REVIEW

Giant Chondroma of the Saddle Area: Case Report and Literature Review*

Lubin Qiu¹, Yongjun Zhu¹, Haijun Wang², Yatang Wang¹, Qiang Wu³, and Jianming Yang¹

¹Department of Otorhinolaryngology, the Second Hospital of Anhui Medical University, Hefei, People's Republic of China, ²Department of Neurosurgery, the First Affiliated Hospital of Sun Yat-sen University, Guangzhou, People's Republic of China, and ³Department of Pathology, the Second Hospital of Anhui Medical University, Hefei, People's Republic of China

ABSTRACT

A 63-year-old man presented with sexual dysfunction of 6-year duration, 5-year history of bilateral vision loss, and left nasal obstruction for 3 years. Brain computed tomography and magnetic resonance imaging showed a large mass lesion in the saddle area and extending upward to the dorsum sellae, bilateral cavernous sinus, and suprasellar region, and down into the sphenoid sinus and nasal cavity; the optic nerves and optic chiasm were elevated upward and compressed. Endocrine tests indicated that all serum level of anterior pituitary hormones decreased. The preoperative diagnosis included invasive pituitary adenoma, chordoma, osteosarcoma, chondrosarcoma, and craniopharyngioma. The tumour was subtotally removed through transsphenoidal approach. Histopathology examination revealed a chondroma. Postoperatively, the patient was stable and his visual acuity and visual field defect improved and his pituitary function return to normal except for hypothyroidism.

Keywords: Computed tomography (CT), histology, magnetic resonance (MR), sellar chondroma, transsphenoidal approach

INTRODUCTION

Chondromas are benign and slow-growing tumours, which remain clinically asymptomatic for a prolonged period of time in the majority of cases and consist of mature hyaline cartilage. Chondromas in the sellar region are exceedingly rare and usually secondary to adjacent synchondrosis or extracranial chondromas. Their clinical symptom is not characteristic and their radiographic appearance usually does not distinguish them from their more common counterparts, such as pituitary adenoma, chordoma, osteosarcoma, chondrosarcoma, and craniopharyngioma, so their diagnosis depend on histopathological examination. Surgical resection is the treatment of choice for sellar

chondroma, especially for cases with symptoms of visual field defect, but a total resection of the tumour is usually impossible, as these tumours are usually solid and extend superiorly in their location and are firmly adherent to adjacent structures such as cavernous sinus; however, sellar chondroma requires relief of the compression to the chiasm or optic nerve as soon as possible, and a partial resection of the chondroma is beneficial to relieve compression on the anterior visual pathway. To our knowledge, there have been no more than 20 cases of sellar chondroma in the literature since the first case was reported by van Duyse in 1937.¹ We described herein a case of sellar chondroma and literature review of the chondroma's presentation, pathology, and management options.

*Lubin Qiu and Yongjun Zhu contributed equally to this work.

Received 7 May 2013; revised 4 July 2013; accepted 7 July 2013; published online 15 November 2013

Correspondence: Lubin Qiu and Jianming Yang, MD, Department of Otorhinolaryngology, the Second Hospital of Anhui Medical University, Hefei, 230601, People's Republic of China. E-mail: 916qlb@sina.com

CASE REPORT OF SELLAR CHONDROMA

A 63-year-old man was brought to a local hospital due to 3 years of left nasal obstruction. The past history included sexual dysfunction of 6-year duration, and 5-year history of bilateral vision loss. Computed tomography (CT) imaging at a local hospital revealed a large mass in the sphenoid sinus with calcification within the mass and showed bone destruction in the saddle area. The patient was referred to a tertiary hospital and received multidisciplinary care. The detailed physical examination on admission revealed that this patient had a bilateral superior altitudinal visual field defect in both the eyes, his visual acuity was 0.4 on the right and 0.6 on the left (Figure 1). No other neurological abnormalities including the other cranial nerves were found. Endocrine tests indicated that all serum levels of anterior pituitary hormones were decreased, but diabetes insipidus was not present. In addition, this patients has apparent hypoproteinaemia and body oedema, his skin was dry and cool.

Preoperative plain and contrast-enhanced CT revealed large mass in the sellar region and upper clivus, the mass was not enhanced by contrast medium, most of the calcification was located at the sellar region and upper clivus. The mass extended into the suprasellar and bilateral parasellar regions and sphenoid sinus, and entered into the nasal cavity. Bone-window images disclosed bone destruction of the pituitary fossa, the dorsum sellae and posterior clinoid process. There was no mass effect on cerebral parenchyma and no peripheral oedema. Because no enhancement of the hypointense areas was seen on the CT images, the boundary of the tumour is not clear from CT scans (Figure 2A, B).

Magnetic resonance (MR) imaging showed a huge mass lesion in the saddle area and presented as hypointense on the T1-weighted image, hyperintense on the T2-weighted image. The mass had a cystic core with irregular contrast enhancement: mixed high and low signal intensities were seen on the enhanced MR image. The mass lesion occupied the entire hypophyseal fossa, and extended upward to the dorsum sellae, bilateral cavernous sinus, and suprasellar

FIGURE 1 The analysis of visual fields shows that both eyes had bilateral superior altitudinal visual field defect. (A) visual fields of left eye, preoperative; (B) visual fields of left eye, postoperative; (C) visual fields of right eye, preoperative; (D) visual fields of right eye, postoperative.

FIGURE 2 Preoperative CT scan showing a large mass with scattered calcification at the sellar region and upper clivus. Bone-window images disclosed skull base bone destruction (A, B). Preoperative MRI showing a huge lesion with scattered cystic core in the saddle area and was slightly enhanced and showing heterogeneous signal intensities. The lesion occupied the entire hypophyseal fossa, dorsum sellae, bilateral cavernous sinus, and suprasellar region, and down into the sphenoid sinus and nasal cavity. The optic chiasm were elevated upward and compressed, the pituitary gland was not identified, and the boundary of lesion could be basically separated from the adjacent structures and there was peri-tumoural oedema (C, D).

FIGURE 3 Endoscopic view preoperative (A) shows a tumour analogue in the posterior segment of the left nasal cavity, the surface of which is pale red, smooth, and the choanal has been completely blocked. Postoperative endoscopy (5 months) (B, C) reveals that the anterior wall of the sphenoid sinus and posterior section of nasal septum have been partially resected, the nasal cavity is now unobstructed. Postoperative endoscopic examination (9 months) (D) shows that the anterior wall of the sphenoid sinus has recovered. N = nasal septum; S = sphenoid sinus; T = tumour.

region, and extended down into the sphenoid sinus and eroded the sphenoid bone into nasal cavity; the optic nerves and optic chiasm were elevated upward and compressed. Tissue of the pituitary gland was not identified in any of the sections. The whole mass could be basically separated from the adjacent structures on the whole and there was no perifocal oedema around the brain parenchyma (Figure 2C, D).

Endoscopic view of the left nasal cavity shows a tumour analogue in the posterior segment of nasal cavity, the surface of its mucous is pale red, smooth, and the choanal has been completely blocked (Figure 3A).

According to the clinical symptoms and the results of radiological images, the preoperative diagnosis included invasive pituitary adenoma, chordoma, osteosarcoma, chondrosarcoma, and craniopharyngioma. The patient was treated with hormone replacement and transfusion of plasma and albumin preoperatively.

The otorhinolaryngologists and neurosurgeon decided a two-stage surgical plan given the size of the tumour bulk. The first step is to remove the entire tumour within hypophyseal fossa, sphenoid sinus, nasal cavity, and most of the tumour within the bilateral cavernous sinus, dorsum sellae, and suprasellar region via transsphenoidal approach. The second step is to resect residual tumour within the bilateral cavernous sinus and suprasellar region either via transcranial route or with radiation therapy.

During removal of the tumour, we found that there was no capsule around the tumour and the surface of the tumour in the nasal cavity was nasal mucosa, most of the tumour tissue was soft and greyish, containing mucilage. At first, we resected the tumour tissue within the sphenoid sinus and nasal cavity; blood oozing was easily controlled through oppression. We found that the bone of the floor of the sellar tursica was completely destroyed and the sella enlarged after entirely removing the tumour tissue in the sphenoid

sinus; the lesion was carefully debulked using endoscopic curette. When removing the tumour tissue within the cavernous sinus, this part of the tissue was found to be a little tough and hypervascular during operation, it had the same characteristics as pituitary fibrous adenoma and was firmly adherent to the structures of the cavernous sinus. In order to avoiding profuse bleeding and nerve trauma, with 30-degree endoscopes facilitating direct visualization, we proceeded to remove the tumour tissue by curettage without any tear through using pituitary ring curette and suction tube. The second stage of the operation with a transcranial approach showed the cranial nerves. During removal of the tumour, the cranial nerve and the cavernous CA were not found inside the tumour, and the venous bleeding from the sinus itself was easily controlled. The calcified part of the tumour, which was mainly located in the sella region, was removed using a diamond drill and a sucker and tumour forceps. The histological diagnosis was chondroma after the operation (Figure 4A–C; IHC a–f).

Postoperatively, the patient was stable and his visual acuity and visual field defect improved markedly. His pituitary function is normal except for hypothyroidism. Treatment with L-thyroxine at a dose of 50 µg per os (p.o.) a day was started and dose was increased to 100 µg p.o. a day; he became asymptomatic after a 2-month replacement treatment. One-month postoperative MRI demonstrated subtotal removal of the tumour, the residual lesion situated in the suprasellar area and outside of the cavernous sinus and the suprasellar portion did not come down into the sellar cavity (Figure 5A, C). The patient refused to receive secondary operation by an intracranial subfrontal approach or radiotherapy, and follow-up visits demonstrated no neurological deficits and normal pituitary functions. Follow-up with MRI 9 months post operation showed no growth of the residual tumour (Figure 5B, D). Postoperative endoscopic examination 5 months

FIGURE 4 Histology and immunohistochemistry (IHC) of chondroma. General HE stain (A, B, C) showing the tumour consisted of hyaline cartilage; (IHC a) positive IHC to anti-S-100 ($\times 200$); (IHC b) positive IHC to anti-vimentin ($\times 100$); (IHC c–f) negative IHC to anti-ck, anti-ema, anti-gfap, anti-ki67 ($\times 100$), respectively.

FIGURE 5 Postoperative (1.5 months) (A) plain axial T1-weighted and (C) sagittal T1-weighted with contrast MRI showing subtotal removal of the tumour, the residual lesion situated in the suprasellar area and outside of the cavernous sinus. Postoperative (9.5 months) (B) axial T1-weighted and (D) sagittal T1-weighted with contrast MRI showing no recurrence of the lesion.

later revealed a sphenoid sinus free from recurrence, the surrounding walls of the sphenoid sinus were normal except for partial resection of the anterior wall, the posterior section of nasal septum has been partially resected, and the anatomical structure of

nasal cavity and the physiological function of the nose has been preserved (Figure 3B, C). Postoperative endoscopic examination 9 months later showed that the anterior wall of the sphenoid sinus has recovered (Figure 3D).

DISCUSSION

Chondrogenic neoplasms, arising from the base of the skull,^{2,3} both benign and malignant, are not very common.⁴⁻⁹ According to Falconer *et al.*, skull base chondromas can be divided into three groups: sellar chondromas, parasellar chondromas, and clival chondromas,¹⁰ but even among basal chondromas, sellar chondromas are rare, as no more than 20 cases have been reported to date.^{5,8-19} The origin of skull base chondromas is not clear, many theories have been suggested.² De Divitiis *et al.*¹³ first discussed the origin of intrasellar chondromas in 1979 and they stated that it should be the same as that of intracranial chondromas in general.¹³ Basal chondromas have been believed to originate from embryonic crests of chondrogenic cells along baseline syndesmoses.^{2,20-23} It is also thought that sellar chondromas may develop from fibroblasts in the diaphragma sellae.^{5,15} Embryonic cartilaginous rests from the base of the skull might be misplaced in the developmental stage.^{5,13,18} In addition, it is tempting to speculate that basic fibroblast growth factor (b-FGF) secretion, which in turn is stimulated by pituitary tumour-transforming gene (PTTG), may have contributed to the development and progression of the chondroma through a paracrine effect.^{4,24}

Chondromas are usually very large when diagnosed, so they have varying degrees of invasion into adjacent structures such as the optic nerves, optic chiasm, internal carotid artery, or other cranial nerves.^{5,8,9,10,13,15,18,20,23,25-31} The common clinical characteristics of intrasellar chondromas^{8,10,13,15,17,18,32} include chiasmatic syndrome associated with irregular bitemporal hemianopsia or inferior quadrantanopsia, decline of visual acuity, and hypopituitarism; the signs of parasellar or clival chondromas are characterized by involvement of the third, fourth, fifth, and sixth cranial nerves, occasionally with incongruous homonymous hemianopsia when the optic tract is invaded.^{5,10,18,20,23,28,30,31}

CT and MR imaging findings that contribute to the diagnosis of chondroma are as follows: calcification from granular to massive of the sellar turcica, resulting from varying degrees of degeneration and necrosis; bone destruction of the sellar turcica; marked delayed contrast enhancement on CT scan; hyperintensity on T2-weighted images reflecting the chondroid matrix; and markedly low signal intensity similar to that of cerebrospinal fluid (CSF) on infrared (IR) images.^{5,8,10,17,18,25,28,33-38} According to Lacerte *et al.*, intradural chondromas have two distinct types of CT images.³⁹ One is more common and shows mixed density with minimal or moderate enhancement, the other has a central hypodense area that is composed of cystic degeneration or of a very loose-texture connective tissue without necrosis in pathological assessment. Tanohata *et al.* emphasize that

enhancement of chondromas increases after 30 minutes of contrast injection.^{2,37} Coronal CT of bone windows shows multiple small circular and curvilinear calcifications; 60% of intracranial chondromas have mottled shapes and 50% have bone destruction.^{2,5,18,20,25,30,31,32,40-42} MR imaging of the chondroma mass depends on the hyaline cartilaginous or ossification content.⁵ Vasopressin storage, bone marrow, and adenohipophysial hyperactivity are the three main factors for high T1 signal intensity in normal conditions.⁴³ Diseases including blood products, fat, high concentrations of protein, or calcifications imply the majority of pathological entities that lead to high T1 signal hyperintensity of various degrees in the sellar and parasellar regions.^{5,43} T2-weighted imaging often demonstrates hyperintense areas.^{5,17,18,20,25,44-46} T2 spin-echo scan shows a peripheral heterogeneous hypointense area and a well-demarcated hyperintense central area with rare perifocal oedema,^{2,39,47,48} and the chondromas are slightly to moderately enhanced with contrast medium.^{5,9,20,25,30,31} The MR images with the inversion recovery sequence show chondromas as markedly hypointense in contrast to chordomas, which appear as only hypointense,^{5,46} the former usually have a more lateral location, farther from the midline, at the petrooccipital synchondrosis.^{2,32,49} The classical chondroid calcifications are curvilinear and have low signal intensity on MR images acquired with spin-echo sequences. They also may appear as areas of hyperintense signal on T1-weighted images, depending on the degree of mineralization.² The infrasellar midline masses of intracranial chordomas usually have T1 signal hypointensity and T2 signal hyperintensity, with suggestive hypointense intratumoural septations and posterior extension indenting the pons.^{43,50} So it is very difficult to distinguish them from other tumours arising at the same location, such as chordomas, meningiomas, craniopharyngiomas, or pituitary adenomas, chondrosarcoma, and osteosarcoma before the operation.^{5,21}

Surgical resection is the treatment of choice for sellar chondroma, especially for cases with symptoms of visual field defect, cranial nerve palsies, and hypopituitarism (chiasmatic syndrome),^{5,21,25} but a total resection of the tumour is usually impossible, as these tumours with weakly vascularized nature are usually solid and extend superiorly in their location and are firmly adherent to adjacent structures such as the cavernous sinus, etc.^{2,25} These elements pose great risk to the cranial nerves or the carotid artery when removing the tumour.^{2,5,8-10,13,15,18,23,25,27-32}

The extent and scope of surgical resection should be varied according to the nature of the tumour and its varying degrees of involvement of the cavernous sinus.²⁵ Usually, sellar chondroma requires relief of the compression to the optic chiasm or optic nerve as soon as possible, so partial resection can still be

beneficial.⁵ Numerous studies have demonstrated detailed knowledge about the microsurgical anatomy of the saddle area that contributes to successful surgical exploration and description of safe entrances in this challenging area.^{25,51–58} Precise preoperative radiological findings, including MR images and thinly sliced CT scans, are crucial for selecting the surgical approach.²⁵ When attempting surgical removal of chondroma in the saddle area, we think that attention should be directed to the surgical possibilities of resection and the probability of nerve preservation.

Both transsphenoidal and transcranial microsurgery to sellar lesions are safe and efficient treatment. The invasion scope of the tumour and the characteristics of the tumour tissue determine the choice between the transcranial and transsphenoidal approaches.^{59–61} Tumours with bilateral cavernous sinus involvement are better suited for a bifrontal transbasal type of approach. Those with extensive invasion of the cavernous sinus unilaterally are generally best suited for a frontotemporal transcavernous approach. Suprasellar lesions are best exposed by an approach that affords the surgeon an adequate inferior-to-superior viewing angle, which is generally accomplished by removal of all or part of the orbital rim. Some tumours with more modest extensions outside the bounds of the sella are now treated with a more minimalistic approach via a small incision in the eyebrow.^{59–61} These strategies can decrease frontal lobe retraction, which may be particularly important to cases requiring a bilateral approach. The transsphenoidal approach is the choice for tumours occupying the sella, extension into the sphenoid sinus, and even into the nasal cavity, and for tumours with suprasellar expansion without lateral extension. The transsphenoidal approach is contraindicated for tumours with significant lateral parasellar extension or massive suprasellar expansion.^{59–61} We do not recommend transsphenoidal approaches to lesions with lateral and suprasellar extensions because of inadequate exposure and decompression.^{59–61}

From our experience, it must be emphasized that, in the case of large tumours with considerable suprasellar extension, essential experience is needed to properly assess the amount of curettage. In the unusual circumstance of a fibrous lesion because of its firm consistency, we think that it would be best to use a curved curette and Blakesley-Weil forceps, removing the lesion mass piece by piece under direct visualization with the use of 30-degree and 45-degree 4-mm rigid endoscope without producing a tear. Some pathological tissue may be hidden in the lateral corners of the sella; using a 45-degree-angle fiberoptic mirror, this tissue can be identified and removed with a 90-degree-angle curette and suction tube. The oozing is of no real consequence, since it drains into the sphenoid sinus and the submucosal nasal opening.

Generally, chondromas are benign and slow in growing and have a good prognosis and the cases' life expectancy is good, but MR imaging or CT, visual examination, tests of endocrine function, and control of hypopituitarism are required during follow-up,^{4,5,21} because malignant degeneration of the remnant has been reported in some cases.^{5,8–10,13,15,18,20,23,27–32} If a benign, diagnosed chondroma shows rapid recurrence, invasion, or metastasis, other lesions such as chondrosarcoma, chordoma, Ollier's disease, and Maffucci's syndrome should be suspected and the histology specimen should be reviewed for correct diagnosis.^{5,39}

ACKNOWLEDGMENTS

We thank Feiyan Chen for providing cryoconserved tissue from patients.

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

This work was supported by the Department of Pathology of the Second Hospital of Anhui Medical University.

Note: Figures 3 and 4 of this article are available in colour online at informahealthcare.com/oph.

REFERENCES

- [1] van Duyse, M. A propos d'un cas de syndrome chiasmatique et hypophysaire (Myxochondrome résultant de la métaplasie d'un crano-pharyngiome). *Bull Soc Belge Ophthalmol* 1937;75:70–77.
- [2] Gana R, Maaqili RE, Fatemi NE, Bellakhdar F. Intracerebral chondroma. *Pan Arab J Neurosurg* 2008;12:73–75.
- [3] Mapstone TB, Wongmongkolrit T, Roessman U, Ratcheson RA. Intradural chondroma: a case report and review of the literature. *Neurosurgery* 1983;12:111–114.
- [4] Sahli R, Christ E, Kuhlen D, Giger O, Vajtai I. Sellar collision tumor involving pituitary gonadotroph adenoma and chondroma: a potential clinical diagnosis. *Pituitary* 2011;14:405–408.
- [5] Aoki A, Mori K, Tajima A, Maeda M. Sellar chondroma case report. *Neurol Med Chir (Tokyo)* 1999;39:870–874.
- [6] Semenova LA, Bulychева IV. Chondromas (enchondroma, periosteal chondroma, enchondromatosis). *Arkh Patol.* 2007;69:45–48.
- [7] Kozłowska K, Bień D, Niemczyk K. Chondroma of the skull base. *Otolaryngol Pol* 2004;58:995–998.
- [8] Paillas JE, Alliez B. Intracellar chondroma-case report. *Neurochirurgia (Stuttg)* 1974;17:136–140.
- [9] Munemitsu H, Matsuda M, Hirai O, Fukumitsu T, Kawamura J. Intracellar chondroma. *Neurol Med Chir (Tokyo)* 1981;21:775–780.
- [10] Falconer MA, Bailey IC, Duchon LW. Surgical treatment of chordoma and chondroma of the skull base. *J Neurosurg* 1968;29:261–275.
- [11] Asenjo A, Chirorino R. Mixochondroma endocraneano (Estudio anato-clinico de dos casos). *Rev Esp Otoneurooftal* 1956;15:15–21.

- [12] David M, Constans JP. Les chondromes intracraniens. *Sem Hop* 1957;33:1195–1214.
- [13] de Divitiis E, Spaziante R, Cirillo S, Stella L, Donzelli R. Primary sellar chondromas. *Surg Neurol* 1979;11:229–232.
- [14] Hardy J, Bertrand C, Maltais R, Robert F, Thierry A. Volumineux chondrosarcome calcifié de la; région sellaire (exerese trans-sphenoidale; sous controle radioscopique televise). *Neurochirurgie* 1966;12:491–502.
- [15] Inoue K, Sugiyama Y, Ishii R. Intracellular fibromyx chondroma. *Surg Neural* 1978;10:167–170.
- [16] List CF. Osteochondroma arising from the base of the skull. *Surg Gynecol Obstet* 1943;76:480–492.
- [17] Osborn AG. Image interpretation session. Intracellular chondroma. *Radiographics* 1991;11:148–150.
- [18] Pospiech J, Mehdorn HM, Reinhardt V, Grote W. Sellar chondroma in a case of Ollier's disease. *Neurochirurgia (Stuttg)* 1989;32:30–35.
- [19] van Duyse M. A propos d'un cas de syndrome chiasmatique et hypophysaire (Myxochondromeré; sudlet alnat metaplasie d'un crano-pharyngiome). *Bull Soc Belge Ophthalmol* 1937;75:70–77.
- [20] Reuter K, Weber AL. Parasellar chondrosarcoma in a patient with Ollier's disease. *Neuroradiology* 1981;22:151–154.
- [21] Munemitsu H, Matsuda A, Hirai O, Fukumitsu T, Kawamura J. Intracellular Chondroma case report. *Neurol Med Chir (Tokyo)* 1981;21:775–780.
- [22] Berkmen YM, Blatt ES. Cranial and intracranial cartilaginous tumors. *Clin Radiol* 1968;19:327–333.
- [23] Dutton J. Intracranial solitary chondroma: case report. *J Neurosurg* 1978;49:460–463.
- [24] Ezzat S, Smyth HS, Ramyar L, Asa SL. Heterogenous in vivo and in vitro expression of basic fibroblast growth factor by human pituitary adenomas. *J Clin Endocrinol Metab* 1995;80:878–884.
- [25] Terasaka S, Sawamura Y, Abe H. Surgical removal of a cavernous sinus chondroma. *Surg Neurol* 1997;48:153–159.
- [26] Higashida T, Sakata K, Kanno H, Tanabe Y, Kawasaki T, Yamamoto I. Intracranial chondroma arising from the skull base: two case reports featuring the image findings for differential diagnosis. *No Shinkei Geka* 2007;35:495–501.
- [27] Acampora S, Troisi F, Fusco G, Del Gaizo S. Voluminous intracranial chondroma. *Surg Neurol* 1982;18:254–257.
- [28] Ghogawala Z, Moore M, Strand R, Kupsky WJ, Scott RM. Clival chondroma in a child with Ollier's disease. *Case report. Pediatr Neurosurg* 1991–1992;17:53–56.
- [29] Ishida M, Ashida K, Matsunaga T, Wakasa K, Sakurai M. Chondrosarcoma of the ethmoidal and sphenoidal sinuses. A case of chondrosarcoma arising from postparanasal sinuses. *ORL J Otorhinolaryngol Relat Spec* 1986;48:174–179.
- [30] Kikuchi K, Kowada M, Monma F, Rikimaru S, Tamakawa Y. An operated case of clivus chondroma [abstract]. *No Shinkei Geka* 1979;7:271–276.
- [31] Kitahara T, Morii S, Miyasaka Y, Ohwada T, Yada K. Two cases of basal chondroma [abstract]. *No Shinkei Geka* 1979;7:809–814.
- [32] Chakraborty S, Tamaki N, Kondoh T, Kojima N, Kamikawa H, Matsumoto S. Maffucci's syndrome associated with intracranial enchondroma and aneurysm. *Surg Neurol* 1991;36:216–220.
- [33] Daniels DL, Czervionke LF, Bonneville JF, Mark LP, Pech P, Hendrix LE, Smith DF, Haughton VM, Williams AL. MR imaging of the cavernous sinus: value of spinecho and gradient recalled echo images. *AJR Am J Roentgenol* 1988;151:1009–1014.
- [34] Hirsch WL, Hryshko FG, Sekhar LN, Brunberg J, Kanal E, Latchaw RE, Curtin H. Comparison of MR imaging, CT and angiography in the evaluation of the enlarged cavernous sinus. *AJR Am J Roentgenol* 1988;151:1015–1023.
- [35] Hirsch WL, Sekhar LN, Lanzino G, Pomonis S, Sen CN. Meningiomas involving the cavernous sinus: value of imaging for predicting surgical complications. *AJR Am J Roentgenol* 1993;160:1083–1088.
- [36] Ozgen T, Pamir MN, Akalan N, Bertan V, Onol B. Intracranial solitary chondroma. *J Neurosurg* 1984;61:399–401.
- [37] Tanohata K, Maehara T, Aida N, Umino S, Matsui K, Mochimatsu Y, Fujitsu K. Computed tomography of intracranial chondroma with emphasis on delayed contrast enhancement. *J Comput Assist Tomogr* 1987;11:820–823.
- [38] Tashiro T, moue Y, Nemoto Y, Shakudo M, Mochizuki K, Yahata S, Tada T, Hata M, Oda J, Onoyama Y. Magnetic resonance (MR) imaging of chordoma and chondroma in the skull base. Differential diagnosis by IR sequence. *Nippon Acta Radiol* 1992;52:589–593.
- [39] Lacerte D, Gagne F, Coptly M. Intracranial chondroma: report of two cases and review of the literature. *Can J Neurol Sci* 1996;23:132–137.
- [40] Sebbag M, Schmidt V, Leboucq N, Bitoun J, Castan PH, Frerebeau PH. Chondrome dure-mérien; a propos d'un cas et revue de la littérature. *J Radiol* 1990;71:495–498.
- [41] De Coene B, Gilliard C, Grandin C, Nisolle JF, Trigaux JP, Lahdou JB. Unusual location of an intracranial chondroma. *AJNR Am J Neuroradiol* 1997;18:573–575.
- [42] Traflet RF, Babaria AR, Barlat G, Doan HT, Gonzalez C, Mishkin MM. Intracranial chondroma in a patient with Ollier's disease. *J Neurosurg* 1989;70:274–276.
- [43] Bonneville F, Cattin F, Marsot-Dupuch K, Dormont D, Bonneville JF, Chiras J. T1 signal hyperintensity in the sellar region: spectrum of findings 1. *Radiographics* 2006;26:93–113.
- [44] Brownlee RD, Sevic RJ, Rewcastle NB, Tranmer BI. Radiologic-pathologic correlation. Intracranial chondroma. *AJNR Am J Neuroradiol* 1997;18:889–893.
- [45] Takizawa H, Sugiura K, Baba M, Chisiki T, Kamatsuka E, Tachisawa T, Kadoyama S, Hata Y, Kameda N. Parasellar chondroma: a case report. *Br J Neurosurg* 1987;1:147–151.
- [46] Tashiro T, Inoue Y, Nemoto Y, Shakudo M, Nochizuki K, Yahata S, Tada T, Hata M, Oda J, Onoyama Y, Katsuyama J, Hakuba A. Magnetic resonance (MR) imaging of chordoma and chondroma in the skull base. Differential diagnosis by IR sequence [abstract]. *Nippon Igaku Hoshasen Gakkai Zasshi* 1992;52:589–593.
- [47] Holthouse DJ, Robbins PD, Knuckey NK. Solitary intradural fibro-chondroma in a 16-year-old boy. *J Clin Neurol* 1999;6:355–357.
- [48] Khosrovi H, Sadrolhefazi A, El-Kadi H, Bloomfield SM, Scochet SS. Intradural convexity chondroma: a case report and review of diagnostic features. *West Virginia Med J* 2000;96:612–616.
- [49] Doran SE, Gebarski SS, Hoff JT. Tumors of skull. In: Youmans JR, editor. *Neurological Surgery: A Comprehensive Reference Guide to the Diagnosis and Management of Neurosurgical Problems*. Philadelphia: WB Saunders, 1996:2998–3023.
- [50] Erdem E, Angtuaco E, Van Hemert R, Park J, Al-Mefty O. Comprehensive review of intracranial chordoma. *RadioGraphics* 2003;23:995–1009.
- [51] Parkinson D. A surgical approach to the cavernous portion of the carotid artery: anatomical study and case report. *J Neurosurg* 1965;23:473–483.
- [52] Parkinson D. Carotid cavernous fistula. History and anatomy. In: Dolenc W, editor. *The Cavernous Sinus*. Vienna: Springer-Verlag; 1987:3–29.
- [53] El-Kalliny M, Van Loveren H, Keller JT, Tew J. Tumors of the lateral wall of the cavernous sinus. *J Neurosurg* 1992;77:508–514.

- [54] Harris FS, Rhoton Jr AL. Anatomy of the cavernous sinus: a microsurgical study. *J Neurosurg* 1976;45: 169–180.
- [55] Rhoton Jr AL, Hardy DG, Chambers SM. Microsurgical anatomy and dissection of the sphenoid bone, cavernous sinus and sellar region. *Surg Neurol* 1979;12:63–104.
- [56] Sekhar LN, Sen CN, Jho HD, Janecka IP. Surgical treatment of intracavernous neoplasms: a four-year experience. *Neurosurgery* 1989;24:18–30.
- [57] Umansky F, Nathan H. The lateral wall of the cavernous sinus with special reference to the nerves related to it. *J Neurosurg* 1982;56:228–234.
- [58] Van Loveren HR, Keller JT, El-Kalliny M. The Dolenc technique for cavernous sinus exploration (cadaveric prosection). *J Neurosurg* 1991;74:837–448.
- [59] Day JD. Surgical approaches to suprasellar and parasellar tumors. *Neurosurg Clin N Am* 2003;14:109–122.
- [60] Baysefer A, Erdogan E, Gezen F, Ynak ET, Seber N. Surgical treatment of sellar region neoplasms. *Turkic/i Neurosiirgeri* 1997;7:1–4.
- [61] D'Ambrosio AL, Syed ON, Grobelny BT, Freda PU, Wardlaw S, Bruce JN. Simultaneous above and below approach to giant pituitary adenomas: surgical strategies and long-term follow-up. *Pituitary* 2009;12:217–225.