Introduction

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Craniopharyngiomas are rare epithelial tumors arising along the path of the craniopharyngeal duct, and as a consequence, they can be found from rhinopharynx to hypothalamus [6, 30, 31]. They develop in a deep-seated area of the brain, involving in many cases several vital structures, such as the hypothalamus, that are of paramount importance for vegetative, endocrine, and emotional functions as well as for maintaining body homeostasis. As a matter of fact, the functional impairment and anatomical distortion of the hypothalamus that may be caused by such kind of tumor have to be considered as critical factors influencing patient outcome.

However, apart from hypothalamic dysfunction, craniopharyngiomas may lead to the emergence of a large spectrum of symptoms and signs; usual clinical presentations include visual alteration, signs of chiasmatic and/or retrochiasmatic compression, and pituitary dysfunction, often presenting as panhypopituitarism [31].

Craniopharyngiomas account for only 2–5 % of the total amount of intracranial tumors. Generally, they tend to show a double-peak distribution model, i.e., during childhood (5–14 years) and in late adulthood, from 50 to 74 years [4]. A rather balanced distribution between sexes has been observed, with 55.6 % of lesions diagnosed in males and 44.1 % in females [46].

Historically, Friedrich Albert von Zenker firstly described a cystic suprasellar mass holding inside pieces of cholesterol crystals which was probably a craniopharyngioma [61]. Later, in 1904, Jakob Erdheim depicted the main histopathological aspects of such a kind of neoplasms [16], and, in agreement with the already obtained results presented by Mott and Barret [44], he endorsed that craniopharyngiomas develop from epithelial cells arising from a partially involuted hypophyseal-pharyngeal duct. On the other hand, from the clinical point of view, Rupert Boyce and Cecil Beadles were the pioneers to describe the case of a 35 year-old patient who became comatose and rapidly died because of a huge, ossified, and cystic lesion leading to brainstem, optic chiasm, and optic tract dysfunction; that mass was

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suspected to be a craniopharyngioma [3]. Hereinafter, Babinski [1] and Frohlich [21] reported analogue clinical features in patients with cystic pituitary masses but without any symptoms and/or signs of acromegaly, questionable to not be a pituitary adenoma but a cystic intrasellar craniopharyngioma.

Regarding the neuropathological outward, two subtypes of craniopharyngiomas have been described: adamantinomatous and papillary. The adamantinomatous form affects children (5–10 years) and adults (50–60 years), the papillary type almost only adults; in general, the adamantinomatous form is much more common than the papillary one (ratio: 9/1) [47, 60]. Regarding the main macroscopic characteristic, the adamantinomatous subtype shows adhesions to the nearby neurovascular structures as well as irregular interface, and its cystic components are usually filled with dark fluid, i.e., the so-called motor oil, containing cholesterol crystals; calcifications are present and described in the large majority of cases. Instead, the papillary form generally shows no adherence to the neighboring structures; cystic contents are often clear and no calcifications are found [11]. The two craniopharyngiomas histotypes have different immunohistochemical features leading to peculiar biological behavior. In particular, the adamantinomatous form shows positivity for CK7, CK8, and CK14 [35, 56, 59] and may contain mutations in CTNNB1 [37], encoding beta-catenin, a component of the adherents junction and mediator of Wnt signaling [27, 28, 53]. A mutated, degradation-resistant form of beta-catenin is implicated as the primary driver of oncogenesis in adamantinomatous craniopharyngioma and is usually present in the cellular nucleus [29]. Specifically, clusters of cells with nuclear beta-catenin form are principally represented in invasive tumors. This pattern is evocative of an implication of the beta-catenin signaling in the migratory behavior of these tumors; the cellular clusters with nuclear beta-catenin may coordinate the growth and infiltration of the tumor into the nearby vital tissues, thus explaining the increased aggressiveness of this adamantinomatous subtype versus the papillary one [6, 36].

Craniopharyngiomas originate from the midline skull base and, subsequently, they begin to insinuate into the nearby low-resistant structures, such as the arachnoid cisterns, the third ventricle, and the parasellar areas. However, as already said, differently from pituitary tumors, they often adhere to the neurovascular structures of the suprasellar space, such as perforating vessels coming from the anterior and posterior cerebral arteries and/or internal carotid arteries, the optic chiasm and optic pathways, and the hypothalamus. Despite of their histological classification as benign tumors, an aggressive and infiltrative behavior is often observed.

The peculiar location and significant size that such tumors may reach, together with the frequent implication of critical neurovascular structures as well as the presence of calcific components, can limit the degree of resection in many cases.

Moreover, craniopharyngiomas have a tendency to recur even after apparent total removal. Surgical removal of recurrent craniopharyngioma may be more difficult, principally due to scar tissue formation and new adhesions [8, 42]. The recurrent craniopharyngioma usually adheres intensively to the surrounding hypothalamic-hypophyseal areas, thus making the second surgery at a higher risk of fatal neural and vascular injury. According to major literature studies [8, 17, 57], the rate of recurrence ranges from 0 to 53 % in cases of total removal and from 30 to 100 % in cases of subtotal or partial removal.

Along many years, several authors defined classifications for craniopharyngiomas as related to the growth path and the surgical route used, all sharing the principle of subdividing craniopharyngiomas along the length of extension in the primary vertical axis, considering the optic chiasm, diaphragma sellae, third ventricle, and more recently to infundibulum.

Hoffman classified craniopharyngiomas with respect to the sella turcica, the optic chiasm, and the floor of the third ventricle into *prechiasmatic*, *retrochiasmatic*, *subchiasmatic*, and *intraventricular* craniopharyngiomas [26]. *Yasargil* divided them as follows: (a) *purely intrasellar–infradiaphragmatic*; (b) *intra- and suprasellar*, *infra- and*

supradiaphragmatic; (c) *supradiaphragmatic parachiasmatic and extraventricular*; (d) *intra and extraventricular*; (e) *paraventricular*; and (f) *purely intraventricular* [60]. On the other hand, *Samii* et al. classified craniopharyngiomas into grades based on their vertical projections: *grade I* (intrasellar or infradiaphragmatic), *grade II* (cisternal with or without an intrasellar component), *grade III* (lower half of the third ventricle), *grade IV* (upper half of the third ventricle), and *grade V* (reaching the septum pellucidum or lateral ventricles) [51]. On another perspective, according to the relationships between the tumor, the arachnoid and the pia mater, *Ciric and Cozzens* classified craniopharyngiomas into different types, i.e., *intra-pial intraventricular*, *intra-pial intra-arachnoidal*, *extra-pial intra-arachnoidal* (invasive variant), *extra-pial extra-arachnoidal intrasellar*, *intra-arachnoidal suprasellar* (Dumbbell variant), and *intrasellar extra-arachnoidal* [9]. More recently, *Kassam* et al. proposed a further classification, principally linked with the use of the endoscopic and/or microscopic endonasal pathway, which is based on the relation of the craniopharyngioma with the infundibulum, accordingly, the authors described: *type 1*, *preinfundibular*; *type 2*, *transinfundibular*; *type 3*, *post- or retro-infundibular* (further subdivision is based on rostral or caudal extension, whether it is to the anterior third ventricular (infundibular recess, hypothalamic) and interpeduncular fossa); and *type 4*, *isolated third ventricular* [32]. When dealing with pediatric cases, classification criteria may be different, and, accordingly, Muller and coworkers proposed to classify craniopharyngiomas according to the degree of hypothalamic invasion, using the involvement of the mammillary bodies as a landmark of distinction between anterior and posterior hypothalamic implications [45].

Historically, regarding surgical resection of a craniopharyngioma, it has to be remained that the first surgical attempt was performed by A. E. Halstead [25], and, subsequently, Lewis [39], Cushing [12], and Schloffer [52] removed craniopharyngiomas using either transcranial or transnasal approach. However, craniopharyngiomas remain one of the most challenging intracranial tumors because of the everlasting controversy about the most appropriate surgical approach for each case. A major reason for this controversy is the enormous variability in the topographical location of the tumor, which can affect one to several compartments, from the sella turcica to the third ventricle, as well as its unpredictable degree of adherence [46]. As a consequence, many different possible transcranial approaches have been advocated for the management of such tumors (i.e., frontotemporal, subfrontal, supraorbital, transventricular). These pathways have been adopted and evolved through decades in the attempt of achieving better outcome with lower morbidity and mortality rates [9, 40, 50, 60]. Accordingly, variable modifications and combinations of these approaches have been used for resection of giant or extensive craniopharyngiomas, with the presumption that sufficient exposure of all parts of the tumor is essential for its safe and complete removal [23].

Surgical resection by means of the widely used frontotemporal approach provides the shortest path to the suprasellar space. This route has been accepted throughout the years as the standard technique for the surgical management of craniopharyngiomas, and its effectiveness has rendered it worldwide approved in the routinely neurosurgical practice [50, 60]. This safe and simple approach can be successfully used in craniopharyngiomas with a wide spectrum of extensions; it provides adequate access to the tumor and enables its complete removal with a reasonable morbidity and approach-related complication rate [23]. Many variations of this traditional approach have been used for lesions with hypothalamic extension. On the other hand, in the last decades, the evolution of surgical techniques has led to a progressive reduction of the invasiveness of any neurosurgical approaches, and, according to the keyhole concept, the supraorbital eyebrow route has been validated as an alternative to the conventional transcranial pathways. It allows bilateral and wide surgical exposition, offering the same possibilities with low approach-related morbidity, imputable to the minimal brain retraction obtained with this approach [19, 48].

On another perspective, besides transcranial approaches, transsphenoidal route has also been initially recommended for the craniopharyngiomas that are located within an enlarged sella without calcification and adhesion to parasellar structures. As a matter of fact, this route was introduced according to the indications defined by Guiot and Derome in the early 1960s [24], being proposed only for infradiaphragmatic lesions, with enlarged sella, that preferably already developed panhypopituitarism. Hereinafter, extended transsphenoidal approaches have been introduced for craniopharyngioma even with a significant suprasellar component. Weiss in 1987 [58] termed and originally described the extended transsphenoidal approach, intending a transsphenoidal approach with removal of additional bone along the tuberculum sellae and the posterior planum sphenoidale, between the optic canals, with subsequent opening of the dura mater above the diaphragma sellae. This novel pathway allows midline access and visibility to the suprasellar space from below, obviating brain retraction, and makes possible to manage transsphenoidally midline located suprasellar lesions, traditionally approached transcranially, namely, anterior cranial fossa meningiomas and craniopharyngiomas. First of all such procedures were done with the aid of the microscope [18, 33, 58]. Subsequently, endoscopy has contributed to the more contemporary knowledge and development of the possibilities of the transsphenoidal approach [5, 7, 13, 20, 22, 32, 34]. The wider and panoramic visualization given by the endoscope, and the rapid growth of neuroradiological diagnostic techniques together with the intraoperative neuronavigation systems, augmented the possibility of the transsphenoidal approach, thus allowing its extension toward different areas of the midline skull base. Accordingly, as craniopharyngiomas are often located at the midline, the endonasal pathway offers the advantage of accessing the tumor upon dural opening, without brain or optic nerve retraction and with a direct view through a straight surgical route, even if in a reverse modality as that obtained with the traditional transcranial approaches. Specifically, in recent years, the endoscopic endonasal approach has enabled to overcome many disadvantages of the microsurgical transsphenoidal route to the sella, permitting the management of different purely supra- and retrosellar cystic/solid craniopharyngiomas, regardless to the sellar size or the pituitary function.

Generally, craniopharyngiomas amenable to treat via an endonasal approach should possess key features, such as median midline location, absence of any solid parasellar component, and/or encasement of the main vascular structures. The transsphenoidal approach can avoid transcranial surgery with its inherent risks. However, especially when performing extended approaches, the possible risk of postoperative CSF leak and meningitis has to be highlighted. The validity of the endoscopic endonasal technique for the treatment of such featured craniopharyngiomas has been confirmed throughout recent surgical series, appeared in the pertinent literature [7, 13, 20, 34, 38].

At any rate, despite of the advancements in neurosurgical procedures, techniques, materials, and instrumentations, irregular margins, and tendency to adhere to the nearby vital neurovascular compartment, craniopharyngiomas resection continues to be a surgical challenge [43]. Relationships between the tumor and the surrounding nervous structures, in particular the third ventricle, optic pathways, the pituitary stalk, and major arteries and veins, may be predictive of a difficult surgical removal. Nowadays, the advantage of neuroimaging has led to a better knowledge of intricate relationship between craniopharyngiomas, hypothalamus, pituitary stalk, and optic apparatus, hence ensuring proper selection of surgical approaches.

As a matter of fact, historically, although patients with large craniopharyngiomas involving the third ventricle usually showed symptoms of hypothalamic derangement, such as increased weight gain, impaired sexual function, abnormal somnolence, unexplained high body temperature, inappropriate emotional responses, and/or defective memory, these disturbances were largely ignored or not linked to the anatomical involvement of the hypothalamus by the lesion [46]. Therefore, in the past years, initial surgical

approaches for such kind of neoplasm were designed with the primary aim of radical tumor removal and relief of chiasm compression caused by the tumor [46], being unaware of preoperative and eventually postoperative hypothalamic-related symptoms.

As a consequence, proper treatment of every craniopharyngioma remains to be found, and even if total surgical resection has to be thought as the gold standard, several other options and techniques can be taken into account for the optimal general management of craniopharyngiomas. Cyst drainage [54, 55], wide marsupialization of the cysts into CSF spaces (cysto-ventriculocisternostomy) with neuroendoscopic technique [14], stereotactic aspiration with the instillation of bleomycin or interferon-alpha [2, 41], and endocavitary irradiation [15] have been proposed for the treatment of cystic components of craniopharyngiomas.

When approaching pediatric patients, given that total tumor excision at the first attempt, whenever possible, is the preferred strategy for these difficult neoplasms, it may be suggested to accept the risk of a subtotal surgical resection—above all when hypothalamus is involved—in order to allow adequate neuropsychic and motor development and reduce the risk of hypothalamic injuries [10, 49].

Owing these data, the management of craniopharyngioma patients requires interdisciplinary cooperation of different expertise of the cogent disciplines and should be reserved to specialized centers.

The attempt of total removal is the most suitable aim of the surgical treatment, with lower morbidity and mortality: nowadays, preservation of the quality of life and neurocognitive functioning as long-term survival maintenance are important aspects to be considered. In particular, apart from the pre- or postoperative neurological disorders, hypothalamic obesity can lead to a significant decline in the quality of life and should be taken into consideration in the follow-up of patients with craniopharyngiomas. These aspects concerning the general management of craniopharyngiomas can be thought as "modern treatment targets."

In conclusion, craniopharyngiomas remain one of the most challenging intracranial tumors requiring patience, flexibility, surgical insight, and diligent postoperative management [6]. In every case, treatment should be patient-tailored according to age, presenting symptoms, tumor characteristics, prior treatment, treatment tolerance, and comorbidities.

References

1. Babinski J (1900) Tumeur du corps pituitaire sans acrome´galie et avec arreˆt de de´veloppement des organes genitaux. Revue neurologique, Paris
2. Backlund EO, Axelsson B, Bergstrand CG, Eriksson AL, Noren G, Ribbesjo E et al (1989) Treatment of craniopharyngiomas–the stereotactic approach in a ten to twenty-three years' perspective. I. Surgical, radiological and ophthalmological aspects. Acta Neurochir (Wien) 99:11–19
3. Boyce R, Beadles CF (1893) A further contribution to the study of the pathology of the hypophysis cerebri. J Pathol 1:359–383
4. Bunin GR, Surawicz TS, Witman PA, Preston-Martin S, Davis F, Bruner JM (1998) The descriptive epidemiology of craniopharyngioma. J Neurosurg 89:547–551
5. Cappabianca P, Cavallo LM, Esposito F, De Divitiis E (2008) Craniopharyngiomas. J Neurosurg 109:1–3
6. Cappabianca P, Cavallo LM, Solari D, Di Somma A, Del Basso De Caro ML (2015) Introduction and outcome. In: Evans JJ, Kenning TJ (eds) Craniopharyngiomas: a comprehensive guide to diagnosis, treatment, and outcome. Elsevier, Amsterdam, pp 137–153
7. Cavallo LM, Frank G, Cappabianca P, Solari D, Mazzatenta D, Villa A et al (2014) The endoscopic endonasal approach for the management of craniopharyngiomas: a series of 103 patients. J Neurosurg 121(1):100–113
8. Cavallo LM, Prevedello DM, Solari D, Gardner PA, Esposito F, Snyderman CH et al (2009) Extended endoscopic endonasal transsphenoidal approach for residual or recurrent craniopharyngiomas. J Neurosurg 111:578–589
9. Ciric IS, Cozzens JW (1980) Craniopharyngiomas: transsphenoidal method of approach – for the virtuoso only? Clin Neurosurg 27:169–187
10. Cohen M, Bartels U, Branson H, Kulkarni AV, Hamilton J (2013) Trends in treatment and outcomes of pediatric craniopharyngioma, 1975–2011. Neuro Oncol 15:767–774
11. Crotty TB, Scheithauer BW, Young WF Jr, Davis DH, Shaw EG, Miller GM et al (1995) Papillary craniopharyngioma: a clinicopathological study of 48 cases. J Neurosurg 83:206–214
12. Cushing H (1932) Intracranial tumors. Charles C Thomas, Baltimore

13. de Divitiis E, Cappabianca P, Cavallo LM, Esposito F, de Divitiis O, Messina A (2007) Extended endoscopic transsphenoidal approach for extrasellar craniopharyngiomas. Neurosurgery 61:219–227; discussion 228
14. Delitala A, Brunori A, Chiappetta F (2004) Purely neuroendoscopic transventricular management of cystic craniopharyngiomas. Childs Nerv Syst 20: 858–862
15. Derrey S, Blond S, Reyns N, Touzet G, Carpentier P, Gauthier H et al (2008) Management of cystic craniopharyngiomas with stereotactic endocavitary irradiation using colloidal 186Re: a retrospective study of 48 consecutive patients. Neurosurgery 63:1045–1052; discussion 1052–1043
16. Erdheim J (1904) Uber hypophysenganggeschwwuste und hirncholesteatome. Gerold, Wien
17. Fahlbusch R, Honegger J, Paulus W, Huk W, Buchfelder M (1999) Surgical treatment of craniopharyngiomas: experience with 168 patients. J Neurosurg 90:237–250
18. Fahlbusch R, Honneger J, Buchfelder M (2000) Transsphenoidal microsurgery for craniopharyngiomas. In: Schmidek HH (ed) Shmidek & sweet operative neurosurgical techniques. Indications, methods and results, vol 1. W. B. Saunders, Philadelphia, pp 503–511
19. Fatemi N, Dusick JR, de Paiva Neto MA, Malkasian D, Kelly DF (2009) Endonasal versus supraorbital keyhole removal of craniopharyngiomas and tuberculum sellae meningiomas. Neurosurgery 64:269–284; discussion 284–266
20. Frank G, Pasquini E, Doglietto F, Mazzatenta D, Sciaretta V, Farneti G et al (2006) The endoscopic extended transsphenoidal approach for craniopharyngiomas. Neurosurgery 59(suppl 1):ONS75–ONS83
21. Frohlich A (1901) Ein fall von tumor der hypophysis cerebri ohne akromegalie. Wien klin Rdsch
22. Gardner PA, Prevedello DM, Kassam AB, Snyderman CH, Carrau RL, Mintz AH (2008) The evolution of the endonasal approach for craniopharyngiomas. J Neurosurg 108:1043–1047
23. Gerganov V, Metwali H, Samii A, Fahlbusch R, Samii M (2014) Microsurgical resection of extensive craniopharyngiomas using a frontolateral approach: operative technique and outcome. J Neurosurg 120:559–570
24. Guiot G, Derome P (1972) Indications for transsphenoid approach in neurosurgery. 521 cases. Ann Med Interne (Paris) 123:703–712
25. Halstead AE (1910) Remarks on the operative treatment of 256 tumors of the hypophysis. Surg Gynecol Obstet 10:494–502
26. Hoffman HJ, De Silva M, Humphreys RP, Drake JM, Smith ML, Blaser SI (1992) Aggressive surgical management of craniopharyngiomas in children. J Neurosurg 76:47–52
27. Hofmann BM, Kreutzer J, Saeger W, Buchfelder M, Blumcke I, Fahlbusch R et al (2006) Nuclear beta-catenin accumulation as reliable marker for the differentiation between cystic craniopharyngiomas and rathke cleft cysts: a clinico-pathologic approach. Am J Surg Pathol 30:1595–1603
28. Holsken A, Kreutzer J, Hofmann BM, Hans V, Oppel F, Buchfelder M et al (2009) Target gene activation of the Wnt signaling pathway in nuclear beta-catenin accumulating cells of adamantinomatous craniopharyngiomas. Brain Pathol 19:357–364
29. Hussain I, Eloy JA, Carmel PW, Liu JK (2013) Molecular oncogenesis of craniopharyngioma: current and future strategies for the development of targeted therapies. J Neurosurg 119:106–112
30. Jane JA Jr, Laws ER (2006) Craniopharyngioma. Pituitary 9:323–326
31. Karavitaki N (2014) Management of craniopharyngiomas. J Endocrinol Invest 37(3):219–228
32. Kassam AB, Gardner PA, Snyderman CH, Carrau RL, Mintz AH, Prevedello DM (2008) Expanded endonasal approach, a fully endoscopic transnasal approach for the resection of midline suprasellar craniopharyngiomas: a new classification based on the infundibulum. J Neurosurg 108:715–728
33. Kitano M, Taneda M (2009) Extended transsphenoidal surgery for suprasellar craniopharyngiomas: infrachiasmatic radical resection combined with or without a suprachiasmatic trans-lamina terminalis approach. Surg Neurol 71(3):290–298
34. Koutourousiou M, Gardner PA, Fernandez-Miranda JC, Tyler-Kabara EC, Wang EW, Snyderman CH (2013) Endoscopic endonasal surgery for craniopharyngiomas: surgical outcome in 64 patients. J Neurosurg 119(5):1194–1207
35. Kurosaki M, Saeger W, Ludecke DK (2001) Immunohistochemical localisation of cytokeratins in craniopharyngioma. Acta Neurochir (Wien) 143:147–151
36. Larkin SJ, Ansorge O (2013) Pathology and pathogenesis of craniopharyngiomas. Pituitary 16:9–17
37. Larkin SJ, Preda V, Karavitaki N, Grossman A, Ansorge O (2014) BRAF V600E mutations are characteristic for papillary craniopharyngioma and may coexist with CTNNB1-mutated adamantinomatous craniopharyngioma. Acta Neuropathol 127:927–929
38. Leng LZ, Greenfield JP, Souweidane MM, Anand VK, Schwartz TH (2012) Endoscopic, endonasal resection of craniopharyngiomas: analysis of outcome including extent of resection, cerebrospinal fluid leak, return to preoperative productivity, and body mass index. Neurosurgery 70:110–123; discussion 123–114
39. Lewis D (1910) A contribution to the subject of tumors of the hypophysis. JAMA 55:1002–1008
40. Liu JK, Christiano LD, Gupta G, Carmel PW (2010) Surgical nuances for removal of retrochiasmatic craniopharyngiomas via the transbasal subfrontal translamina terminalis approach. Neurosurg Focus 28, E6
41. Liu W, Fang Y, Cai B, Xu J, You C, Zhang H (2012) Intracystic bleomycin for cystic craniopharyngiomas in children (abridged republication of cochrane systematic review). Neurosurgery 71:909–915

42. Minamida Y, Mikami T, Hashi K, Houkin K (2005) Surgical management of the recurrence and regrowth of craniopharyngiomas. J Neurosurg 103:224–232
43. Mortini P, Gagliardi F, Boari N, Losa M (2013) Surgical strategies and modern therapeutic options in the treatment of craniopharyngiomas. Crit Rev Oncol Hematol 88(3):514–529
44. Mott FW, Barret JO (1899) Three cases of tumor of the third ventricle. Arch Neurol 1:417–440
45. Muller HL, Gebhardt U, Teske C, Faldum A, Zwiener I, Warmuth-Metz M et al (2011) Post-operative hypothalamic lesions and obesity in childhood craniopharyngioma: results of the multinational prospective trial KRANIOPHARYNGEOM 2000 after 3-year follow-up. Eur J Endocrinol 165:17–24
46. Pascual JM, Prieto R, Castro-Dufourny I, Carrasco R, Strauss S, Barrios L (2014) Development of intracranial approaches for craniopharyngiomas: an analysis of the first 160 historical procedures. Neurosurg Focus 36, E13
47. Paulus W, Stockel C, Krauss J, Sorensen N, Roggendorf W (1997) Odontogenic classification of craniopharyngiomas: a clinicopathological study of 54 cases. Histopathology 30:172–176
48. Reisch R, Perneczky A (2005) Ten-year experience with the supraorbital subfrontal approach through an eyebrow skin incision. Neurosurgery 57:242–255
49. Sainte-Rose C, Puget S, Wray A, Zerah M, Grill J, Brauner R et al (2005) Craniopharyngioma: the pendulum of surgical management. Childs Nerv Syst 21:691–695
50. Samii M, Samii A (2000) Surgical management of craniopharyngiomas. In: Schmidek HH (ed) Schmidek & sweet operative neurosurgical techniques. Indications, methods and results, vol 1. W. B. Saunders, Philadelphia, pp 489–502
51. Samii M, Tatagiba M (1997) Surgical management of craniopharyngiomas: a review. Neurol Med Chir (Tokyo) 37:141–149
52. Schloffer H (1907) Erforgleiche operationen eines hypophysentumors auf nasalem wege. Wien Klin Wochenschr 20:621–624
53. Sekine S, Shibata T, Kokubu A, Morishita Y, Noguchi M, Nakanishi Y et al (2002) Craniopharyngiomas of adamantinomatous type harbor beta-catenin gene mutations. Am J Pathol 161:1997–2001
54. Spaziante R, de Divitiis E (1997) Drainage techniques for cystic craniopharyngiomas. Neurosurg Quart 7:183–208
55. Spaziante R, de Divitiis E, Irace C, Cappabianca P, Caputi F (1989) Management of primary or recurring grossly cystic craniopharyngiomas by means of draining systems. Topic review and 6 case reports. Acta Neurochir (Wien) 97:95–106
56. Tateyama H, Tada T, Okabe M, Takahashi E, Eimoto T (2001) Different keratin profiles in craniopharyngioma subtypes and ameloblastomas. Pathol Res Pract 197:735–742
57. Van Effenterre R, Boch AL (2002) Craniopharyngioma in adults and children: a study of 122 surgical cases. J Neurosurg 97:3–11
58. Weiss MH (1987) The transnasal transsphenoidal approach. In: Apuzzo MLJ (ed) Surgery of the third ventricle. Williams & Wilkins, Baltimore, pp 476–494
59. Xin W, Rubin MA, McKeever PE (2002) Differential expression of cytokeratins 8 and 20 distinguishes craniopharyngioma from rathke cleft cyst. Arch Pathol Lab Med 126:1174–1178
60. Yasargil MG, Curcic M, Kis M, Siegenthaler G, Teddy PJ, Roth P (1990) Total removal of craniopharyngiomas. Approaches and long-term results in 144 patients. J Neurosurg 73:3–11
61. Zenker FA (1857) Enorme Cystenbildung im Gehirn, vom Hirnanhang ausgehend. Arch Pathol Anat Physiol Klin Med 12:454–466