Granular cell tumor of the neurohypophysis: Case report and review of the literature

Leonard Saiegh 1, Majed Odeh 2, Mohammad Sheikh-Ahmad 1, Maria Reut 1, Zvi Ram 3, Carmela Shechner 1

1 Endocrinology Department, Bnai-Zion Medical Center, Haifa, Israel
2 Department of Internal Medicine A, Bnai-Zion Medical Center, Haifa, Israel
3 Department of Neurosurgery, Tel Aviv Medical Center, Tel Aviv, Israel

Correspondence to: Leonard Saiegh, MD.
Endocrinology Department,
Bnai-Zion Medical Center, Haifa, Israel.
tel: +97248359510; fax: +97248359519; e-mail: leonard.saiegh@gmail.com

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Abstract

A 54-year-old woman presented with a stalk mass that was discovered incidentally with mild visual fields defect. The mass was operated surgically by the fronto-temporal approach, and histology met the diagnosis of neurohypohesial granular cell tumor (GCT). After surgery, the patient suffered from an irreversible severe bi-temporal visual deficit and an irreversible hypopituitarism. We review the literature and discuss the clinical nature of GCTs, treatment options and outcome. In an effort to avoid the severe complications that may result from surgical removal of neurohypohesial GCT, we discuss also the possibility of choosing the conservative approach with close follow-up. The tumor's firm consistency, tendency to hemorrhage, involving the pituitary stalk and lack of dissection plane from basal brain structure render surgery difficult, and maximal resection often requires sacrificing the stalk. Moreover, small asymptomatic neurohypohesial GCTs are common findings, most probably benign tumors with slow growing nature. Hence, for a neurohypohesial tumor which is suspected to be a GCT, we offer to consider the alternative approach, with close clinical, visual field and radiological study follow up.

INTRODUCTION

Granular cell tumors (GCTs) may occur in any anatomic site in the body, but they have a rare occurrence in the sellar region (Rickert et al. 1997). Yet, they have been incidentally reported in 6.5% to 17% of autopsies examining posterior pituitary and stalk (Schancklin 1947; 1953). The majority of reported cases have revealed a benign behavior of the tumor with malignancy being extremely rare (Shuangshoti et al. 1998). Nonetheless, there are few papers that reported large tumors that led to the emergence of symptoms (Satyamurti & Huntington 1972). Depending on the size of the lesion, the trans-sphenoidal or trans-cranial approach is selected in order to remove GCTs (Alleyne et al. 2002). Due to the firm consistency of the tumor and to its high vascularity, sometimes only subtotal removal of the tumor is possible (Schaller et al. 1998).

We present here a case report of a woman with a stalk mass that was discovered incidentally with a minimal visual field deficit. The mass was operated surgically by the fronto-temporal approach, and histology met the diagnosis of GCT. After surgery, the patient suffered from an irreversible severe bi-temporal visual deficit and an irreversible hypopituitarism.
In this paper, we review the literature and discuss the clinical nature of GCTs regarding epidemiology, etiology, clinical presentation, radiology, differential diagnosis and histology. Given the firm consistency and high vascularity of GCTs, involvement of the stalk, and given their benign course, we also discuss indications for surgery. In an effort to avoid the severe complications that may result from total surgical removal of these tumors, we discuss the possibility of choosing only partial resection of the tumor, or alternatively just conservative treatment with close follow-up.

CASE REPORT

A 54-year-old woman presented complaining of headache and nausea. A week before her presentation she had had a serious head trauma with post-traumatic syncope. On presentation, other than peri-orbital hematoma, her physical examination was unremarkable. On computed tomography (CT) scan she was suspected to have harbored a suprasellar mass, and CT angiography ruled out a vascular mass. Magnetic resonance imaging (MRI) revealed a well defined suprasellar mass, adjacent to cerebral third ventricular floor behind optic chiasm, with suspected mild compression of the chiasm. The tumor measured 16x16x16 mm, was isodense on T1-weighted images, with nonhomogeneous gadolinium enhancement, and it was hypodense on T2-weighted images. The mass did not involve the pituitary gland or the cavernous sinuses, and as appeared by MRI, was suspected to originate from the third ventricular floor, hypothalamus or pituitary infundibulum (Figure 1).

The patient did not complain of headache before the trauma, neither did she report any changes in her visual acuity or visual loss. She did not have a clinical history of any kind of pituitary dysfunction or diabetes insipidus. A total body CT scan did not reveal any suspected malignancy; laboratory tests for carcinoembryonic antigen, carbohydrate antigen 19-9, carbohydrate antigen 125, carcinoma antigen 15-3, β₂ microglobulin, alpha-fetoprotein and β-human chorionic gonadotropin were all negative, and the patient refused to undergo a lumbar puncture test. Endocrinological laboratory tests revealed normal basal cortisol and free thyroxin (FT4) levels, normal adrenocorticotropic hormone stimulation test and postmenopausal elevated follicle-stimulating hormone. A slightly high prolactin level of 22.9 ng/ml was observed (normal levels lower than 20 ng/ml). At presentation, visual field test showed a minimal defect in the superior temporal field of the left eye, with minimal deterioration a month later (Figure 2).

The tumor was removed through the fronto-temporal approach; a mass was identified attached to the lower aspect of the optic nerves and optic chiasm. The mass was not firm or vascular, but it was intimately involving the pituitary stalk. The mass was separated from the carotid arteries and nerves, no serious bleeding was encountered, and it was completely removed requiring scarifying the stalk. On histology, the tumor was composed of large cells with abundant granular cytoplasm, positive staining to S100, neuron specific enolase (NSE), CD68, alpha 1-antitrypsin and periodic acid-schiff (PAS). Staining for synaptophysin and glial fibrillary acidic protein (GFAP) was negative. MIB-1 staining was positive in less than 1% of the cells, and histology supported the diagnosis of GCT. Post-operatively, the patient suffered from polyuria and polydipsia, low basal morning cortisol levels and low FT4 levels. Therefore, treatment with corticosteroids, levothyroxin and vasopressin was initiated. Post surgery, a serious deterioration of the patient’s visual fields was observed; with development of severe bilateral temporal hemianopia (Figure 3).

Nowadays, a year and a half after surgery, the patient has normal prolactin levels, with no improvement in her adrenal and thyroid function tests. She still needs

![Fig. 1. MRI. T1-weighted pre-contrast coronal image, showing an isodense suprasellar mass (a). T1-weighted coronal post-contrast image showing a nonhomogeneous gadolinium enhanced suprasellar mass (b). T2-weighted coronal image showing a hypodense suprasellar mass (c).](image-url)
replacement therapy with levothyroxin and corticosteroids, and suffers from diabetes insipidus treated with vasopressin. Her visual fields have not witnessed any improvement, still showing complete bilateral temporal hemianopia. In addition, since surgery, the patient complains of weakness, weight gain and depression, while MRI did not show any recurrence of the tumor.

DISCUSSION

Clinically significant primary tumors of the neurohypophysis are extremely uncommon and consist essentially of two types: the first is glioma of the infundibulum and the posterior pituitary, and the second type is the GCT (Thapar & Kovacs 1998). GCTs are well-circumscribed round or globular tumors that usually compose of sheets of large polyhedral cells and have an ample cytoplasm of PAS positive granules (Menon et al. 2008). GCTs can arise in a variety of sites, and they are most commonly seen in the dermis or subcutaneous soft tissue of the head and neck (Rickert et al. 1997). These extracranial tumors have been referred to as myoblastomas, occurring commonly in skin and viscera where excision is curative (Satyamurti & Huntington 1972). Classifications of tumors of the central nervous system, including spinal meninges (Markesbery et al. 1973) and cerebral hemispheres (Dickson et al. 1986). Because of their enigmatic origin, GCTs have been called by various names, including pituicytomas, infundibulomas and choristomas (Cone et al. 1990).

Epidemiology

Symptomatic GCTs of the neurohypophysis comprise less than 0.1% of primary brain tumors, which in turn account for approximately 1.5–2% of all adult neoplasms (Wilkinson et al. 2003). GCTs of the neurohypophysis are more commonly described in female patients and usually present in the 4th and 5th decade of life (Cohen-Gadol et al. 2003). Although only approximately 50 cases of symptomatic GCTs within the neurohypophysis have been reported in the literature, as many as 91 granular cell nodules located in either the posterior hypophysis or the pituitary stalk were reported in an autopsy study of 1364 cadavers, indicating that neurohypophysial GCTs may be much more common than noted clinically (Luse & Kernohan 1955). This finding was further supported by Tomita & Gates (1999), when they evaluated 100 pituitary glands at autopsy and found that 9% of the pituitaries harbored a GCT, none of which had been suspected during the individual’s lifetime. They claimed that if the entire thickness of paraffin blocks had been examined in their study, the 9% incidence of GCTs may have been doubled. Eight of nine GCTs in their study measured 0.3 to 0.7 mm, and the largest measured 1×1.5 mm. Sano et al. (1993) showed that the incidence of GCTs was 1–2% of unselected autopsy of pituitary glands. In another study Shanklin (1947, 1953) reported a high frequency of the granular cell aggregates “tumorettes” in the posterior lobe, namely 17%, with frequent location in the proximal part of the infundibular process and stalk. But, the “tumorettes” which Shanklin described included not only granular cell islets but also pituicytic and folliculostellate cell.

Etiology

Tomita & Gates (1999), in a study of autopsies, found that the prevalence of GCTs was 9%, and in one third of
the cases it coexisted with anterior pituitary. Therefore, it was claimed that such coexistence implies a histogenetic connection between GCTs and pituitary adenomas. However, Schanklin (1947, 1953) argued that the source of GCTs are basophiles invading the infundibular process, occasionally observed at autopsy of normal brain.

GCT is histogenetically heterogeneous and therefore remains a purely descriptive entity (Thapar & Kovacs 1998). It was ascertained by a molecular-genetic examination that GCTs do not belong to a distinct tumor entity characterized by chromosomal imbalances, but are rather a degenerative phenomenon of various origins (Rickert & Paulus 2002). Other researchers have suggested that these tumors originate from pituicytes; thus, they have used the term “pituicytoma” to describe these tumors (Figarella-Branger et al. 2002; Halbauer et al. 2003). In fact, the origin of GCTs is still unknown and it may either be at the neuro-glial or mesenchymal base. The findings obtained from an analysis of the close anatomic relationship of GCTs with nerves; the ultrastructural demonstration of myelin figures, axon-like structures, basal laminae and the immunohistochemical reactivity with neuron-specific enolase and myelin proteins have all strongly suggested a neural origin or differentiation of GCTs (Armin et al. 1983; Fisher & Wechsler 1962; Fust & Custer 1948; Mazur et al. 1990; Miettinen et al. 1984; Mukai 1983; Nakazato et al. 1982; Nathrath & Remberger 1986). Moreover, the presence of calretinin further supports a neural origin or differentiation of these tumors (Fine & Li 2003). On the other hand, the cellular expression of S-100 protein in GCT cells supports the theory of a derivation from Schwann cells (Kobayashi et al. 2006). However, the presence of GFAP in GCT cells, which is normally expressed in pituicytes, is a subject of considerable debate (Salm et al. 1982), and the lack of a basement membrane surrounding the tumor cells and negative staining for Leu-7, constitute evidence against the theory that Schwann cells are the origin of the tumor (Lafitte et al. 1994; Liwnicz et al. 1984). Currently, it is accepted that GCTs outside the sellar region arise from Schwann cells, whereas in the sellar region, they derive from the pituicytes (Ji et al. 1995), but the term “pituicytoma” is currently defined as a separate entity of tumor with specific features (Brat et al. 2007). It follows that GCTs of the neurohypophysis should not be called pituicytomas, and until further evidence becomes available regarding the exact origin of this type of tumor, the purely descriptive and non-committal term “GCT of the neurohypophysis” seems to be the most appropriate nomenclature for this tumor (Nishioka et al. 1991).

Clinical presentation
The high prevalence of GCTs in autopsy studies indicates that GCTs may be much more common than noted clinically. As mentioned above, GCTs are more commonly described in female patients, and usually present in the 4th or 5th decade of life (Cohen-Gadol et al. 2003). Nonetheless, cases of GCTs in also unusual ages have been reported. Popovic et al. (2007) reported a case of a 21-year-old woman who presented with visual and headache complaints and had growth hormone deficiency, hypogonadotrophic hypogonadism and hypocortisolism. Similarly, Benito Filho et al. (2005) reported an 8-year-old boy with precocious puberty and a suprasellar mass that proved to be a GCT. Surprisingly, a rare case of a 42-year-old woman with acromegaly caused by GCT has also been reported by Losa et al. (2000), and the authors suggested that the tumor produced GH releasing hormone.

GCTs grow very slowly over a long period of time, and in the majority of cases they never reach a size large enough to produce clinical symptoms. Yet, once they become symptomatic, they seem to progress rapidly (Giangaspero & Cenacchi 1999). As with other non-functional pituitary tumors, symptoms of GCTs are related primarily to tumor size and mass effect (Huang & Castillo 2005), and no reliable presenting clinical features distinguish GCTs from other lesions in the sellar or suprasellar region (Schaller et al. 1998). Although symptoms are typically chronic and progressive, acute onset of symptoms has been noted in a few cases (Houtteville et al. 1976). Visual loss (Iglesias et al. 2000) and short-term history of confusion (Boecher-Schwarz et al. 1992) are among the symptoms that have been described. Spontaneous intratumoral hemorrhage and intraventricular hemorrhage were reported in only one case (Graziani et al. 1995), and Cone et al. (1990), and Liss & Kahn (1958) described two cases of suprasellar GCT that became symptomatic due to tumoral hemorrhage 4 and 10 years after subtotal removal and irradiation, respectively. Typically, symptomatic GCTs present with insidious visual impairment and anterior pituitary insufficiency with mild hyperprolactinemia which is, most probably, related to pituitary stalk compression (Schaller et al. 1998). Although the tumor arises from the neurohypophysis, diabetes insipidus was a presenting feature in only a few patients (Schlachter et al. 1980). In their study of 11 cases of GCTs, Cohen-Gadol et al. (2003) described the clinical presentation as follows: seven of 11 cases were symptomatic, and in 4 patients the tumor was found incidentally. The most common presenting complaint was visual field deficit, reported in 3 patients with a mean duration of 3 months. Formal visual testing was abnormal in 8 of 11 patients, revealing bitemporal hemanopsia in 6, and right-sided visual deficits in 2 patients. Two patients complained of intractable headache, and no patient presented with diabetes insipidus. Neuropsychological changes in more advanced stages of the tumor may be explained by compression of the hypothalamus (Schaller et al. 1998).

Radiology and differential diagnosis
Very few cases of GCTs of the neurohypophysis have been described in the radiology literature (Cone et al. 1990; Glazer et al. 1956; Ji et al. 1995). Other than their
most common location in the neurohypophysis and infundibulum, typical radiologic patterns supporting the diagnosis of GCTs are yet to be determined. CT scan studies of lesions that proved to be GCTs typically showed well demarcated tumors of relatively high attenuation that are isodense or slightly hyperdense compared with gray matter before administration of intravenous contrast, while moderate contrast enhancement was typical (Kudawara et al. 1999).

MRI findings in GCTs of the neurohypophysis are similar to the MRI appearance of meningiomas. Yet, it may be indistinguishable from pituitary adenoma, the most common pituitary mass (Iglesias et al. 2000). In GCTs, MRI showed masses that are isointense to the brain on T1 weighted sequences, with enhancement being uniform or heterogeneous and of moderate to remarkable intensity after the administration of Gd-DTPA (Becker & Wilson 1981; Vogelgesang et al. 2002). Such intense enhancement reflects the high vascularity of these tumors (Huang & Castillo 2005). T2-weighted images showed an iso- or hypo-signal intensity (Hurley et al. 1994; Iglesias et al. 2000). Absence of the normal pituitary bright spot may be a clue that the tumor is of a neurohypophysial origin, but this finding is not specific, as the posterior pituitary bright spot may be absent in 10% to 20% of normal subjects (Elster 1993). When the tumor clearly arises from the pituitary stalk, and remains separate from the superior to the intact pituitary gland, GCT, albeit rare, should be considered as a likely diagnosis. The radiological differential diagnosis would include other lesions centered at the pituitary stalk. For example, Langerhans cell histiocytosis, which is localized to the stalk, usually shows strong homogenous contrast enhancement, and is associated with abnormal thickness of the hypophysis, as well as the rare pituitary stalk menigioma which may not have a dural attachment (Beems et al. 1999).

The differential diagnosis should also include lesions centered on the hypothalamus and third ventricular floor like craniopharyngiomas and optic hypothalamic chiasmatic astrocytomas (Aquílina et al. 2006). Other relevant entities in the radiologic differential diagnosis include germinomas, usually centered at or just behind the pituitary infundibulum and enhancing homogeneously on post gadolinium MRI (Aquílina et al. 2006). Also, dermoids and dermoids should be included in the differential diagnosis, but they can be easily recognized by their hyperintensity on proton weighted and fluid attenuated inversion recovery (FLAIR) sequences (Aquílina et al. 2006). Regarding FDG-PET, Wilkinson et al. (2003) described a case study of a patient with hypophysial GCT which was hypometabolic on FDG-PET scan. Although the histologic features were consistent more with a benign lesion, more FDG-PET studies in patients with GCTs are required in order to determine if glucose hypometabolism is a consistent finding (Wilkinson et al. 2003). Features such as lack of calcification and a suprasellar blush on angiography have been proposed as means of differentiating GCTs from other tumors in the suprasellar region (Doron et al. 1965; Lima et al. 1960). However, Bubl et al. (2001) described a case of large GCT with foci of calcifications, and they suggested that the existence of calcifications even though not a usual finding in GCTs, may appear in large tumors. Then they argued that differential diagnosis should include also pituitary adenoma, meningioma, craniopharyngioma, aneurysm, teratoma and chordoma. Although GCTs are not cystic tumors, Mumert et al. (2011) described a case study of a patient with symptomatic GCT and cystic components on imaging. Regarding magnetic resonance angiography and cerebral angiography, the findings were variable in terms of neovascularization of the mass (Doron et al. 1965).

**Histology**

Macroscopically, GCT is a lobulated, rubbery-firm, spherical, well demarcated and non capsulated mass (Giangaspero & Cencacchi 1999). Because the clinical and imaging findings of neurohypophysial GCTs are not specific, the only way to establish the correct diagnosis is via the histo-morphologic study of these tumors (Popovic et al. 2007). The tumors are composed of densely packed, large, round or oval cells lacking any characteristic arrangement, with eosinophilic, granular, PAS-positive and diastase-resistant cytoplasm (Liwnicz et al. 1984; Rickert et al. 1997). Sometimes, small areas of foamy cells can be observed and perivascular lymphocytic aggregates are also a common feature of GCTs (Liwnicz et al. 1984). Despite the frequently absent mitotic activity, Kasahima et al. (2000) in their study of GCTs with histologically atypical features, reported lesions that were characterized by remarkable cellular and nuclear polymorphism, nuclear hyperchromatism, large nucleoli, mitosis and necrosis.

Regarding immunohistochemical profiles, the findings were variable: in most cases the tumors were mostly immunoreactive to S100 and occasionally to macrophage-histiocyte markers (Kudawara et al. 1999; Nishioka et al. 1991; Shuangshoti et al. 1998). Variable findings were also reported regarding staining for GFAP which is normally expressed in the pituicytes (Salm et al. 1982). Some groups found negative staining for GFAP (Liwnicz et al. 1984; Shuangshoti et al. 1998; Nathrath & Remberger 1986; Nishioka et al. 1991) while others found a positive reaction in a few cells (Lafitte et al. 1994). These immunohistochemical characteristics regarding S100 protein and GFAP staining are similar to those seen in “transitional” pituicytes (Shuangshoti et al. 1998). So, Vinores (1991) suggested that in the “transitional” pituicyte, intermediate filaments are lost with autophagocytosis, and the GFAP immunoreactivity partially remains in the “transitional” pituicyte cell type. According to this hypothesis, he suggested that it is likely that neurohypophysial GCTs are derived from pituicytes. None the less, GCTs may also show positiv-
ity in staining for α-1-antitrypsin, α-1-antichymotripsin and cathepsin B suggestive of histiocytic differentiation (Thapar & Kovacs 1998). GCTs should be clearly distinguished from other intrasellar neoplasms, especially pituitary adenomas and pituitary adenomas (Takei et al. 2005). Because the immunohistochemical profiles of these three tumors are similar, yet the histological characteristics mentioned above are different, histological findings are crucial for this distinction.

**Treatment options and outcome**

Although no large systematic studies of GCTs have been conducted, these tumors seem to be typically benign and indolent despite reports of occasional invasion and recurrence (Shuangshoti et al. 1998). Relapse, although infrequent, has been attributed to incomplete excisions, and it usually manifests several years later (Cone et al. 1990). Although complete surgical excision appears to be the most appropriate treatment, in view of the location, high vascularity and the benign, slow growth nature of the tumor, a partial removal that adequately decompresses the optic chiasm is sometimes recommended (Albuquerque et al. 1992). The firm and vascular nature of GCTs often encountered intraoperatively (Doron et al. 1965; Lafitte et al. 1994; Massie 1979; Poppen & Packard, 1966; Symon et al. 1971), along with the lack of obvious dissection plane between the tumor and the normal brain (Schaller et al. 1998), often prohibit their gross total resection. Moreover, because this tumor may originate to be intimately involved with the pituitary stalk, maximal resection often requires sacrificing the stalk (Cohen-Gadol et al. 2003).

Many factors determine the choice of the surgical approach, including the size and suprasellar extension of the tumor. The tough consistency and the vascular nature of GCTs render trans-sphenoidal resection difficult, and in general, trans-sphenoidal surgery alone is not sufficient for resection of firm sellar tumors (Alleyne et al. 2002). In their description of six cases, despite employing a range of surgical approaches, including the sub-frontal, trans-callosal and trans-sphenoidal routes, Becker & Wilson (1981) accomplished only a subtotal resection in each case. They stated that priority should be given to the decompression of the optic apparatus, rather than to complete tumor resection. Moreover, Cohen-Gadol et al. (2003) have shown in their series of 11 cases that subtotal resection is effective with no recurrence occurring, and therefore, they also argued that the goal of surgery should be decompression of the surrounding structures rather than total removal of the tumor. In their review of the literature, there were 15 patients that had undergone subtotal resection with available follow-up data; nine of them received radiation treatment, and 6 did not. There were 2 recurrences in the former group, and 2 in the latter, with average follow-up periods of 4.7 and 5.4 years, respectively. In another review of the literature, Schaller et al. (1998) found low survival in patients who had been treated conservatively. Therefore, they have recommended surgical treatment for all symptomatic neurohypophesial GCTs. It is noteworthy, however, that no comparison was done regarding tumor characteristics between the groups of treatment modality.

Although GCT usually is a benign slow growing tumor, re-growth after subtotal resection has been reported (Satyamurti & Huntington 1972); however, malignant GCTs are exceedingly rare (Rhee et al. 2002). Kershensnik et al. (1994) reviewed more than 30 cases of intracranial malignant GCT in the literature. Six of the 33 reported malignant GCTs were located in the head and neck region. However, there were no reports of malignant GCTs arising from the neurohypophysis (Moriyama et al. 1996). High mitotic activity was observed in the cerebral malignant GCT (Albuquerque et al. 1992; Geddes et al. 1996) and in the neurohypophysial GCT with local recurrence (Lima et al. 1960; Shuangshoti et al. 1998). Thus, mitotic activity seems to be particularly pertinent to the prognostic factor in intracranial GCT (Kasashima et al. 2000).

The role of radiation therapy for GCTs remains controversial, especially in the light of their slow growth (Becker & Wilson 1981; Cone et al. 1990). Nonetheless, adjuvant radiation treatment may be reasonable for a tumor with atypical features, like mitotic ones (Cohen-Gadol et al. 2003). In their review of the literature, Schaller et al. (1998) concluded that radiation therapy alone may provide no benefits. Becker & Wilson (1981) reviewed cases of GCT with and without radiotherapy as an adjunctive or primary mode of therapy. They concluded that radiotherapy was of little or no benefit, and did not alter the short or long-term prognosis, a conclusion corroborated by other case reports (Doron et al. 1965; Liss & Kahn 1958).

Although the tumor is usually slow-growing and benign, early detection can prevent symptomatic complications that emerge when the tumor enlarges. Compression of the optic nerve, pituitary gland, hypothalamus and third ventricle are possible when growth is unchecked. So, yearly surveillance MRI scans should be obtained to check for recurrence of the tumor (Rhee et al. 2002). Although complete surgical excision appears to be the optimal choice, in view of the location, high vascularity and benign slow growing nature of the tumor, partial removal adequately decompressing the optic chiasm could be the optimal recommendation (Cohen-Gadol et al. 2003). Large symptomatic tumors causing visual symptoms, and small tumors found incidentally may vary in growth pattern, and more follow-up data are required before any definitive conclusion about the treatment or prognosis of neurohypophesial GCTs can be made (Moriyama et al. 1996).

Regarding our patient; before surgery she did not have hypopituitarism or diabetes insipidus, and visual field test showed a minimal defect on superior temporal field of left eye that suspected to worsen. On surgery, the mass was not extremely firm or vascular, but it was...
intimately involving the pituitary stalk, and it was completely removed scarifying the stalk with no serious bleeding encountered. After surgery a serious deterioration to the patient’s visual fields was observed, developing bilateral temporal hemianopia, hypopituitarism and diabetes insipidus. The deterioration of the visual fields is suspected to originate from vascular damage to the shared vascular supply of the mass and the optic chiasm, and hypopituitarism most probably is a result of scarifying the pituitary stalk.

SUMMARY

So far, the current consensus of treating GCTs is to attempt radical resection within safe limits and reserve radiation adjuvant therapy to those tumors with atypical features showing recurrence on serial imaging. The tumor’s firm consistency, tendency to hemorrhage, involving the pituitary stalk and lack of dissection plane from basal brain structure render surgery difficult and maximal resection often requires sacrificing the stalk. If the intraoperative gross appearance looks like GCT, the mass is firm and seems to be hypervascular, then effort should be done to avoid bleeding and not to carry out heroic attempts trying to remove the hall mass. Instead, only decompensation of the surrounding structures should be pursued. Moreover, small asymptomatic neurohypophysial GCTs are common findings, most probably benign tumors with slow growing nature. Hence, in our patient, the tumor should have been suspected to be a GCT, and even though a mild deterioration in her visual field was observed, trying to avoid the post-surgical development of hypopituitarism caused by scarifying the stalk, close follow-up with repeated MRI and visual field studies might have been a better approach. For a neurohypophyseal tumor which is suspected to be a GCT, this case report and review of the literature, offers to consider the alternative approach, with close clinical, visual field and radiological study follow up.

REFERENCES

Leonard Saiegh, Majed Odeh, Mohammad Sheikh-Ahmad, Maria Reut, Zvi Ram, Carmela Shechner