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Surgical Outcomes of Pituitary Apoplexy

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Objective : Pituitary apoplexy is a rare clinical syndrome caused by pituitary hemorrhage, hemorrhagic infarction, or ischemic infarction within a pituitary tumor or surrounding structure. We analyzed surgical outcomes of pituitary apoplexy.

Methods : From 1995 to 2004, we reviewed our experience of 29 cases with pituitary apoplexy. In all patients, pre- and postoperative clinical presentation were checked and endocrine study were performed.

Results : The most frequent symptoms were visual disturbance (24 cases, 82.8%) and headache (22 cases, 75.9%). After surgery, headache improved in 86.4%, 88.9% among 18cases who had preoperative reduction in visual acuity and 75.0% among 12 cases who had preoperative reduction in visual field improved. In endocrine study, long-term steroid and thyroid hormone replacement therapy was necessary in 42.9% of 14 cases presenting preoperative hypopituitarism. Postoperative transient hypopituitarism developed in 5 cases (33.3%) and they all recovered in follow up study. Postoperative endocrinological recovery were in 77.9% of 9 cases with preoperative prolactinoma, 1 case in 2 cases with acromegaly and one case with Cushing disease. Postoperative complications were diabetes inspidus(DI) in 1 case (3.4%), cerebrospinal fluid(CSF) leakage in 2 cases (6.8%) and death in 1 case (3.4%) due to sepsis.

Conclusion : We report good results through surgery of pituitary apoplexy in a clinical and endocrine outcomes. The surgery should be performed as soon as possible to be a suitable method for treating pituitary apoplexy.

KEY WORDS : Pituitary apoplexy · Transsphenoidal approach · Surgical outcome.

Introduction

Pituitary apoplexy is a clinical syndrome inducing sudden headache, reduction in visual field or acuity and change of consciousness level that occurs by rapid expansion of a mass due to hemorrhage, hemorrhagic infarction or ischemic infarction of a pituitary tumor or surrounding structure[4-6,8,9,11,19].

Regarding hemorrhage within pituitary adenoma, the incidence of pituitary apoplexy has been variously reported from 1.5% to 27.7%[22]. It is known to be caused by radiotherapy, head trauma, dynamic pituitary function test or bromocriptine therapy and general anesthesia or stresses by various operations. However most of the causes are still unknown[2,18,20]. The result of several studies suggest that surgery and medical therapy has roles in the optimal treatment of pituitary adenoma[4,7]. With regard to the treatment of pituitary apoplexy, although there is a study suggesting a conservative treatment, it is known that relatively good results are obtained by surgery with medical treatment[2]. Some studies report that conservative treatment somewhat relieved headache and was effective for improvement in the optic nerve, but review of the cases where symptoms and signs improve greatly after surgery, the surgical treatment is considered necessary[16].

We undertook this study to define the immediate (postoperative 24 hours) and long term (3 months after surgery) efficacy of surgery and combined medical therapy. The purpose of this study is to analyze surgical outcomes of pituitary apoplexy and review of literature.

Materials and Methods

The clinical material consisted of 133 cases of pituitary adenoma treated in our institute between January 1995 and March 2004. Total 29 surgical cases(21.8%) were pathologically diagnosed pituitary adenoma with apoplexy. There were no other pathologic finding.

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Table 1. Summary of patients with pituitary apoplexy

Case	Age	Sex	Clinical presentations	Visual Sx.			Endocrine study		Postop. complication
				Acuity	Field defect	Ocular palsy	Preop.	Postop.	
1	56	F	headache	+	–	–	Prolactin : 342[a]	Prolactin : 21[a]	CSF leakage
2	40	F	headache	+	+	–	T4:3.2[e]	–	–
3	55	M	headache	+	–	–	T4:2.9[e]	T4:2.6[e]	–
4	44	M	altered consciousness	–	–	–	Prolactin:782[a]	Prolactin:52[a]	–
5	44	M	headache	+	–	–	Cortisol:1.9[d] T4:2.5[e]	–	CSF leakage
6	44	M	headache	–	–	+	Prolactin:248[a]	Prolactin:24[a]	–
7	55	F	headache	+	+	–	Cortisol:2.2[d] ACTH:104	– ACTH:35[c]	recur
8	40	M	headache	+	+	+	Cortisol:30.4[d]	Cortisol:9.8[d]	–
9	58	M	headache	+	–	–	Cortisol:1.1[d] GH:60[b]	Cortisol:1.6[d] GH:1.4[b]	–
10	45	F	–	+	–	+	IGF-1:1512[b] Cortisol:1.6[d]	IGF-1:315[b] Cortisol: 2.1[d]	recur
11	24	F	headache	–	–	+	T4:2.1[e]	T4:1.9[e]	–
12	38	M	headache	+	–	–	Prolactin:453[a] Cortisol:1.2[d]	Prolactin:12[e] Cortisol:1.9[d]	–
13	65	F	headache	–	–	–	T4:2.1[e]	T4:2.3[e]	–
14	23	M	headache	+	+	–	Prolactin:855[a]	Prolactin:130[a]	–
15	68	F	headache	–	–	–	T4:3.5[e] GH:79[b]	– GH:4.5[b]	–
16	28	M	headache	+	+	–	IGH-1:1194[b]	IGF-1:892[b]	DI
17	61	F	headache	+	–	+	–	–	–
18	26	F	headache	+	+	–	Prolactin:278[a]	Prolactin:12[a]	–
19	44	F	headache	–	+	–	–	–	–
20	22	F	–	–	–	–	Prolactin:269[a]	Prolactin:16[a]	–
21	48	M	headache	+	–	–	Cortisol:2.7[d]	–	–
22	31	F	headache	–	–	–	Prolactin:415[a]	Prolactin:23[a]	–
23	66	F	altered consciousness	–	–	+	Cortisol:2.3[d]	–	death
24	63	F	blindness	+	+	–	T4:2.7[e]	–	–
25	31	M	headache	–	+	–	–	–	–
26	62	F	–	+	+	–	Cortisol:1.7[d]	Cortisol:2.1[d]	–
27	74	M	headache headache	+	–	+	Prolactin:411[a]	Prolactin:13[a]	–
28	37	F	blindness	+	+	+	Cortisol:1.2[d]	Cortisol:0.9[d]	–
29	50	F	–	–	+	–	Cortisol:2.7[d] T4:3.1[e]	–	–

a ; ng/ml , *b* ; ng/mlF, *c* ; pg/ml, *d* ; ug/dl, *e* ; ng/dl ACTH : adrenocorticotropic hormone, Sx : symptom, Preop. : preoperative, Postop. : postoperative, T4 : thyroxine, GH : growth hormone, IGF-1 : insulin-like growth factor, DI : diabetes inspidus, CSF : cerebral spinal fluid, recur : recurrence

We retrospectively reviewed their medical records and investigated their clinical presentations, visual symptoms, endocrine studies, pathologic finding and surgical complications. The recovery degree of visual disturbance was measured by Snellen visual acuity and visual field examination (perimetry) at one week and 3 months after surgery. Complete recovery of visual acuity was a return to baseline acuity before pituitary apoplexy and partial recovery was an improvement of acuity but it didn't return to baseline acuity. The hormone studies were performed by measuring freeT4, T3, TSH, PRL, LH, FSH, GH, IGF-1, cortisol, ACTH and ADH at the time of admission and postoperative 24 hours, 1 week and 3 months.

Results

There were 29 cases who were confirmed as pituitary apoplexy and treated surgically.

Male to female ratio was 1:1.4 (12:17) and the average age was 46.3 (22~74 years old)(Table 1). The average length of follow up was 20 months (3~84 months) and the reoperation was 2 cases (6.9%) due to recurrence after surgery.

Clinical presentation

The time interval from onset of clinical presentation to surgery was various from 1~136 days (average 30 days). Visual disturbance was the most common presenting complaint in 24 cases (83%). Eighteen cases (62%) had reduction in visual acuity, 12 cases (41%) had reduction in visual field and among them, 9 cases (31%) had both symptoms. Headache was presenting complaint in 22 cases (76%), ocular paresis in 8 cases (28%) and altered consciousness in 2 cases (6.9%).

Improvement in symptoms and visual acuities and fields were achieved postoperative one week (Table 2). Headache was recovered in 19 cases (86.4%) and improvement was 3 cases (13.6%) whose mild headache persisted. After postoperative 3 months, there were no further improvement of visual symptom.

From the 16 cases (88.9%) of improved visual acuity, there was 11 cases (61.1%) of complete recovery and 5 cases (27.8%) of partial recovery. There were no improvement in 2 cases with preoperative blindness. The patients who showed visual field recovery was 9 cases (75.0%), among whom 7 cases (58.3%)

showed complete recovery and 2 cases (16.7%) partial recovery. Seven cases (87.5%) showed ocular paresis recovery, among whom 2 cases (25.0%) showed partial recovery and other 5 cases (62.5%) complete recovery. One of 2 cases with altered consciousness improved to normal and the other died due to medical complication of pneumonia after surgery.

Endocrine study

In preoperative hormone study, hypopituitarism was 14 cases (48.3%), normal pituitary function was 3 cases (10.3%), and pituitary hyperfunction was 12 cases (41.4%)(Table 1). Among 14 cases in hypopituitarism, 10 cases (71.4%) appeared low cortisol level, 8 cases (57.1%) presented low free T4 level and among them, 4 cases had both. Nonfunctioning tumor was 17 cases (58.6%), prolactinoma in 9 cases (31.0%), acromegaly in 2 cases (6.9%), Cushing disease in 1 case (3.5%).

Table 2. Visual improvement postoperative one week

	Complete recovery(%)	Partial recovery(%)	No change(%)
Visual acuity	11(61.1)	5(27.8)	2(11.1)
Visual field defect	7(58.3)	2(16.7)	3(25.0)
Ocular palsy	5(62.5)	2(25.0)	1(12.5)

With regard to hormone study at 3 months after surgery, hormone level was recovered to the normal range in 7 cases (77.9%) of prolactinoma and improved but abnormal range in 2 cases (22.1%), one of 2 cases was treated by bromocriptine. One of 2 acromegaly and a Cushing disease were recovered to the normal range but, the other case of acromegaly did not. Among 15 cases which didn't appear pituitary hypopituitarism before surgery, 5 cases (33.3%) showed temporary hypopituitarism. Among of them, 2 cases (13.3%) were hypothyroidism and 3 cases (20.0%) were hypocortisolism but they all recovered in follow up study. Five (50.0%) of 10 cases in hypocortisolism and 3 (37.5%) of 8 cases in hypothyroidism before surgery required long-term hormone replacement therapy (Table 1).

Pathological findings and surgical complications

Transsphenoidal approach(TSA) was undertaken in 27 cases and 2 cases were undertaken two staged operations. One of 2 cases, the second operation was performed craniotomy, 12 days after partial excision in TSA, the other cases, the second operation was performed against the remaining tumors in sellar turcica through TSA, 4 months after first operation was performed in craniotomy.

In pathologic findings, 27 cases (93.1%) showed hemorrhage of pituitary adenoma and 2 cases (6.9%) showed pure ischemic infarction. Among 27 cases, hemorrhagic infarction was observed in 3 cases (10.3%). Postoperative complications were developed DI in 1 case (3.4%), CSF leakage in 2 cases (6.8%).

DI was needed long term treatment and CSF leakage patients improved by lumbar drainage. Also, there was no case which underwent additional radiotherapy after surgery. Two cases (6.9%) were recurred at 12 months and 64 months, then reoperation was undertaken.

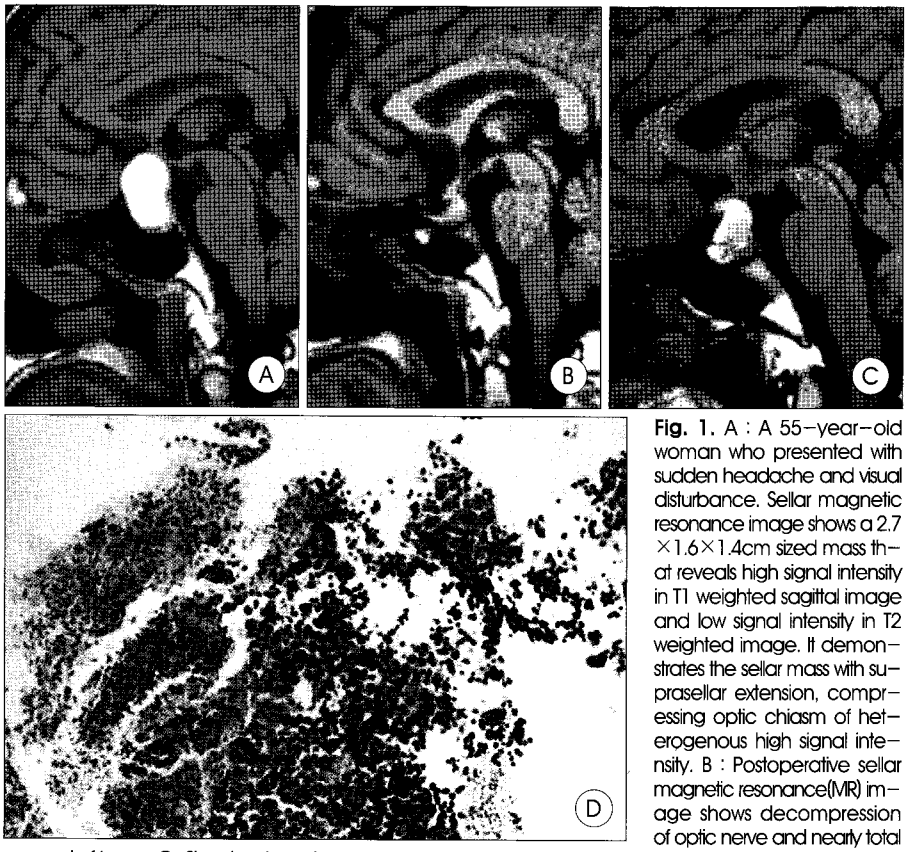

Fig. 1. A : A 55-year-old woman who presented with sudden headache and visual disturbance. Sellar magnetic resonance image shows a 2.7×1.6×1.4cm sized mass that reveals high signal intensity in T1 weighted sagittal image and low signal intensity in T2 weighted image. It demonstrates the sellar mass with suprasellar extension, compressing optic chiasm of heterogenous high signal intensity. B : Postoperative sellar magnetic resonance(MR) image shows decompression of optic nerve and nearly total removal of tumor. C : She developed progressing headache and decreased visual acuity at postoperative 12 months. Sellar MR image shows mass with the size of 2.0×1.7×1.7cm, high signal intensity lesion in T1WI and T2WI with suspicious chronic stage hemorrhage. D : Microscopic feature of pituitary adenoma with apoplexy. Photomicrograph reveals solid sheets of tumor cell with hemorrhage (H&E, ×40).

Illustrative case

A 55-year-old female (case 7) who presented with sudden headache and visual disturbance. Sellar MRI (magnetic resonance imaging) showed a 2.7×1.6×1.4cm sized mass that reveals high signal intensity in T1 weighted images (T1WI)

and low signal intensity in T2 weighted images (T2WI). It demonstrates heterogenous high signal intensity sellar mass with suprasellar extension compressing optic chiasm. She had hypocortisolism in hormone study (Table 1). TSA was undertaken and pathologic diagnosis was pituitary adenoma with hemorrhage (Fig. 1). After surgery, she improved in pituitary dysfunction and recovered from decreased visual acuity and field defect (Fig. 1). Postoperative sellar MRI shows decompression of optic nerve and nearly total removal of tumor (Fig. 1). She developed headache and decreased visual acuity at postoperative 12 months. Sellar MRI shows mass with the size of 2.0×1.7×1.7 cm, high signal intensity lesion in T1WI and T2WI with suspicious chronic stage hemorrhage. It also demonstrates the suprasellar extension with compression of optic nerve. So the patient's diseases was diagnosed as pituitary apoplexy accompanied with recurrence of a pituitary tumor (Fig. 1).

Discussion

Pituitary adenoma is a tumor that occurs in pituitary gland, accounts for 10~15% of intracranial tumor[14], and its symptoms appear by compressing surrounding structure and cranial nerves due to growth of a tumor. Pituitary apoplexy is a clinical syndrome that occurs due to sudden expansion of a tumor caused by spontaneous hemorrhage, hemorrhagic infarction or ischemic infarction of a pituitary adenoma. Major clinical symptoms and signs include sudden headache, reduction in visual fields or acuity, altered consciousness, and partial or total hypopituitarism.

The incidence of pituitary apoplexy is variously reported from 1.5% to 27.7%[22]. In this study, between 1995 and 2004, 133 cases of pituitary adenoma were managed surgically, giving an incidence of classical pituitary apoplexy is 29 cases (21.8 %). Wakai et al[22] reported that hemorrhage in a pituitary adenoma is about 5.4 times as high as other intracranial tumors and there was no gender difference in the incidence. Also, there was no difference in the incidence between men and women in this study.

It is reported that predisposing factors causing pituitary apoplexy include radiotherapy, head trauma, bromocriptine therapy, general anesthesia or surgical stress, pregnancy, cerebral angiography, coagulopathy and dynamic pituitary function test[1,3,4,19]. It was presumed that, under general anesthesia, pituitary apoplexy can be caused by the change of intracranial pressure. In case of a patient with acromegaly, apoplexy may occur due to a sudden change in arterial pressure during cerebral angiography[12]. The cause of the occurrence of pituitary apoplexy has been unknown, but various hypothesis are maintained by various authors. Rolih et al[20] asserted ; 1) the inherent weakness of tumor blood vessels, 2) imbalance with blood supply caused by rapid growth of a pituitary adenoma, 3) compression of infundibulum caused by growing a pituitary adenoma, 4) arteriosclerotic embolus.

Mohr et al[14] reported that apoplexy occurred due to abnormal distribution of blood vessels in a pituitary tumor and blood pressure caused by the growth of a tumor or abnormal supply of tears of vessels. Also, there is a report that low blood pressure causing hypoxia in a tumor may induce infarction of pituitary. Rovit et al[21] explained the compression of trabecular artery which was a branch of superior hypophyseal artery due to the expansion of diaphragm sellae caused by the growth of a tumor, which led to occurrence of the first infarction and the second modification, so that pituitary apoplexy occurred. Onesti et al[15] maintained that pituitary apoplexy was caused by ischemic necrosis of adenomatous tissue, an intrinsic vasculopathy of pituitary tumors.

It is difficult to exactly diagnose pituitary apoplexy due to similarities with other diseases. Computed tomography(CT) is an excellent tool for detecting pituitary adenomas as well as intratumoral hemorrhage from 24 hours to 48 hours[18]. A recent hemorrhage may appear as a round and well-defined high density lesion that shows minimal or no enhancement[4]. In several study, MRI was 100% sensitive in disclosing tumor and revealed hemorrhage in 88%[18]. Typically, pituitary hemorrhage less than 7 days appears hypointense on both T1WI and T2WI, but increased signal intensity is evident at the tumor periphery on T1WI at 7 to 14 days[20]. Angiography may be important in rule out an intracranial aneurysm in patients with pituitary apoplexy in whom CT scanning or MRI fails to demonstrate pituitary hemorrhage[18].

The clinical presentation of pituitary apoplexy is various and symptoms and signs do not occur in all cases. Typically most common symptom is headache in other studies, but in this study visual disturbance is most common. Maybe in this area, many people have regarded headache as simple symptom so most of them have endured it until other symptom would appear. Symptoms that commonly occur include visual disturbance, which is caused by the rapid expansion of a tumor due to sudden bleeding of a tumor and hemorrhagic infarction or ischemic infarction as the tumor grows toward optic apparatus. Lateral expansion of a tumor into the cavernous sinuses may lead to extraocular ophthalmoplegia, trigeminal nerve dysfunction, venous stasis and carotid artery compression.

Nausea and vomiting may occur due to subarachnoid hemorrhage caused by increased intracranial pressure and altered consciousness, hemiplegia, high fever and endocrine dysfuction may occur. Apoplexy usually occurs from 24 hours to 48 hours and may result in sudden aggravation of patient's clinical presentation and syncope appears due to acute pituitary adrenal

insufficiency[17]. If a patient takes cortisone acetate, the symptoms may improve greatly. We had good results through surgery of pituitary apoplexy. In this study, headache was recovered in 19 cases (86.4%) and from the 16 cases (88.9%) of improved visual acuity, there was 11 cases (61.1%) of complete recovery and 5 cases (27.8%) of partial recovery, after surgery. With regard to hormone study at 3 months after surgery, hormone level was recovered to the normal range in 7 cases (77.9%) of prolactinoma and improved but abnormal range in 2 cases (22.1%), one of 2 cases was treated by bromocriptine.

Parent[16] has reported that the compression of a tumor on the optic nerve brings about changes in the structure and function of the optic nerve in histopathology, decrease of visual acuity and visual fields may occur due to damage of the structure of the optic nerve caused by pressure on blood vessels. In pituitary apoplexy, the surgical decompression is effective for visual disturbance. Onesti et al[15] reported case of 6 patients with complete recovery of visual field defect among 8 patients. Parent[16] maintained that there were no many relations between the time interval from onset of clinical presentation to surgery, the severity of the initial symptoms. Laws et al[10] reported that only 1 patient among 62 patients showed complete loss of vision. Cohen et al[7] reported that 4 patients among 100 patients showed no improvement in visual symptoms and these patients had no light perception before an operation, and thus maintained whether or not a patient was able to perceive light before an operation was an important prognostic factor in the recovery of the optic nerve[7]. McFadzean et al[13] administered high-dose steroids along with radiotherapy before surgery, as a result it turned out that a few patients showed improvement in their visual symptoms and signs before surgery but most patients showed no change. The patients who showed visual field recovery was 9 cases (75.0%), among whom 7 cases (58.3%) showed complete recovery and 2 cases (16.7%) partial recovery, in this study.

In this study, hormonal dysfunction occurs due to reduction of the anterior pituitary function temporarily or permanently, 5 of 10 cases in hypocortisolism and 3 of 8 cases in hypothyroidism before surgery required long-term hormone replacement therapy. In pre-operative endocrine studies, prolactinoma was most common in pituitary hyperfunction.

Louwerens et al[12] reported in the hormone evaluation after surgery that GH deficiency was 88~100%, gonadotrophin deficiency 58~76%, ACTH deficiency 66~75%, TSH deficiency 42~50%, and DI 2~4%. Replacement of other hormones is not usually required in the acute setting, but close observation of hypothyroidism and hypogonadism should be done during pre- and postoperative period and may need replacement transiently[20]. It is known that early hormone replacement can reduce the mortality and morbidity rate. Some studies report that conservative treatment somewhat relieved headache and was effective for improvement in the optic nerve, but review of the cases where symptoms and signs improve greatly after surgery, the surgical treatment is considered necessary[16].

In pathologic findings of this study, 27 cases (93.1%) showed hemorrhage of pituitary adenoma and 2 cases (6.9%) showed pure ischemic infarction. Among 27 cases, hemorrhagic infarction was observed in 3 cases (10.3%). Randeva[18] reviewed 35 cases with pituitary apoplexy -hemorrhage in 31, ischemic necrosis in 18, blood clot in 2. Wakai[22] reported 560 cases and 93 cases(16.6%) had evidence of pituitary hemorrhage. In this study, all cases had pathological findings.

Post operative DI was 3.4% in this study. Randeva[18] found post operative diabetes insipidus to be transient in 16% and permanent in 6%. The incidence of DI was rare, but the rate was higher in decompressive surgery than conservative treatment.

Conclusion

We reported good results through surgery of pituitary apoplexy in a clinical and endocrine outcomes. Surgery should be performed as soon as possible to treat pituitary apoplexy and it is considered necessary to additional study for etiology and prognosis.

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Commentary

Although Bailey was the first to describe a clinical case of pituitary apoplexy in 1898 and Brougham coined the term pituitary apoplexy in 1950, pituitary apoplexy is an ill-defined clinical entity. (From Greek apoplexia being struck down, from apoplessein to disable by a stroke.)

Some authors apply the term strictly to hemorrhage within a pituitary adenoma, whereas others include hypoxic infarction in a mass, or even the infarction of the pituitary gland that can occur in association with hemorrhagic delivery in the absence of an adenoma. Finally some restrict the motion of pituitary apoplexy to those cases resulting in pituitary insufficiency.

Because of this semantic confusion, no precise prevalence of this relatively rare clinical entity is available, but the majority of described cases occurred in patients harboring previously unknown pituitary adenomas.

Classically defined, pituitary apoplexy refers to the abrupt and occasionally catastrophic occurrence of acute hemorrhagic infarction of a pituitary adenoma. The clinical syndrome is easily recognized, consisting of acute headache, meningismus, visual impairment, ophthalmoplegia, and alterations in consciousness even coma. Without timely intervention, patients are likely to die of subarachnoid hemorrhage and increased intracranial pressure or succumb to acute, life-threatening hypopituitarism.

The incidence of pituitary apoplexy presenting with classical symptoms(classic pituitary apoplexy) in about 3% of all pituitary adenomas. However, clinically silent histopathological evidence of pituitary hemorrhage(subclinical pituitary apoplexy) is much more common, and has been observed in up to 25% of the surgically removed pituitary adenomas.

In this paper, 29 surgical cases were pathologically diagnosed pituitary adenoma with apoplexy among 133 cases of pituitary adenoma on the same period (from January 1995 to march 2004), the incidence was a little bit higher than other series, it maybe include subclinical type of pituitary apoplexy in this study.

There is little consensus in the literature about which tumor types, if any, are most vulnerable to apoplectic hemorrhage. Some authorities have suggested that hormonally active tumors associated with acromegaly and Cushing's disease are especially prone to apoplexy, whereas others have found large prolactinoma or large, nonfunctional tumors to be substantial risk.

Regarding treatment modality, it is generally accepted that the initial management of pituitary apoplexy consists of careful monitoring of fluid and electrolyte balance coupled with immediate replacement of deficient hormones, in particular corticosteroids.

However, the role and timing of pituitary surgery in this condition remains controversial. Some authors advocate urgent surgical decompression of the pituitary fossa, while others have suggested that restoration of neuro-ophthalmic function may still occur even when decompression is delayed.

In contrast, other authors favour a more conservative approach in management of pituitary apoplexy, particularly in the absence of progressive neuro-ophthalmic signs.

Acording to my limited experience, acute hemorrhagic infarction of a pituitary tumor constitutes a true neurosurgical emergency for which prompt recognition and glucocorticoid replacement are the most important first management steps.

In contrast to hemorrhagic apoplexy, infarctive apoplexy tends to affect only the tumor and thus presents with mild symptoms and lack pituitary deficiencies, and also treatment was not so urgent, may possiblely treat with delayed treatment or conservative treatment.

Although conservative medical therapy may offer an alternative option for certain patients, urgent surgical pituitary decompression is the preferred treatment particularly in classic pituitary apoplexy patients.

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