

## Metastatic bronchogenic adenocarcinoma inside a pituitary adenoma: case report and literature review

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### Summary

A 70-year-old man with a metastasizing bronchogenic adenocarcinoma inside a pituitary adenoma is presented. Preoperative MRI findings of the lesion were suggestive in view of the peculiar dishomogeneous appearance of the tumour. A review of the literature revealed only ten previous cases to which we add our personal one. The salient clinico-pathological and diagnostic features of this rare lesion are discussed in the light of the available literature.

**Key words:** metastatic adenocarcinoma, neoplasm-to-neoplasm metastasis, pituitary adenoma, trans-sphenoidal surgery

### Introduction

Metastasis to the pituitary gland is rarely recognized ante-mortem and it is an uncommon but well-described clinical entity (Cushing, 1913; Piney and Coates, 1924; Abrams, Spiro and Goldstein, 1950; Gurling, Scott and Baron, 1957; Halpert, Erickson and Fields, 1960; Johnson and Atkins, 1965; Duchon, 1966; Hagerstrand and Schonebeck, 1969; Houck, Olson and Horton, 1970; Roessmann, Kaufmann and Riede, 1970; Willis, 1971; Burns *et al.*, 1973; Kovacs, 1973; Kistler and Pribram, 1975; Teeares and Silverman, 1975; Sanchez, 1977; Cox, 1979; Poon *et al.*, 1979; Yap, 1979; Leramo *et al.*, 1981; Max, Deck and Rottenberg, 1981; Bynke and Ottosson, 1982; Buonaguidi *et al.*, 1983; Case records of MGH, 1983; Eick *et al.*, 1985).

While reports of an extracranial tumour metastasizing inside an intracranial neoplasm are very rare and mostly regard meningiomas (Helpap, 1965; Wolintz and Mastro, 1970; Rubinstein, 1972; Gyori, 1976; Chambers *et al.*, 1980; Savoiaro and Lodri-

### Riassunto

Viene presentato un caso di adenocarcinoma broncogeno metastatico in un adenoma ipofisario. La RMN cerebrale preoperatoria evidenziava l'aspetto caratteristicamente dishomogeneo del tumore. Una revisione della letteratura ha permesso di individuare soltanto dieci casi analoghi ai quali va aggiunto il caso da noi descritto. I principali aspetti clinico-patologici e diagnostici di questa rara lesione sono discussi alla luce della letteratura.

**Parole chiave:** adenocarcinoma metastatico, metastasi intracraniale, adenoma ipofisario, chirurgia trans-sfenoidale

ni, 1980; Lodrini and Savoiaro, 1981; Bernstein, Grumet and Wetzel, 1983; Schmitt, 1984; Doron and Gruszkiewicz, 1987; Russell and Rubinstein, 1989; Zon *et al.*, 1989; Bucciero *et al.*, 1992; Cervoni *et al.*, 1994) and, less frequently, acoustic neuromas (Wong and Bennington, 1962; Chambers *et al.*, 1980; Schmitt, 1984) and gliomas (Schmitt, 1984), metastasis inside a pituitary adenoma is quite exceptional. In the literature we were able to find only ten cases of this particular lesion (Richardson *et al.*, 1971; Van der Zwan *et al.*, 1971; Burns *et al.*, 1973; Kovacs, 1973; James *et al.*, 1984; Molinatti *et al.*, 1985; Van Seters *et al.*, 1985; Zager *et al.*, 1987; Post *et al.*, 1988). We report an additional case of this unusual lesion analyzing its clinical and diagnostic features in the light of the literature.

### Case report

A 70-year-old man came to our attention for progressive severe visual impairment in the left eye associated with blurring in the right one. The visual field examination documented a complete amaurosis in the left eye and a right superior temporal quadrantanopia. Cranial MRI showed a large supra-intra-sellar lesion (figs. 1 and 2a, b) hyperintense after gadolinium administration on T<sub>1</sub>-weighted imaging (SE and SR sequences), with a central hypointense area: it invaded the sphenoidal sinus and extended bilaterally in the

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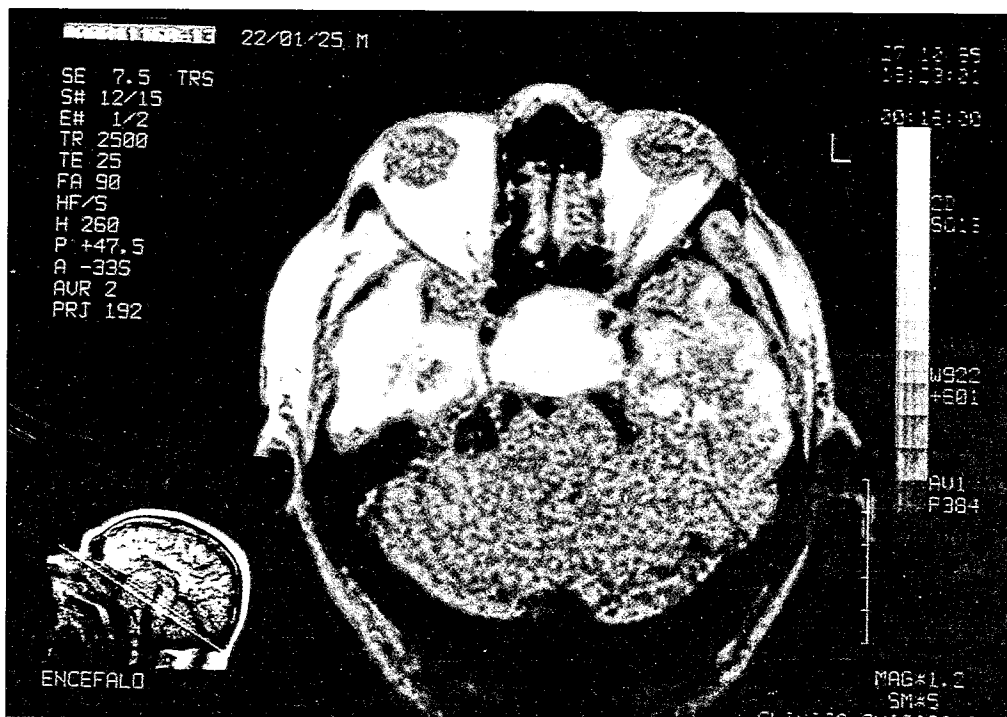
cavernous sinuses. A preoperative chest X-ray showed a nodular opacity in the superior right pulmonary field. This finding was confirmed by a subsequent chest CT-scan which also documented invasion of the right supraclavicular lymph nodes. Through a trans-naso-sphenoidal approach a subtotal removal of the lesion was performed. A contemporary intraoperative biopsy of a supraclavicular lymph node was accomplished. Histological examination revealed areas of pituitary gonadotropic cell adenoma mixed with a pattern of metastatic adenocarcinoma (fig. 3a, b). Supraclavicular lymph node specimen confirmed the diagnosis of metastatic mucinous adenocarcinoma. The patient underwent radio- and chemotherapy since a radical removal of the primitive tumour was not feasible. At a six-month follow-up, the patient was alive without further neurological disturbances and visual function was unmodified. After 20 months the patient died owing to spreading of metastatic disease with multiple dissemination in the liver and brain.

**Discussion**

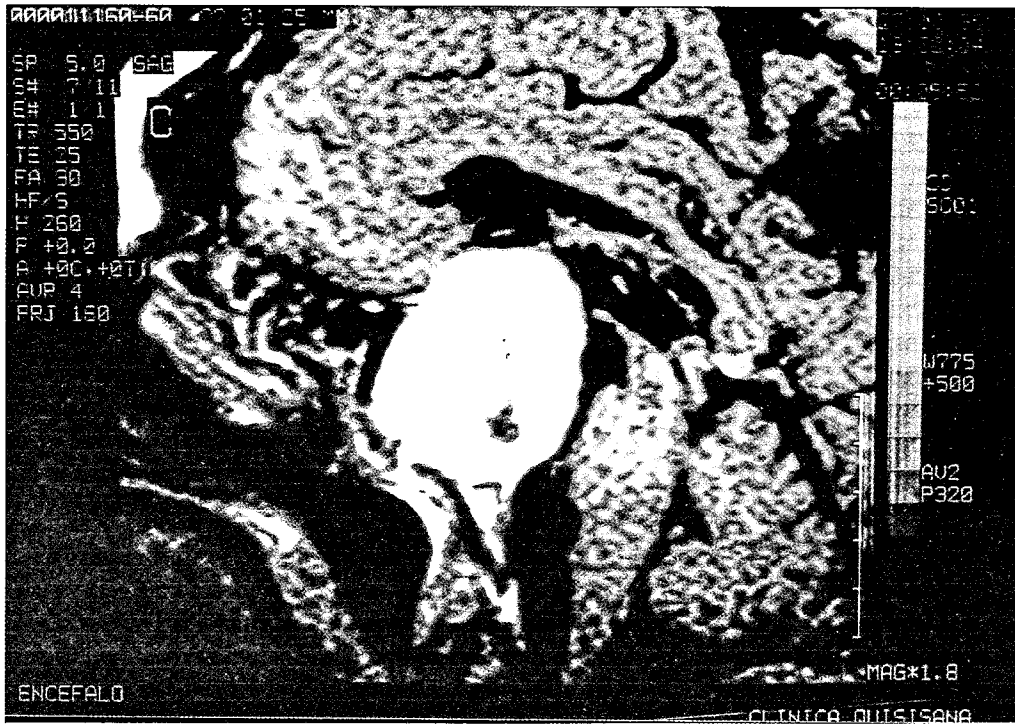
Neoplasm-to-neoplasm metastasis is a rare but well-recognized occurrence. Very few cases involving a pituitary adenoma have been reported (Richardson and Katayama, 1971; Van der Zwan, Luyendijk e Bots, 1971; Burns and Kadar, 1973; Kovacs, 1973; James et al., 1984; Molinatti et al., 1985; Van Seter et al., 1985; Zager and Hedley-Whyte, 1987; Post et al., 1988). Extracranial malignant tumours metastasizing inside intracranial neoplasms have, in fact, a predilection for meningiomas and, less frequently, for acoustic neurinomas or gliomas (Wong et al., 1962; Helpap, 1965; Zülch, 1965; Wolintz and Matri, 1970; Rubinstein, 1972; Gyori, 1976; Chambers et al., 1980; Savoiaro and Lodrini, 1980, 1981; Bernstein, Grumet and Wetzal, 1983; Schmitt, 1984; Doron and Gruszkiewicz, 1987; Russell and Rubinstein, 1989; Zon et al., 1989; Bucciero et al., 1992; Cervoni et al., 1994).

To date only ten cases of metastasis inside a pituitary adenoma have been reported in the available world literature. Some observations (Schmitt, 1984) suggest that “despite its rare occurrence,

the metastatic spread to intracranial tumours is not really surprising and probably not merely coincidental since the host tumours usually belong to richly vascularized types. Intense vascularization probably provides an increased chance of circulating cancer cells being caught and forming metastases”. In fact the vascular network may act as a filter to metastatic emboli from the bloodstream (Lodrini et al., 1981; Doron et al., 1987; Zon et al., 1989). The importance of this factor seems to be confirmed by the fact that the presence of intratumoral metastases has also been reported in other highly vascular tumours, such as renal carcinoma (Bernstein et al., 1983; Cervoni et al., 1994). This finding is consistent with the reports which reveal that the host tumours were predominantly types with a rich blood supply (Bernstein et al., 1983; Schmitt, 1984), a condition that seems to be essential for development of these “collision tumours”, as they were named by Foulds in 1940. On the other hand, not only the particular vascularization of the tumour can be claimed to be responsible for the development of these unusual lesions. In fact, as reported by Bernstein et al. (1983), “the usual hypervascularity of meningiomas has been considered a factor for hematogenous spread of metastasis, although the preponderance of occurrence of meningiomas over the more vascular intracranial host tumours would suggest that a rich blood supply and ease of expansile growth are not the major factors in the capacity of a neoplasm to accept metastasis. There does appear to be a propensity for adenocarcinoma rather than other types of cancer to metastasize to primary brain tumours”. The same authors underline that a close biological affinity between adenocarcinoma and meningeal tissue has been described: they also point out that there is no satisfactory explanation for the ability of some neoplasms to accept or reject metastasis. According to other authors (Willis, 1971) carcinoma of the breast mainly shows a predilection to metastasize inside the pituitary gland, followed by bronchogenic carcinoma. Moreover, a specific cell-to-cell in-



**Fig. 1** - Axial MRI shows a large sellar lesion extending bilaterally into the cavernous sinuses. The contiguity of the mass with the internal carotid artery on both sides is also visible.



**Fig. 2a** - Sagittal MRI shows a large sellar tumour (pituitary adenoma), extended out of the sella turcica into the sphenoidal sinus compressing the 3rd ventricle and containing a central hypointense area attributable to the metastatic bronchogenic tissue located within the pituitary adenoma.



**Fig. 2b** - Coronal MRI shows a large sellar tumour (pituitary adenoma), extended out of the sella turcica into the sphenoidal sinus compressing the 3rd ventricle and containing a central hypointense area attributable to the metastatic bronchogenic tissue located within the pituitary adenoma.

teraction may favour the implantation of metastases in the host tumour, Gyory (1976) and Bernstein *et al.* (1983), for example, believed that the scarcity of psammoma bodies in some meningiomas might induce the formation of metastatic deposits because these psammoma bodies conferred some degree of protection from implants. Some observations also appear to be significantly suggestive of the existence of a definite correlation between certain tumour tissues. Breast cancer and meningioma are, for exam-

ple, marked by some features that cannot be merely coincidental (Bucciero *et al.*, 1992). Both tumours may express estrogen and progesterone receptors, and would appear to be hormone-dependent for their development and growth (Burns *et al.*, 1973; Knuckley, Stoll and Epstein, 1989; Zon *et al.*, 1989; Bucciero *et al.*, 1992); moreover, overexpression of the *c-myc* oncogene has been reported in both neoplasms (Chambers *et al.*, 1980; Kozbar and Croce, 1984; Kuzumoto, Tamura and Hoshino, 1990). The pres-

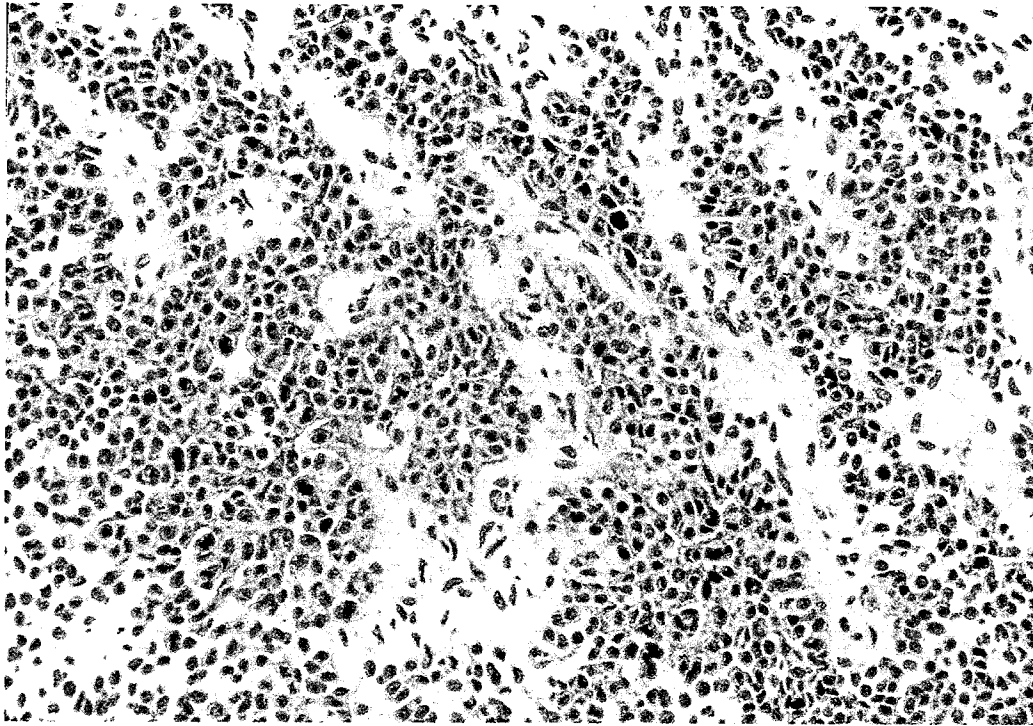


Fig. 3a - Typical pattern of gonadotropic cell adenoma. E.-E. x 200.



Fig. 3b - Cytokeratin stain showing positive immunoreactivity in adenocarcinoma cells with pituitary adenoma x 300.

ence of the c-myc oncogene in both tumours may be related to the estrogen-induced proliferation of breast carcinoma cells (Dubik, Dembinski and Shiu, 1987) or to the amplification of the same oncogene that may induce meningiomas through loss of the putative tumour suppression gene (Kuzumoto *et al.*, 1990). Other observations (Magdelenat *et al.*, 1982; Martuza, Miller and Maclaughlin, 1985) underline the presence of greater levels of progesterin and estrogen receptors as well as an increased incidence

of these receptors in postmenopausal women who have breast carcinoma. The interaction between the cells of meningioma and breast carcinoma could induce the presence of the above mentioned receptors in both of them (Bucciero *et al.*, 1992; Cervoni *et al.*, 1994). Similar interactions could be postulated to explain the affinity of carcinomas for pituitary adenomas.

In general, the incidence of intrasellar metastases ranges from 1.1 to 26.1 with an average of between 1% e 4% (Abrams *et al.*, 1950;

Delarue *et al.*, 1964; Johnson *et al.*, 1965; Leramo *et al.*, 1981; Barz, 1983; Atlas, Grossman and Gomori, 1987; Branch *et al.*, 1987; Knuckley *et al.*, 1989; Chiang, Brock and Patt, 1990; Cervoni *et al.*, 1994). These patients were aged between 40 and 80 with an average age of 50. They usually present more than one tumoral localization and are often terminally ill (Abrams *et al.*, 1950; Houck *et al.*, 1970; Roessmann *et al.*, 1970; Kovacs, 1973; Teears *et al.*, 1975; Leramo *et al.*, 1981; Max *et al.*, 1981; Buonaguidi *et al.*, 1983; Eick *et al.*, 1985; Branch *et al.*, 1987; Nelson *et al.*, 1987).

Intrasellar metastases may arise from many organs (Abrams *et al.*, 1950; Johnson *et al.*, 1965; Duchon, 1966; Hagerstrand *et al.*, 1969; Roessmann *et al.*, 1970; Kovacs, 1973; Kistler *et al.*, 1975; Teears *et al.*, 1975; Cox, 1979; Leramo *et al.*, 1981; Max *et al.*, 1981; Buonaguidi *et al.*, 1983; James *et al.*, 1984; Eick *et al.*, 1985; Branch *et al.*, 1987; Nelson *et al.*, 1987; Zager *et al.*, 1987; Post *et al.*, 1988; Chiang, Brock and Patt, 1990; Stalldecker *et al.*, 1994). The most frequent primary tumour in females is infiltrating ductal carcinoma of the breast followed by pulmonary, gastric and thyroid carcinoma. In males, bronchogenic carcinoma is more frequent than that of the prostate, bladder, pancreas, kidney and colon. Localization of a carcinomatous metastasis in a pituitary adenoma is indeed an extremely rare phenomenon, as demonstrated by the fact that only 10 cases have been reported in world literature. The presence of a metastasis from carcinoma within a pituitary adenoma was described for the first time in 1971, in 2 separate reports: although in both cases a ductal carcinoma metastasized into a pituitary adenoma, in the case described by Richardson and Katayama (1971) the latter was of the acidophylic type while in the case described by Van der Zwan *et al.* (1971) it was chromophobic. In 1973, Burns and Kadar described a case of renal carcinoma metastasizing inside a chromophobic adenoma. In the same year, Kovacs (1973) described a case of bronchogenic carcinoma infiltrating a chromophobic-cell adenoma. In 1984 James *et al.* reported a case of metastasis from renal carcinoma inside a pituitary chromophobic adenoma. In 1985 Molinatti *et al.* described a case of pulmonary carcinoma metastasizing within a pituitary adenoma which caused homolateral paresis of the III cranial nerve. In 1985, Van Seter *et al.* (1985) described another case of occult gastric carcinoma metastasizing within a pituitary adenoma. Zager and Hedley-White (1987) described a case of plurimetastatic ductal carcinoma in a patient who presented rapid deterioration of eyesight: post-mortem findings revealed a pituitary adenoma within which there were small, isolated areas of metastatic carcinoma. Lastly, Post *et al.* (1988) described 2 cases of metastatic carcinoma within a pituitary adenoma. It is worth mentioning that while the onset symptoms are not different from those encountered in other sellar neoplasms histological and, sometimes, radiological data are interesting. As Zager and Hedley-White (1987) point out in their case report, no tumour cells are seen in the normal pituitary gland, thus confirming the predilection of the carcinomatous cells for the adenomatous tissue. Furthermore, as the authors themselves confirm, radiological features of pituitary metastases and adenomas are not very different: ballooning of the sella, double density sellar floor, bony erosion and soft tissue invasion may be observed in both types of lesions. However, as pointed out by some authors (Lodrini *et al.*, 1981; Bucciero *et al.*, 1992) and observed in our own case, in cases of metastasis within a tumour the presence of a hyperdense or hyperintense lesion (visible on CT and MR imaging after contrast medium injection and related to adenoma or meningioma) containing one or more hypodense areas (attributable to the metastatic intratumoral implants) could suggest a picture of adenoma or meningioma containing metastatic cancer areas.

These unusual CT and MRI findings should be regarded with particular attention and the possibility of a tumour-to-tumour phenomenon should be kept in mind (Lodrini *et al.*, 1981; Doron *et al.*, 1987; Bucciero *et al.*, 1992).

During the last few years, the investigation usually chosen for cerebral metastases and for those of the skull base in particular has been MRI: the optimal method for diagnosing intracranial metastases seems to be the use of T1-weighted sequences and administration of gadolinium DTPA (Molinatti *et al.*, 1985; Atlas *et al.*, 1987; Wittenberg, Stark and Forman, 1988; Reining *et al.*, 1989; Outwater *et al.*, 1991; Suzuki *et al.*, 1993; Stalldecker *et al.*, 1994). In our case, T1-weighted images with gadolinium enhancement documented the presence of an intra-suprasellar lesion extending from the floor of the sella as far as the III ventricle, which appeared compressed in its anterior-inferior portion (fig. 2), and then invaded the cavernous sinus bilaterally (fig. 1). In sagittal and coronal scans a central area compatible with necrotic phenomena on the basis of signal intensity (fig. 2 a, b) was clearly visible and could be attributed to the bronchogenic metastatic deposit within the pituitary adenoma.

The phenomenon responsible for a carcinoma metastasizing inside a pituitary adenoma is still a rare event and many aspects of its biological significance are still uncertain, requiring further clinical and experimental studies. Improvement of ultrastructural and receptorial investigations could probably supply further indications by identifying particular features of the tumour cells responsible for this phenomenon.

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