patient with ocular melanoma had a favorable clinical response to melatonin treatment after the diagnosis of CPA metastasis. His disease status is currently stable. The other patient with squamous cell cancer had no evidence of the systemic spread (hematogenous) of tumor distantly, but instead had slow retrograde contiguous extension of tumor into Meckel's cave and leptomeninges, presumably via trigeminal nerve ramifications.

As expected, patients with CPA metastases had a high incidence of clinical symptoms related to the nerves of the internal auditory canal (IAC) (Table 1). Thirteen of the 14 patients (92.9%) had 7th and/or 8th cranial nerve symptoms, and the other had only a single trigeminal nerve symptom. Six of the 13 patients with 7th and/or 8th cranial nerve symptoms had bilateral occurrence (46%) (five had bilateral 8th and one had bilateral 7th), and two of the remaining seven patients had unilateral 7th and 8th cranial nerve dysfunction. Seven of the 13 patients who had 7th and/or 8th cranial nerve symptoms also had evidence of other cranial nerve dysfunction.

Extracranial systemic metastasis was common at the time of diagnosis of CPA metastasis and was found in 11 of the 14 patients (78.6%). Before the brain MR examination, clinical symptoms suggesting cerebral parenchymal metastasis were found in only six patients. However, on reevaluation, these symptoms were thought to be related most likely to a direct cranial nerve involvement rather than the presence of intraparenchymal metastasis.

MR Findings

MR features demonstrated much more extensive involvement than did clinical symptoms, including the total number of CPA lesions, incidence of multiple cranial nerve involvement, presence of leptomeningeal disease, and evidence of parenchymal brain metastasis (Table 1). Multiple (100%) (Figs. 1–5) cranial nerve or bilateral 7th and/or 8th (78.6%) (Figs. 1–4) cranial nerve involvement demonstrated by MR imaging was much more common than suggested by clinical symptoms. Associated parenchymal brain metastasis was common (11 of 14 patients, 78.6%).

MR demonstrated a total of 25 CPA lesions in 14 patients (three patients had only unilateral CPA lesions) (Fig. 5); 24 of the 25 showed involvement of the IAC (Figs. 1–5). Symptoms related to the 7th and/or 8th nerves were absent in six of the 24 IAC lesions (25%); three of the 24 IAC lesions (12.5%) had both 7th and 8th symptoms. One patient who had a CPA lesion without IAC involvement and ipsilateral symptoms also had MR evidence of a contralateral symptomatic CPA mass that did involve the IAC.

Eighteen of the 24 IAC lesions (75%) were relatively small (less or equal to 10 × 10 mm), and 18 of the 24 IAC lesions were also isointense (75%) to brain parenchyma on both T1- and T2-weighted MR images (Figs. 1 and 3). The other six lesions that were small but not isointense on MR included two lesions in a patient with ocular melanoma, which showed hyperintensity on T1-weighted images (Fig. 3) and mixed isointensity to mild hyperintensity on T2-weighted images. Two lesions in a patient with lymphoma and two lesions in a patient with breast carcinoma showed isointensity on T1-weighted images and mixed isointensity to high-signal intensity on T2-weighted images. Because most lesions were relatively small and isointense, only eight of the 24 (33.3%) IAC lesions were detected retrospectively on the precontrast study, and only four of these eight lesions were detected prospectively on the precontrast MR studies. These lesions were either hyperintense on T1-weighted images (ocular melanoma) or relatively large lesions.

All the CPA/IAC metastases were demonstrated by the contrast MR studies (13 patients). All but one of these 13 patients had evidence of leptomeningeal involvement demonstrated by

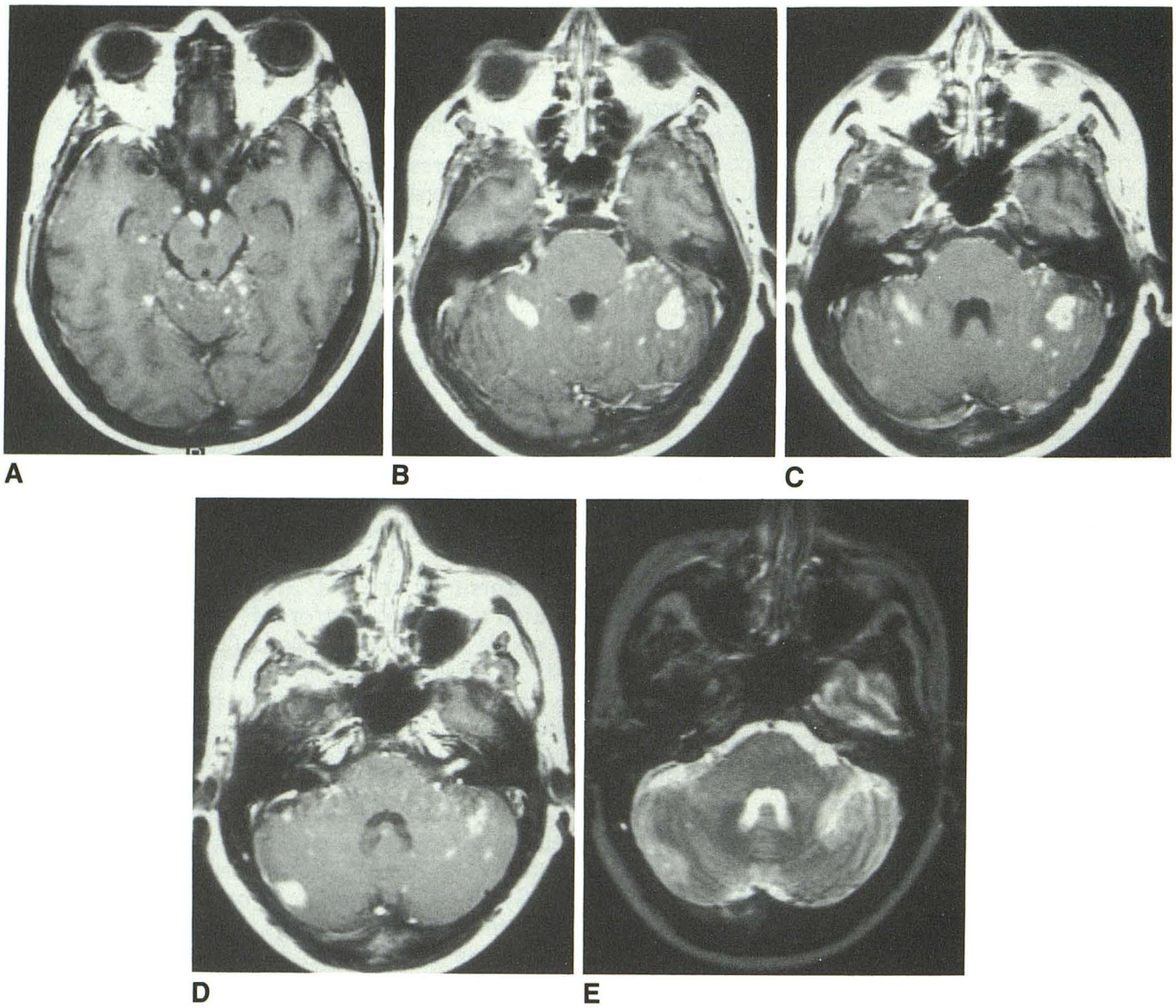


Fig. 1. Patient 8: Lung carcinoma.

A–D, Postcontrast axial T1-weighted images (600/20) from cranial to caudal (*A–D*) show bilateral involvement of 3rd (*A*), 5th (*B* and *C*), and 8th (*C* and *D*) cranial nerves. The involved cranial nerves are not significantly enlarged. Note multiple brain metastases and leptomeningeal disease.

E, Axial T2-weighted image (2500/100) corresponding to *D* demonstrates slightly enlarged left and normal right 8th cranial nerves with signal intensity similar to that of brain parenchyma. Leptomeningeal disease is not evident on the T2-weighted image.

contrast-enhanced MR. The only patient without MR evidence of leptomeningeal involvement on the contrast MR study had no surgery to prove whether the leptomeninges were involved.

Multiple cranial nerve involvement on MR limited to one side (unilateral) (Fig. 5) was noted in two patients (patients 1 and 6). One of these two patients (patient 1) did not have a contrast study for optimal detection of the lesion, and showed additional ipsilateral cranial nerve (5th, 6th, 9th-11th) and leptomeningeal involvement intraoperatively. The second patient with contiguous retrograde spread of nasopharyngeal carcinoma to Meckel's cave and meninges had cranial nerve involvement (5th-12th nerves) limited to the same side (Fig. 5). This patient had only positive neck nodes, most likely representing local lymphangitic spread, and no brain metastasis or other systemic involvement to suggest hematogenous spread of disease.

Discussion

Most CPA lesions are benign tumors, of which the most common are acoustic neurinomas, followed in frequency by meningiomas and epider-

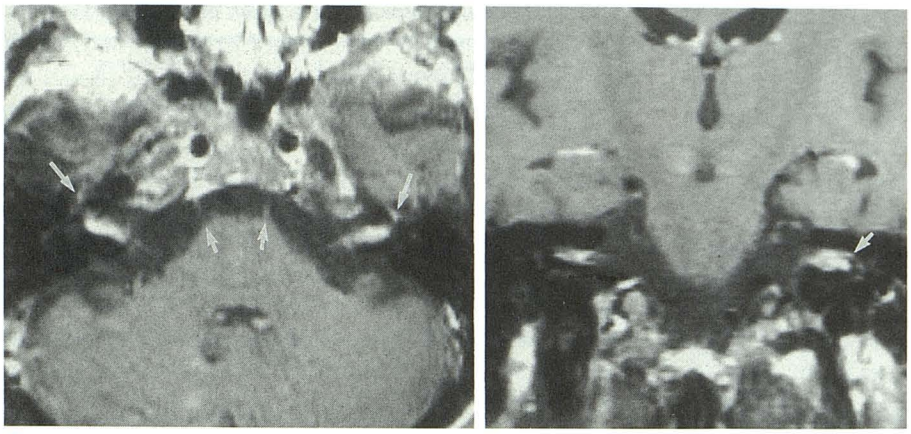
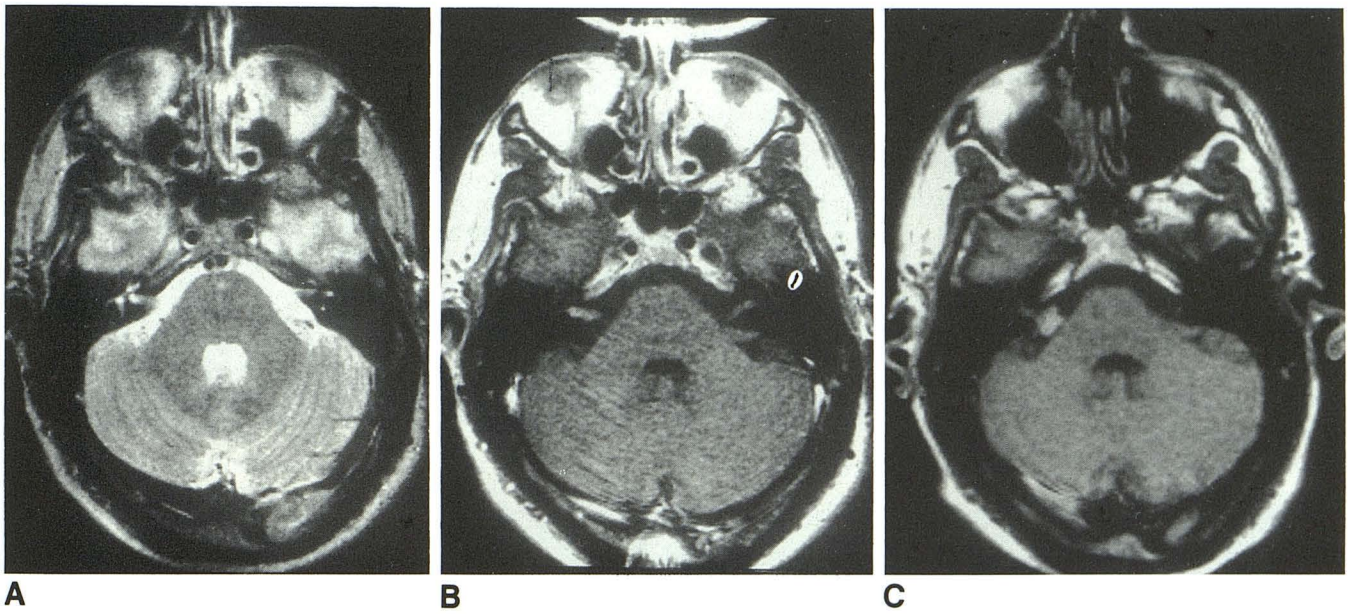


Fig. 2. Patient 3: Lymphoma.

A, Postcontrast axial T1-weighted image (600/20) shows bilateral IAC metastasis. Possible involvement of the geniculate ganglion (*long arrows*) is noted bilaterally. Neither 6th cranial nerve (*short arrows*) is involved.

B, Coronal T1-weighted image (750/20) again shows bilateral IAC involvement. Possible involvement of the left 7th cranial nerve (upper enhancing linear structure: *arrow*) is noted.



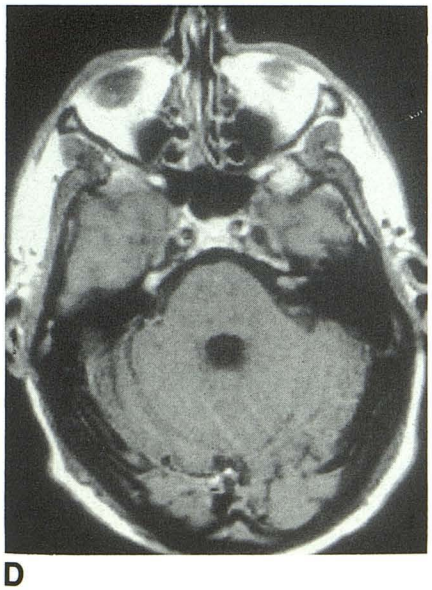


Fig. 3. Patient 5: Ocular melanoma.

A, Axial T2-weighted image (3000/90) shows relatively normal IAC bilaterally with the signal intensity of the 7th and/or 8th cranial nerves similar to that of brain parenchyma.

B, Postcontrast axial T1-weighted image corresponding to *A* shows abnormal enhancement within the IAC bilaterally.

C and *D*, Precontrast axial T1-weighted images (600/25) obtained 3 months after *A* and *B* show progressive enlargement of the bilateral CPA lesions.

moid cysts (Table 2). Vascular lesions (hemangiomas, arteriovenous malformations, aneurysms, and glomus tumors), lipomas, and arachnoid cysts represent 1% or less of all CPA lesions. Reported cases of CPA malignant neoplasms are rare. They include metastatic tumors and the more common primary brain tumors, such as astrocytomas, medulloblastomas, and ependymomas. Metastatic tumors are reported to represent 0.2–2% of all tumors (2–5). To our knowledge, only 10 cases of CPA metastases have been reported in the literature (3, 4, 6, 7). In five of these 10 cases, the primary malignancies were small cell lung cancer, breast cancer, squamous

TABLE 2: Incidence of benign and malignant CPA lesions (%)

Histologic Diagnosis	Incidence (%)	Reference
Schwannoma (acoustic neurinoma)	80–90	Mafee et al (1)
Meningioma	3–13	Martuza et al (2)
Epidermoid cyst	6–7	Mafee et al (1)
Primary malignancy	2	Robinson and Rudge (5)
Metastasis	0.2–2	Brackmann and Bartels (3) Kendall and Symon (4)
Arachnoid cyst, lipoma, vascular lesion	<1	Martuza et al (2)

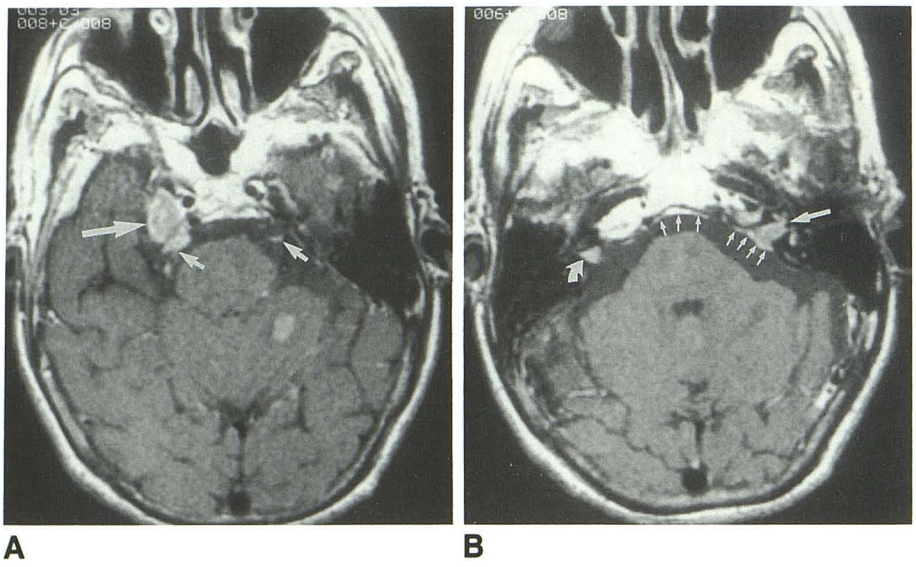


Fig. 4. Patient 8: Lung carcinoma.
A and *B*, Postcontrast axial T1-weighted images (600/20) show involvement of both trigeminal nerves (*A*, *small arrows*) and Meckel's cave (*A*, *large arrow*) and leptomeninges on the right side, and, more caudally (*B*), direct contiguous meningeal spread (*small arrows*) to the left IAC is evident (*large straight arrow*). Note the interface between the leptomeninges and left IAC. Also noted is involvement of the right IAC (*curved arrow*).

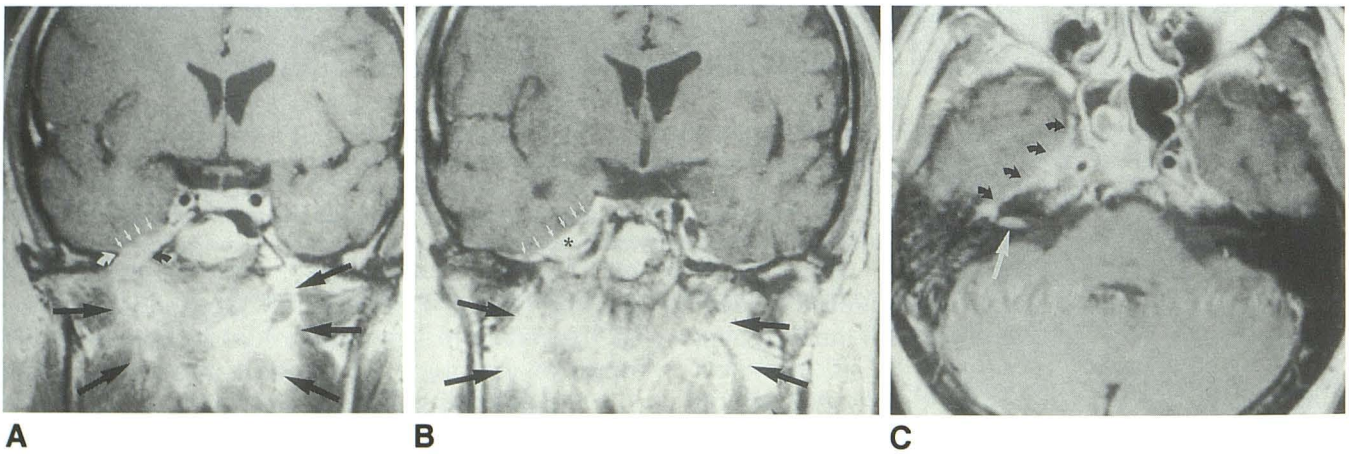


Fig. 5. Patient 6: Squamous cell carcinoma of the nasopharynx.
A-*C*, Postcontrast coronal (*A* and *B*) and axial (*C*) T1-weighted images (800/20) from anterior (*A*) to posterior (*B*) show a large mass (*A* and *B*, *large arrows*) in the nasopharynx with contiguous spread into Meckel's cave (*B*, *asterisk*) through the foramen ovale (*A*, *between curved arrows*). Leptomeningeal involvement of the middle cranial fossa was evidenced by direct erosion of the dura/meninges of the lateral wall of Meckel's cave (*A* and *B*, *small arrows*). Abnormal enhancement of the 7th and 8th cranial nerves within the right IAC (*C*, *straight arrow*), as well as the leptomeningeal involvement with loss of distinction of the lateral wall of Meckel's cave and direct contact of IAC on the same side (*C*, *curved arrows*), is noted. These findings suggest that CSF dissemination and/or leptomeningeal extension may be the cause of disease of the right CPA. No parenchymal involvement is noted.

cell carcinoma of the oropharynx (3), lymphoma (7), and adenocarcinoma of an unknown primary site (6). In the remaining five cases, the histologic features of the primary tumor were not specified (4).

The differentiation between metastatic and benign CPA lesions is important in patient management, but it can be difficult clinically and radiologically. Although metastatic disease to the CPA may occur, a CPA lesion does not necessarily signify metastasis, since benign CPA lesions including acoustic neurinomas and meningiomas may be much more common than metastatic lesions. This is true even in known cancer patients (2). Schoenberg et al (8) reported that the incidence of meningiomas in patients with breast cancer is higher than in patients without malignancy.

The clinical presentations of benign primary CPA lesions are usually distinctly different from those of metastases, and are therefore an essential factor in the differential diagnosis. Acoustic neurinomas or meningiomas are typically slow growing. They are frequently asymptomatic until they achieve a fairly large size (1, 9). In general, benign CPA tumors are frequently associated with unilateral symptoms of 8th cranial nerve dysfunction alone, such as hearing loss, tinnitus, or vertigo. Isolated cases of bilateral 8th nerve and/or other cranial nerve symptoms are rare (9), and usually occur in patients with neurofibromatosis type 2 (NF2). The average age of NF2 patients with bilateral acoustic neurinomas is less than 30 years (9). The average age in our series was 50.9 years. Facial paralysis or hemifacial spasm due to 7th cranial nerve dysfunction is rarely seen as the presenting symptom in benign CPA tumors, except those facial neurinomas that involve the IAC, and only in advanced lesions. The 5th cranial nerve lies more superiorly and medially in the CPA, and, therefore, a large CPA mass may occasionally produce trigeminal nerve symptoms such as pain, numbness, or paresthesia of the face (1).

Despite the small size of the CPA metastasis, the symptoms were fulminant and rapidly progressive in most of our patients (78.6%). The three exceptions were the patient with prostate carcinoma (consistent with the slow-growing nature of this tumor); the patient with ocular melanoma (Fig. 3), who responded favorably to melatonin treatment; and the patient with carcinoma of the nasopharynx (Fig. 5), who had no evidence of systemic hematogenous spread of tumor, but had MR evidence of retrograde contiguous spread along the trigeminal nerve to Meckel's cave and leptomeninges (which may progress more slowly than that of direct hematogenous spread).

As expected with the location of the lesions, symptoms of 8th cranial nerve dysfunction (71.4%) were the most common clinical finding in our series, followed by 7th cranial nerve (42.9%) and 5th cranial nerve (28.6%) dysfunction. However, in our series, 25% of the metastatic lesions in the IAC were asymptomatic. This proportion is probably related to the improved detection rate and the relatively small size of the lesions and/or early stage of the involvement at the time of detection. For example, one patient presented with only unilateral 5th cranial nerve symptoms, despite MR evidence of multiple cranial nerve involvement, including bilateral 5th, 7th, and 8th cranial nerve involvement.

There is limited information about the radiologic findings in the few available reports on CPA metastases (3, 4, 6, 7). CT findings in CPA metastases have been reported to be similar to those in acoustic neurinomas (3, 4, 7). MR studies of CPA metastasis are even more scarce. In the single case report of MR findings in CPA metastasis (6), only T1-weighted images were shown to demonstrate a small and isointense IAC lesion, and no details on specific MR findings or contrast administration were given.

Because acoustic neurinomas account for 80%-90% of CPA tumors, an understanding of their MR features could be useful in differentiating them from CPA metastases. On MR imaging, acoustic neurinomas are typically unilateral and show high signal intensity on T2 weighting. When symptomatic, most (>88%) are medium to large in size (1). Unilateral involvement of both the 7th and 8th nerves is uncommon in acoustic neurinomas. Multiple or bilateral cranial nerve involvement is rare (<3.8%) and usually occurs in patients with NF2 (9).

In contrast, typically metastatic CPA lesions demonstrated by MR in our series were relatively small isointense lesions, and associated with extensive and rapidly progressive neurologic symptoms. There was consistently more extensive MR evidence of cranial nerve involvement than was suspected clinically. The frequent multiple cranial nerve and/or bilateral involvement is again not a characteristic finding of isolated acoustic neurinomas. MR evidence of both 7th and 8th unilateral cranial nerve involvement associated with a relatively small lesion is uncommon in benign

CPA tumors. Because CPA metastases are often small and isointense on T1- and T2-weighted images, and are therefore frequently missed, contrast MR is essential.

The exact mechanism of spread of the malignancy to the CPA is not certain. MR studies demonstrated multiple cranial nerve involvement in all of our patients, and diffuse leptomeningeal disease in all but one patient. We have seen two patients (patients 1 and 6) with multiple cranial nerve involvement limited to the ipsilateral side where the contiguous spread of tumor occurred (Fig. 5). These findings suggest that direct hematogenous spread to both the CPA and multiple cranial nerves may not be the most frequent mechanism to account for the MR findings in our patients. Instead, a direct leptomeningeal involvement and/or dissemination through the CSF may be a more likely mechanism for metastatic CPA involvement. These hypotheses are further supported by the MR findings of contiguous leptomeningeal spread through Meckel's cave along the trigeminal nerve in two of our 14 patients (Figs. 4 and 5). One of the two had no other systemic disease to suggest hematogenous spread of metastatic disease. Therefore, contiguous extension of leptomeningeal disease and/or secondary CSF seeding to the pial surface of various cranial nerves and the CPA may be an important pathway for CPA metastasis.

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