The Journal of Laryngology and Otology
April 1996, Vol. **110**, pp. 370–372

Cerebellopontine angle metastasis from clear-cell renal carcinoma presenting as bleeding from the ear

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Abstract
Metastatic lesions in the cerebellopontine angle are rare. We encountered one such metastatic lesion from clear-cell renal carcinoma that had a striking clinical appearance, bleeding from the ear and multiple lower cranial nerves' involvement. While the overall prognosis in CNS metastasis from systemic malignancy is gloomy, useful palliation can be achieved in patients with solitary lesions.

Key words: Kidney neoplasms; Renal cell carcinoma; Neoplasm metastasis; Cerebellopontine angle; Temporal bone

Introduction

The majority of cerebellopontine angle (CPA) tumours are vestibular schwannomas. Other lesions of the CPA include meningioma, epidermoid, arachnoid cyst and facial nerve schwannoma. Rare tumours of the CPA include medulloplastoma, choroid plexus papilloma, and primary CNS lymphoma. Metastatic tumours presenting as CPA masses are extremely rare. We recently managed one such patient who had a metastatic CPA tumour from a renal cell carcinoma, and presented with bleeding from the ear, and a mass in the CPA.

Case report

A 65-year-old farmer was admitted in January 1995, with a two-month history of blood-stained discharge from his left ear. For 15 days prior to admission, his left lower limb felt unsteady. He did not have headache, vomiting, giddiness, dysphonia nor dysphagia. A year previously, he had developed left loin pain and haematuria for which he was investigated at a district hospital. A left nephrectomy had been performed and histological sections made from the kidney had revealed renal cell carcinoma. He did not receive any further treatment until he developed bleeding from the ear.

Clinical examination revealed a well built and nourished patient. There was no anaemia or lymphadenopathy. Abdominal examination revealed a well healed lumbar scar from the left nephrectomy. Examination of the left ear showed a reddish, fleshy, vascular mass with black crusting protruding out of the external auditory meatus (Figure 1). The mass was non-tender and bled to touch. There was a diffuse, non-tender, pulsatile swelling over the mastoid and retromastoid region; pulsations diminished on occlusion of the left common carotid artery.

Neurological evaluation revealed left-sided corneal hypoaesthesia, infranuclear facial paresis, sensorineural deafness, absent gag reflex, vocal cord paresis and tongue wasting. He also had left-sided intention tremor, dysmetria and dysdiadochokinesia.

Skull radiography (basal view) showed erosion of the petrous bone and mastoid. Cranial CT scan revealed a large hyperdense, contrast-enhancing lesion occupying the left CPA and eroding the petrous bone and mastoid (Figures 2 and 3). The lesion extended into the external ear laterally and displaced the cerebellum medially. There was no hydrocephalus.

Fine needle aspiration cytology (FNAC) of the lesion

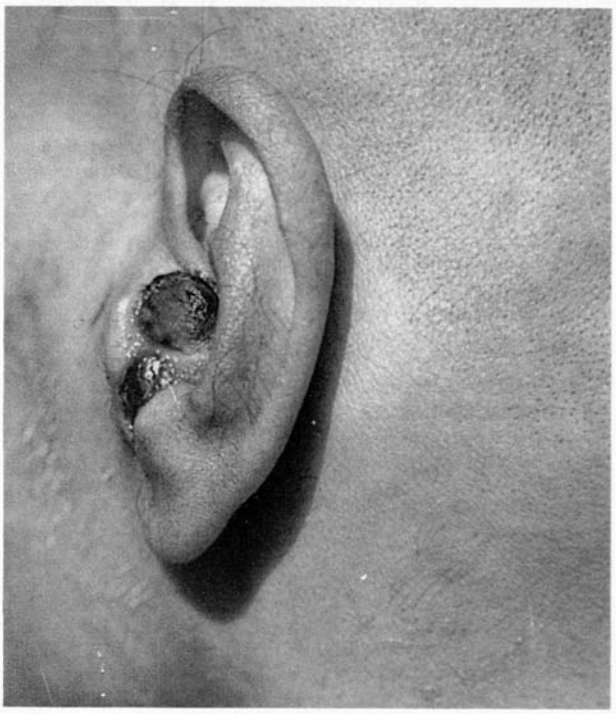

Fig. 1
Clinical appearance of the mass protruding out of the left ear with diffuse retroauricular swelling.

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Accepted for publication: 1 January 1996.

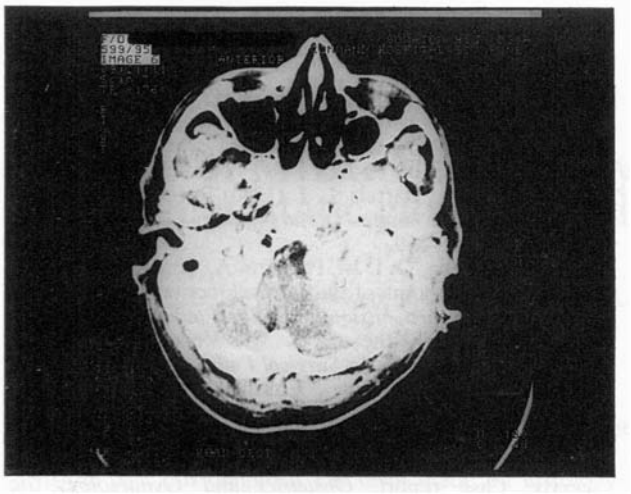

FIG. 2

CT showing contrast-enhancing left CPA mass, extending into external auditory canal.

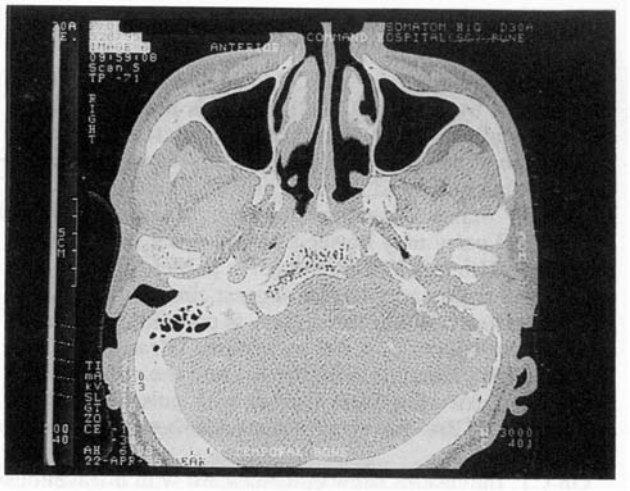

FIG. 3

CT (bone window setting) showing erosion of left petrous bone and soft tissue mass in the external auditory canal.

extruding through the ear was inconclusive, so thus it was decided to excise the mass under anaesthesia. The patient was operated upon in the lateral position. The mass was exposed by a retromastoid incision extending into the neck. The mass had eroded the bone and was lying under the scalp. It was firm, vascular, well-defined and occupied the entire CPA, displacing the cerebellum medially. Partial, piecemeal excision (about 50–60 per cent) could be achieved. In the post-operative period, he was given corticosteroids and antibiotics. Recovery was uneventful.

Histopathology from the tumour tissue (thin sections of 4 μm, stained with haematoxylin and eosin) (Figures 4 and 5) showed tumour cells which were arranged in solid sheets. The cells were polygonal in shape with abundant clear cytoplasm. Nuclei were round to oval, hyperchromatic and showed moderate pleomorphism. Occasional atypical mitotic figures were present. There were no areas of haemorrhage or necrosis. A diagnosis of metastatic clear-cell carcinoma was made, the primary site being kidney.

The patient was given corticosteroids for about two weeks. Subsequently, the posterior fossa was irradiated (4000 rads in 22 fractions over four weeks). He is well so far nearly 10 months after surgery.

Discussion

The rarity of a solitary metastatic lesion presenting in the CPA can be gauged from a statement by Martuza (1985) that a combination of a prior malignant lesion and a secondarily discovered mass should not presume the notion of a metastatis to the CPA. Even in women with breast cancer, the CPA lesion is more likely to be a meningioma (Donelan, 1992). Contrasting views, however, have been expressed that CPA metastases are more common than recognized (Nelson and Dolan, 1991) and that most of these tumours are microscopic, and thus asymptomatic (Belal, 1985). Nevertheless, on review of the literature it appears that solitary metastasis to the CPA presenting as a CPA mass is indeed a rare event.

The primary site of malignancy in patients with a CPA metastasis is often lung (Nelson and Dolan, 1991), prostate (Flickinger *et al.*, 1989), lymphoma (Nakada *et al.*, 1983) or malignant fibrous xanthoma (Jahrsdoerfer *et al.*, 1976). Metastatic melanoma presenting as a CPA lesion has been reported and is noted to have a special predilection for the internal meatus (Berman *et al.*, 1979; Braga *et al*, 1989). Clear-cell adenocarcinoma (diethylstilboestrol-related) of the cervix and vagina has been reported to metastasize to the cerebellum and CPA after a long delay (Burks *et al.*, 1990). While renal cell carcinoma, too, is curiously prone to late dissemination (Stortbecker, 1954; Willis, 1971), and the lesion is usually solitary (Burger *et al.*, 1991), it has a special predilection for infratentorial metastases (Posner and Chernik, 1978; Russel and Rubinstein, 1989). Metastases to the CPA may also occur from an unknown primary adenocarcinoma (Moloy *et al.*, 1989; Kumar *et al.*, 1990).

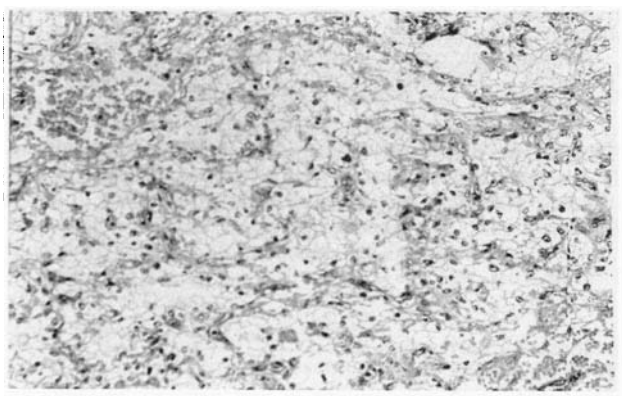

FIG. 4

Histopathology (low magnification) showing sheets of neoplastic cells (H & E; × 50).

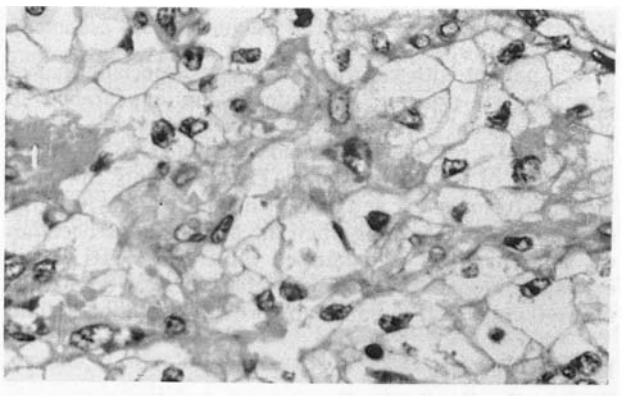

FIG. 5

Histopathology (high magnification) showing clear-cell tumour (H & E; × 300)

https://doi.org/10.1017/S0022215100133651 Published online by Cambridge University Press

Tumour emboli reach the CPA by the haematogenous route (Hill and Kohut, 1976).

Clinical presentation in CPA metastases has been summarized by Lalwani (1992). There is usually an acute onset of audiovestibular symptoms associated with lower cranial nerve involvement. Clinical features include headache, postauricular and mastoid pain and hearing loss and cerebellar ataxia. Jugular foramen involvement and hypoglossal palsy may be seen. Rapid onset of symptoms including VIIth to XIIth cranial nerve palsies together with a history of malignancy should suggest clinical diagnosis of CPA metastasis. While facial palsy is common and occurs early in the course of CPA metastases (Brackmann and Bartels, 1980), bleeding from the ear and the presence of a fleshy mass extruding from the ear with pulsatile mastoid swelling in CPA metastases has not been previously described.

On CT, the lesions show enhancement with intravenous contrast. Rarely, a metastatic tumour may erode the internal acoustic meatus, mimicking vestibular schwannoma (Kendall and Symon, 1977). When the petrous bone is involved, the changes may be osteolytic or osteoblastic (Lalwani, 1992). On MRI, their appearances are variable, with a hypointense signal on T1-and hypo- or hyperintense signal on T2-weighted scans. Brain—tumour interface may appear indistinct. The lesion may enhance with gadolinium.

There is no satisfactory treatment for metastatic adenocarcinoma of the kidney (Stage IV or M+) (Bagley, 1991). In selected patients, in whom there is a proven solitary metastatic lesion, surgical resection may provide long-term disease-free survival. Surgical resection can be achieved by a suboccipital or translabyrinthine approach (depending on the extent of hearing loss) for diagnosis and therapy (Brackmann and Bartels, 1980). Survival is better after resection of a metastasis which presents after nephrectomy than in patients presenting with synchronous primary tumour and metastasis (Bagley, 1991). Complete tumour removal is often impossible and post-operative chemo- and radiotherapy are usually prescribed. When combined with the use of corticosteroids, radiotherapy has been shown to extend survival of a significant number of patients with brain metastases. It must, however, be remembered that the response of metastatic tumours to radiation is predictably correlated with known sensitivity of various neoplasms to radiation, and benefit for renal cell carcinoma metastases should not be expected to be high (Galicich and Arbit, 1990). Vinblastine is a useful single agent in metastatic renal cell carcinoma with an initial response rate of about 24 per cent (Bagley, 1991). Although the prognosis of metastatic CPA tumours is poor, survival for as long as four years has been reported (Brackmann and Bartels, 1980).

Conclusion

In a patient with known malignancy, especially that of lung, breast, prostate, or kidney, the appearance of multiple cranial nerve palsies especially of lower cranial nerves, and cerebellar signs should alert the clinician to the possibility of CPA metastases. These patients can have bleeding from the ear and a fleshy mass extruding from the ear and mastoid swelling when the petrous bone becomes eroded. Diagnosis is confirmed on CT or MRI. After excluding secondaries at other sites, surgical debulking, chemotherapy and radiotherapy provide useful palliation.

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