Cystic lesions of the sellar-suprasellar region — diagnosis and treatment


Abstract

The differentiation of cystic lesions located in the sellar-suprasellar region is a significant problem in clinical practice because of the similarities in their clinical, radiological, and even histopathological picture. Arriving at the right diagnosis is vital for taking appropriate therapeutic decisions.

The most frequent clinical manifestation of lesions located in the sellar-suprasellar region is headache. It often co-exists with symptoms of anterior pituitary gland insufficiency or hyperprolactinaemia caused by compression of the pituitary stalk. Diabetes insipidus, obesity, mental disorders, and circadian rhythm disorders may be associated with lesions penetrating the suprasellar space. It is extremely important to rule out the possible coexistence of pituitary microadenoma and Rathke’s cleft cyst, which became possible with the use of 11C-methionine positron emission tomography/computed tomography (C-MET PET/CT). Reports from literature indicate that pituitary microadenoma may coexist with Rathke’s cleft cyst in 10% of patients. Cystic lesions of the sellar-suprasellar region should also be differentiated from a cystic pituitary adenoma or abscess.

The first-choice therapy in symptomatic cystic lesions of the sellar-suprasellar region is neurosurgery, which usually relieves headache and improves vision impairment, while less frequently restores normal pituitary function. In suprasellar lesions, neurosurgery may trigger or aggravate pre-existing symptoms of damage to the hypothalamus. Patients undergoing neurosurgery for cystic lesions located in the sellar-suprasellar region should be monitored for a few years due to their high recurrence rate, potential malignant transformation of these lesions, and possible adenoma development through metaplasia. The advent of targeted therapy of the BRAF/MEK pathway is associated with new therapeutic opportunities for patients with craniopharyngiomas. (Endokrynol Pol 2018; 69 (2): 212–220)

Key words: cystic lesion, sellar-suprasellar region, diagnosis, treatment

Introduction

The diagnosis and treatment of cystic lesions located in the sellar-suprasellar region requires an interdisciplinary approach with the participation of a radiologist, ophthalmologist, endocrinologist, and a neurosurgeon. These lesions have been detected increasingly more often since various imaging techniques became more accessible, which also means that it is necessary to differentiate incidentalomas from lesions with serious clinical consequences. Determination of the cause of the observed lesion is crucial for choosing the right treatment option.

Management of Rathke’s cleft cysts (RCC) with benign course differs from that of RCC-mimicking craniopharyngiomas, which are locally aggressive [1]. Apart from the aforementioned Rathke’s cleft cysts and craniopharyngiomas, cystic lesions of the sellar-suprasellar region include arachnoid cysts, secondary empty sella syndrome, and cystic adenomas, as well as less frequent xanthogranulomas, and epidermoid cysts [2]. Cystic lesions of the sellar-suprasellar region have not been described in Polish literature so far. In foreign literature the reports are also few, and they lack a comprehensive approach.

The aim of this paper is to highlight the problem of cystic lesions located in the sellar-suprasellar region in the context of their epidemiology, etiopathogenesis, diagnosis, and therapy.

Rathke’s cleft cyst

Epidemiology

Rathke’s cleft cyst is the most frequent incidentaloma of the sellar-suprasellar region. Its incidence based on autopsy is approximately 20% [3]. Symptomatic Rathke’s cleft cysts are, however, uncommon. Their peak incidence falls between the fourth and the sixth decade of life. They are three times more frequent among women than among men [1].

Karol Sagan, Department of Endocrinology, Metabolic Diseases and Internal Diseases, Pomeranian Medical University, ul. Unii Lubelskiej 1, 71–252 Szczecin, tel.: 509–541–633, e-mail: karolsagan@vp.pl
**Etiopathogenesis**

Rathke’s cyst arises from the craniopharyngeal duct. In embryogenesis, cells of the anterior and the posterior wall of the pouch give rise to the anterior pituitary and pars intermedia, respectively. These structures come into contact with the infundibulum, which is made of nervous tissue. When the lumen of the pouch forms a cleft that fails to regress, it leaves a space filled with fluid between the pars nervosa and the ectoderm. Hence, Rathke’s cleft cyst is found in pars intermedia between the anterior and posterior lobes of the pituitary gland. In some cases, Rathke’s cleft cyst also spreads to the suprasellar space.

In approximately 50% of cases, inflammation around the cyst is observed, which can be visualised on magnetic resonance imaging (MRI) [4]. Autopsies also showed the presence of cyst wall metaplasia in 9–39% of patients [2].

**Clinical picture**

Clinical symptoms associated with Rathke’s cleft cyst are caused by elevated pressure in the perisellar region. The most common clinical manifestation is headache. The pain is usually non-pulsating, episodic, and located in the frontal or in the retro-orbital region [5]. Headaches may however have a non-specific location and nature. If the cyst is large, it may compress the optic chiasm resulting in vision impairment [6, 7].

Although Rathke’s cleft cyst is mostly diagnosed incidentally, endocrine dysfunction is found in 80% of patients with symptomatic lesions [3]. Laboratory deviations usually include hyperprolactinaemia and insufficiency of the gonadal, the adrenal, and less frequently of the thyroid axis [8]. These disorders are rather associated with features of inflammation visualised on MRI than with the size of the cyst. In rare cases of lesions penetrating the suprasellar space, diabetes insipidus is also observed. The most common complaints related to endocrine dysfunction include secondary amenorrhoea, reduced libido, and impotence [1].

**Diagnostic imaging**

Rathke’s cyst is visualised on magnetic resonance imaging as a lesion located in the intermediate lobe of the pituitary. Its largest diameter usually does not exceed 20 mm [9, 10]. The cyst may have varying intensity against the cerebrospinal fluid, although it is more often hypointense in T1-weighted images and hyperintense in T2-weighted images. RCC image captured on MRI is described as “an egg in a shell” [11]. The enhancement of the cyst wall following contrast administration suggests inflammation or metaplasia.

**Treatment**

It is assumed that symptomatic lesions require excision, although disease dynamics should be taken into account when making the decision. The removal of Rathke’s cleft cyst is associated with a high risk of diabetes insipidus as a consequence of frequently observed adherence of the cyst walls to the infundibulum. If signs of hypopituitarism are present prior to surgery, they usually persist after neurosurgical treatment.

Patients should be monitored for at least five years following surgery because the risk of recurrence may even reach 50%. This risk is particularly high when inflammation around the cyst is observed. It has been demonstrated that antibiotic therapy may reduce the risk of recurrence in such cases [12]. Radiotherapy may prove effective in the treatment of recurrent Rathke’s cleft cysts, although available evidence is insufficient.

Asymptomatic Rathke’s cleft cysts require observation, although its duration has not been established so far. It is important to remember that a growing number of studies indicate a possible transformation of metastatic Rathke’s cleft cyst into craniopharyngioma [4, 13, 14]. Based on the work of Raluca Trifanescu et al. [1], we have proposed an up-to-date diagnostic and therapeutic algorithm for patients with Rathke’s cleft cyst (Fig. 1).

**Coexistence of Rathke’s cleft cyst with pituitary adenoma**

Recent studies have shown the presence of stem cells in the pituitary and their role in tumourogenesis of the sellar-suprasellar region [15]. Cells related to neoplastic changes, co-expressing cytokeratins, GFAP and S-100, have been found in Rathke’s cleft cyst. The occurrence of cells with a similar phenotype in pituitary adenomas seems to be associated with invasiveness and recurrence of tumours [16].

Several attempts have been made so far to determine the coexistence of pituitary adenomas with Rathke’s cleft cysts [17]. It is a challenge because an adenoma located in a pituitary gland compressed by the cyst often remains undetectable on MRI. Retrospective studies found no statistically significant concurrence of these pathologies [18]. However, Hidetoshi Ikeda and Genichiro Ohhashi, who prospectively analysed 308 patients, demonstrated concomitant occurrence of an adenoma in 34% of inflammatory Rathke’s cleft cysts [19]. Most adenomas (74%) were visualised in 11C-methionine positron emission tomography/computed tomography [C-MET PET/CT], while other adenomas were diagnosed in histopathological examination. It is worth noticing that the most common coexistent adenomas included GH-omas and ACTH-omas. It supports
the hypothesis that these lesions might be associated with metaplasia of cells in the anterior pituitary, as a result of inflammation around the cyst. Further studies also confirmed high sensitivity of C-MET PET/CT in the detection of pituitary microadenomas invisible on MRI [20].

Also, 18F-deoxy-glucose positron emission tomography combined with computed tomography (18F-FDG PET/CT) sometimes reveals benign lesions of the sellar-suprasellar region [21]. However, the low sensitivity of this method for detecting pituitary adenomas limits its utility in diagnosing lesions undetectable on MRI [20].

**Craniopharyngioma**

**Epidemiology**

The incidence of craniopharyngioma is approx. 1–2/1,000,000/year. Peak incidence is observed twice during the lifetime: in the 1st and the 2nd decade and between 50 and 70 years of age [22]. Similarities between Rathke's cleft cyst and craniopharyngioma may be so strong that differentiating one from the other is only possible in postoperative immunohistochemical examination. Moreover, there are lesions with coexistent features characteristic for both of these pathologies, which may be due to the fact that they share a common origin.
**Etiopathogenesis**

Craniopharyngiomas occur in two histopathological variants: adamantinomatous and papillary. The adamantinomatous variant occurs both in children and adults, while the papillary variant is characteristic of the adult population. Recent studies have shown that histopathological differences are caused by different genetic mutations.

In adamantinomatous craniopharyngiomas, CTNNB1 mutation was detected. It is responsible for β-catenin translocation from cell membrane to the nucleus. The incidence of this mutation among adamantinomatous craniopharyngiomas is estimated at 81–100% based on genetic tests and immunohistochemical staining [23, 24].

BRAF V600E mutation is characteristic of the papillary variant of craniopharyngioma. Its occurrence can be confirmed with immunohistochemical staining using VE1 monoclonal antibody, detecting BRAF V600E protein epitope [24]. The incidence of this mutation in papillary craniopharyngioma is estimated at 95–100%. The existence of craniopharyngioma types combining the features of adamantinomatous and papillary variants is still being discussed [25].

Despite a few theories on the development of craniopharyngiomas, none of them has been proven to date. In their recently published study, Liu Y. et al. demonstrated the existence of triggering receptors exposed on myeloid cells-1 (TREM-1) in papillary craniopharyngiomas and in Rathke’s cleft cysts with squamous metaplasia, but not in adamantinomatous craniopharyngiomas [26]. This finding supports the hypothesis of sequential transformation of the sellar-suprasellar cyst lesions [2]. In this approach, Rathke’s cleft cyst is on one end of a pathologic continuum and papillary craniopharyngioma on the other, while mixed-type lesions such as xanthogranulomas fall between these two pathologies. The adamantinomatous variant is likely to originate from the remnants of the craniopharyngeal duct, similarly to Rathke’s cleft cysts. This would account for its common occurrence in children.

A recently published study demonstrated that immunohistochemical staining using VE1 allowed the diagnosis of Rathke’s cleft cyst to be changed to that of papillary craniopharyngioma in three out of 33 cases. Thus, immunohistochemical staining using VE1 appears to have great clinical significance and should be performed particularly in sellar-suprasellar cyst lesions with squamous metaplasia [27].

**Clinical picture**

Clinical symptoms usually resemble those observed in symptomatic Rathke’s cleft cyst. However, since craniopharyngiomas at their early stage of development usually remain clinically silent, they are diagnosed when their size is large, causing symptoms associated with the compression of structures forming the floor of the third ventricle and elevation of intracranial pressure [28]. Thus, symptoms related to craniopharyngioma result from the mass effect and raised pressure in the intrasellar space. Just as in Rathke’s cleft cysts, vision impairment (62–84%), headache, and endocrine dysfunction (52–87%) occur and the symptom characteristics are not different from those observed in Rathke’s cleft cyst. However, symptoms caused by compression of hypothalamic structures, including secondary amenorrhoea, obesity, circadian rhythm disturbances, and cardiovascular disorders, are much more frequent than in patients with Rathke’s cleft cyst [29–31]. They are difficult to treat, and their incidence and severity may increase during therapy from approx. 30% to as many as 80% of cases [32]. This affects the quality of patients’ lives [30]. Patients with craniopharyngioma also present with neuropsychiatric changes [33], including short-term memory loss related to the compression of mammillary bodies. Complaints of impaired memory may be reported by as many as 50% of patients and they are more common among older subjects. Patients with delayed diagnosis may present with hydrocephalus [34–36]. Craniopharyngiomas are often responsible for central diabetes insipidus as a result of damage to subthalamic nuclei or the upper part of the pituitary stalk, just above the sellar diaphragm. Lesions restricted to the intrasellar space do not cause neurogenic diabetes insipidus [37]. In some cases, diabetes insipidus may have an insidious course with symptoms masked by secondary hypothyroidism and adrenal failure. In such cases, fluid retention resulting from anterior pituitary failure compensates for water loss caused by vasopressin deficiency [38–40]. Polyuria and polydipsia develop once hormone substitution is started.

Additionally, diabetes insipidus without excessive thirst, so-called adipsic central diabetes insipidus, constitutes a serious risk for patients. Its rare occurrence is found in cases in which proliferative process damages osmoreceptors in the anterior part of the hypothalamus [41]. This suppresses the feeling of thirst, increases plasma osmolality, and produces hypernatraemia. The condition ultimately leads to reduced brain volume and intracranial or subarachnoid haemorrhage [42]. However, central diabetes insipidus in patients with craniopharyngioma most commonly develops as a postoperative complication, being the consequence of pituitary stalk transection. Triphasic response to this damage is an additional challenge in the diagnosis and therapy [43]. Initially, during the first few days after surgery, clinical symptoms

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Intrasellar arachnoid cyst: primary empty sella syndrome

Epidemiology
Arachnoid cysts are rarely reported in literature. Between 1980 and 2007 only 51 cases with surgical treatment and confirmed histopathological diagnosis were described [51]. The mean age at diagnosis is 46 years [52].

Etiopathogenesis
Intrasellar arachnoid cysts are likely to develop as a consequence of wide aperture of the sellar diaphragm. Autopsies showed that only in 42% this opening is narrow enough to tightly encircle the infundibulum [53]. It was also demonstrated that the intrasellar space occupied by the pituitary decreases with age. Both these conditions result in sellar penetration by subarachnoid space filled with cerebrospinal fluid. The fluid is thus trapped within the sella as in a one-way valve effect [50]. Elevated intracranial pressure, observed in 50% of patients with primary empty sella syndrome, is also likely to play a role in the pathogenesis [54].

Clinical picture
Clinical symptoms associated with an arachnoid cyst are not particularly different from those observed in other cystic lesions. Arachnoid cysts, like other cysts, cause vision impairment, headache and — slightly less frequently — clinical symptoms of hypopituitarism. The latter most commonly include reduced libido, impotence, menstrual cycle disturbances, and difficulties with conception [50]. Clinically significant endocrine dysfunction usually affects 1–2 axes, although thorough endocrine assessment and pituitary stimulation tests may reveal anomalies in all pituitary axes. The most common finding is insufficiency of the gonadal axis. Furthermore, patients with intrasellar arachnoid cysts more commonly present with depression and obesity [55].

The coexistence of empty sella syndrome and pituitary adenoma has been described in literature [56, 57]. The coincidence of these two pathologies requires further studies. Additionally, some patients with an intrasellar arachnoid cyst present symptoms of depression with elevated ACTH levels, which indicates pseudo-Cushing’s syndrome.

Diagnostic imaging
MRI findings are characteristic and, in most cases, allow for the differentiation between arachnoid cyst and pituitary adenoma. Isointensity or slight hyperintensity of the cyst against cerebrospinal fluid is a characteristic feature. Additionally, the lesion is balloon-shaped,
protrudes towards the suprasellar region, and modu-
lates but does not infiltrate the medial wall of the cav-
ernous sinus [29]. The pituitary stalk is usually clearly
visible and shifted backwards.

**Treatment**
Neurosurgery is indicated in patients with clinical
symptoms. The recurrence rate after surgical treatment
is similar to that observed with Rathke’s cleft cysts.

**Secondary empty sella syndrome**

**Epidemiology**
The incidence of secondary empty sella syndrome is
unknown. Recently, it has been diagnosed more and
more often due to the development of diagnostic tech-
niques and new therapeutic options used for treatment
of lesions located in the sellar-suprasellar region.

**Etiopathogenesis**
Secondary empty sella syndrome may be a consequence
of pituitary adenoma apoplexy, neurosurgery, ra-
diotherapy, or pharmacotherapy [58–60]. Lymphocytic
hypophysitis and Sheehan’s syndrome are rare causes
of secondary empty sella syndrome.

**Diagnostic imaging**
Magnetic resonance imaging shows the picture of an
empty sella. The pituitary gland is atrophic and its po-
position within the sella is asymmetric. It pulls the stalk,
which is shifted beyond the median line. The signs of
displacement are not observed in secondary empty
sella syndrome as appears from the absence of pituitary
compression.

**Clinical picture**
Clinical symptoms depend on the size and activity of
the adenoma in which the necrosis developed. A char-
acteristic group of patients with empty sella syndrome
includes patients with clinical symptoms of acromegaly
with GH and IGF-1 levels falling within normal ranges.
These are patients who recovered spontaneously from
adenoma apoplexy. In some cases, necrosis of the tu-
mour is not complete and the adenoma still shows some
hormonal activity [61].

In cases of classic apoplexy, the incidence of hy-
opituitarism is high [62]. Additionally, higher risk of
GH-oma and prolactinoma occurrence in a compressed
pituitary has been described [63]. These lesions may
remain undetectable on MRI.

**Epidermoid cyst**

**Epidemiology**
Epidermoid cysts are typically found in the cerebello-
ponpine angle, although they may occasionally develop
in the sellar-suprasellar region. They account for less
than 1% of sellar-suprasellar cystic lesions [2].

**Etiopathogenesis**
Like in Rathke’s cleft cysts and craniopharyngiomas,
there are two hypotheses related to the development
of epidermoid cysts. The first of them assumes a com-
mon origin of epithelial lesions from the remnants of
the craniopharyngeal duct, while according to the
second hypothesis, called the metaplastic theory, these
lesions develop as a consequence of the transforma-
tion of intrasellar space epithelium triggered by an inflam-
atory process.

**Clinical picture**
Epidermoid cysts produce symptoms associated with
increased intracranial pressure — mainly headache, and
with the compression of optic chiasm, such as impair-
ment of visual acuity and restricted vision field [64].
Cases of pituitary apoplexy were also reported in
patients with epidermoid cyst located in the sellar-
suprasellar region.

**Diagnostic imaging**
MRI typically shows a hyperintense lesion in T1- and
T2-weighted images.

**Treatment**
Surgery is the only effective method of treatment. Due
to the adherence of epidermoid cyst to nervous struc-
tures, complete resection is often impossible, hence
epidermoid cysts recur in 25% of cases.

**Xanthogranuloma**

**Epidemiology**
Xanthogranulomas are very rare lesions of the sellar-
suprasellar region. In professional literature on the
subject published in English, only a few dozen cases
have been described to date.

**Etiopathogenesis**
The theories of the aetopathogenesis of xanthogranu-
lomas are the same as the ones of epidermoid cysts.

**Clinical picture**
Typical symptoms of cholesterol granulomas include
headache and vision impairment. Patients with xan-
thogranuloma may develop panhypopituitarism.
Table I. Clinical symptoms, endocrine dysfunction and characteristic features on MRI of cystic lesions located in the sellar-suprasellar region

<table>
<thead>
<tr>
<th>Cystic lesion</th>
<th>Most common clinical symptoms*</th>
<th>Endocrine dysfunction</th>
<th>Intensity and characteristic features on MRI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rathke’s cleft cyst</td>
<td>Headache, visual field</td>
<td>Insufficiency of all axes is possible, hyperprolactinemia</td>
<td>Located in intermediate lobe, with largest</td>
</tr>
<tr>
<td></td>
<td>impairment, secondary</td>
<td>Possible coexistence of adenoma, most commonly GH-</td>
<td>diameter—usually &lt; 20 mm, various intensity:</td>
</tr>
<tr>
<td></td>
<td>amenorrhea, impotence,</td>
<td>oma, ACTH-oma, rare diabetes</td>
<td>usually hypointense in T1-weighted images</td>
</tr>
<tr>
<td></td>
<td>reduced libido</td>
<td>insipidus</td>
<td>T1 hypointense and hyperintense in T2-</td>
</tr>
<tr>
<td>Craniopharyngioma</td>
<td>Headache, visual field</td>
<td>Insufficiency of all axes is possible, hyperprolactinemia</td>
<td>Various intensity of the lesion, size of the</td>
</tr>
<tr>
<td></td>
<td>impairment, secondary</td>
<td>(sometimes masked or adipsic DI)</td>
<td>lesion is usually &gt; 20 mm, calcifications</td>
</tr>
<tr>
<td></td>
<td>amenorrhea, obesity,</td>
<td></td>
<td>within the lesion, wall enhancement</td>
</tr>
<tr>
<td></td>
<td>impaired memory, circadian</td>
<td></td>
<td>following contrast administration**,</td>
</tr>
<tr>
<td></td>
<td>rhythm disorders, polyuria,</td>
<td></td>
<td>compression of the 3rd ventricle floor</td>
</tr>
<tr>
<td></td>
<td>polydipsia</td>
<td></td>
<td>structures, solid-cystic components</td>
</tr>
<tr>
<td>Intrasellar arachnoid cyst</td>
<td>Headache, visual field</td>
<td>Insufficiency of all hormonal axes, often subclinical</td>
<td>Isointense against cerebrospinal fluid,</td>
</tr>
<tr>
<td></td>
<td>impairment, loss of libido,</td>
<td></td>
<td>balloon-shaped</td>
</tr>
<tr>
<td></td>
<td>infertility, secondary</td>
<td></td>
<td>lesion protruding towards the suprasellar</td>
</tr>
<tr>
<td></td>
<td>amenorrhea, depression</td>
<td></td>
<td>region</td>
</tr>
<tr>
<td>Secondary empty sella syndrome</td>
<td>Rare, dependent on the</td>
<td>No endocrine dysfunction, hyperpituitarism,</td>
<td>The same as in intrasellar arachnoid cyst</td>
</tr>
<tr>
<td></td>
<td>pathogenesis of cyst</td>
<td>hypopituitarism</td>
<td></td>
</tr>
<tr>
<td>Epidermoid cyst</td>
<td>Headache, vision impairment</td>
<td>Possible hypopituitarism</td>
<td>Picture varies</td>
</tr>
<tr>
<td>Cholesterol granuloma</td>
<td>Headache, vision impairment</td>
<td>Possible hypopituitarism in a few axes</td>
<td>The lesion is usually hyperintense</td>
</tr>
</tbody>
</table>

*not ranked by frequency
**also possible in inflammatory Rathke’s cleft cyst

References

Diagnostic imaging
Cholesterol granuloma cannot be differentiated from Rathke’s cleft cyst or craniopharyngioma on MRI [65].

Treatment
Surgery relieves the symptoms of increased intracranial pressure. A low recurrence rate of xanthogranulomas has been observed in case studies reported so far. In patients with symptomatic hypopituitarism, replacement therapy should be started.

Summary
Patients with cystic lesions of the sellar-suprasellar region require in-depth hormone testing and long-term observation of several years’ duration. It is important to rule out hypopituitarism and the co-existence of a pituitary adenoma that is undetectable on magnetic resonance imaging. In some cases, preoperative diagnostic tests are inconclusive, and it is only postoperative histopathological examination with the use of immunohistochemical staining among other methods that leads to the final diagnosis. Table 1 shows clinical symptoms, endocrine dysfunction, and diagnostic imaging used for lesions located in the sellar-suprasellar region.


