



# Pituitary Apoplexy and Cerebral Infarction: Case Report and Literature Review

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## Key words

- Case report
- Cerebral infarction
- Compression
- Internal carotid artery
- Pituitary apoplexy
- Stroke
- Vasospasm

## Abbreviations and Acronyms

ICA: Internal carotid artery

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Citation: *World Neurosurg.* (2020) 141:73-80. <https://doi.org/10.1016/j.wneu.2020.05.276>

Journal homepage: [www.journals.elsevier.com/world-neurosurgery](http://www.journals.elsevier.com/world-neurosurgery)

Available online: [www.sciencedirect.com](http://www.sciencedirect.com)

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## INTRODUCTION

Pituitary apoplexy is a clinical syndrome characterized by headache, vision deficits, and altered consciousness due to infarction and hemorrhage in the pituitary gland. The incidence of apoplexy is 0.6%–9% and can lead to fatal consequences in up to 10% of cases if not addressed promptly.<sup>1,2</sup> Headache is the most consistent symptom, followed by nausea and vomiting.<sup>3</sup> Visual acuity and ocular motility are commonly affected because of the rapid growth and compression of the optic nerves/chiasm and the oculomotor, trochlear, and abducens nerves. Laboratory and radiologic investigations are paramount for diagnosing pituitary apoplexy, followed by medical or surgical management, depending on the chronologic course and severity of symptoms. Acute ischemic stroke associated with carotid artery occlusion is an extremely rare complication of pituitary apoplexy. We report an unusual

■ **BACKGROUND:** Pituitary apoplexy is defined as a sudden neurologic deficit owing to infarction or hemorrhage within the pituitary gland. We report a rare case of apoplexy manifesting with cerebral infarction due to direct compression of the internal carotid artery (ICA) and review the literature.

■ **CASE DESCRIPTION:** A 31-year-old man presented with sudden-onset headache, right hemiparesis, decreased left monocular visual acuity, and a nasal visual field deficit of the left eye. Computed tomography angiography revealed evidence of a hyperdense sellar/suprasellar mass with stenosis of the cavernous and supraclinoid segments of the ICAs bilaterally. Magnetic resonance angiography performed the following day showed complete occlusion of the left cervical ICA and cystic changes of the sellar and suprasellar mass suggestive of pituitary hemorrhage. The patient underwent urgent endoscopic endonasal decompression of the mass, and postoperative digital subtraction angiography demonstrated restored flow within the left cervical ICA.

■ **CONCLUSIONS:** Of 29 previously published cases of cerebral infarction due to pituitary apoplexy, the majority of cases were related to direct ICA compression. Vascular compression is associated with a high rate of mortality (24%) and should be treated urgently by surgical decompression in cases of severe or progressive neurologic symptoms.

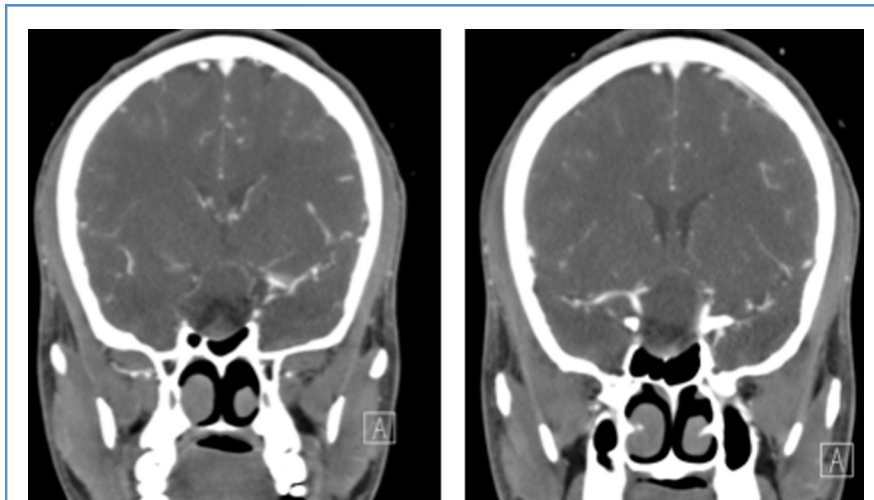
case of a patient with pituitary apoplexy manifesting with right hemiparesis owing to internal carotid artery (ICA) compression bilaterally. We also performed a literature review of 29 reported cases and compared their etiology, treatment, and outcomes.

## Case Description

A 31-year-old, previously healthy man presented to an outside hospital with sudden onset of headache, right hemiparesis, and decreased left monocular visual acuity during manual labor. Computed tomography and computed tomography angiography of the brain showed a sellar/suprasellar mass causing stenosis of the cavernous and supraclinoid ICA bilaterally (Figure 1). The patient was transferred to our hospital for further management. On presentation to the emergency department, he was awake and alert to time, place, and person. His motor strength was graded 4/5 in the right upper and lower limbs with a right footdrop; his left side was graded 5/5.

Visual acuity was 20/25 in the right eye and 20/50 in the left eye. There was a nasal visual field deficit in the left eye, but the right eye visual fields were full. His endocrine, chemical, and hematologic laboratory results all were within normal limits.

Approximately 7 hours after the computed tomography scan, magnetic resonance imaging of the brain was completed, which revealed a left centrum semiovale infarct (Figure 2) and a pituitary mass with cystic changes consistent with apoplexy (Figure 3). Magnetic resonance angiography was concomitantly performed, revealing compression and narrowing of the cavernous and supraclinoid ICA segments bilaterally (Figure 4), with complete cessation of blood flow in the left cervical ICA that was not present on computed tomography angiography performed previously. Digital subtraction angiography was performed and demonstrated complete occlusion of the left ICA and significant narrowing of the right ICA (Figure 5).



**Figure 1.** Computed tomography angiography of the head with contrast demonstrating a heterogeneous sellar/suprasellar mass with filling of the internal carotid artery bilaterally.

The patient underwent emergency endoscopic endonasal resection of the sellar/suprasellar tumor. Postoperatively, his motor strength improved in the right lower limb to 4+/5, while the right upper limb remained 4/5. Postoperative magnetic resonance angiography showed return of flow in the left cervical ICA with persistent stenosis of the cavernous and supraclinoid segments of the ICA bilaterally (**Figure 6**). He was discharged on postoperative day 6, with hydrocortisone

and aspirin (81 mg daily). His weakness improved, and he was neurologically intact and independent in all activities on 6-month follow-up.

#### LITERATURE REVIEW METHODS

A literature review of pituitary apoplexy and cerebral infarction was performed. Search terms “apoplexy” OR “pituitary apoplexy” AND “pituitary adenoma” OR “pituitary macroadenoma” AND “stroke”

OR “cerebral infarction” OR “cerebral ischemia” OR “ischemic stroke” OR “hemispheric stroke” OR “vasospasm” OR “internal carotid artery compression” were entered in Embase, MEDLINE, and Web of Science. Screening guidelines included articles written in English that included basic demographic information, presentation, and follow-up that were recoverable through the online databases. The databases were last queried on November 5, 2019. Owing to differences in medical technology and management, articles published before 1990 were excluded. Limitations to this literature review included reporting bias and possible incomplete retrieval of identified research.

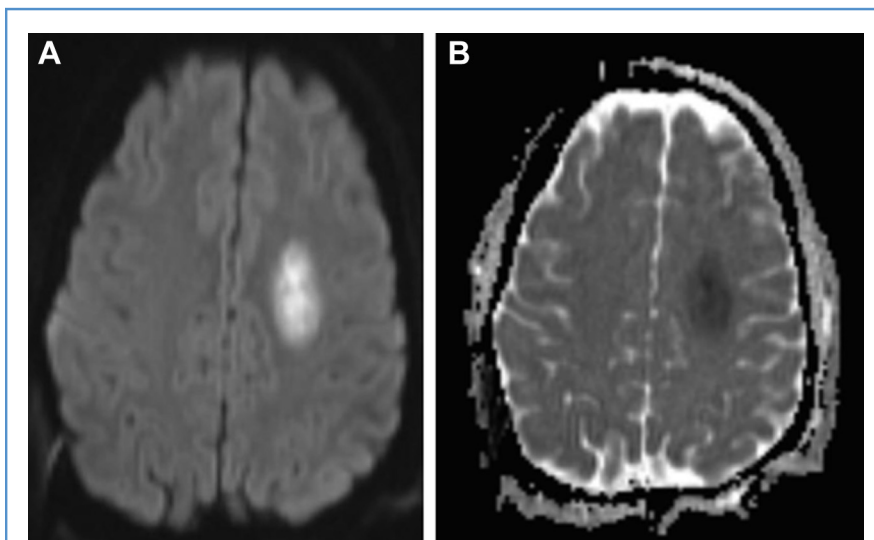
#### Statistical Analysis

IBM SPSS 25.0 (IBM Corporation, Armonk, New York, USA) software was used to analyze the qualitative variables gathered from all the cases. Comparisons of means and distributions of normally continuous variables were carried out using Student t test, and nonparametric distributions were compared with Wilcoxon rank sum test. Categorical variables were presented as estimated frequency and percentage. Statistical analyses of categorical variables were carried out using  $\chi^2$  and Fisher exact test with the Monte Carlo simulation tests as appropriate.

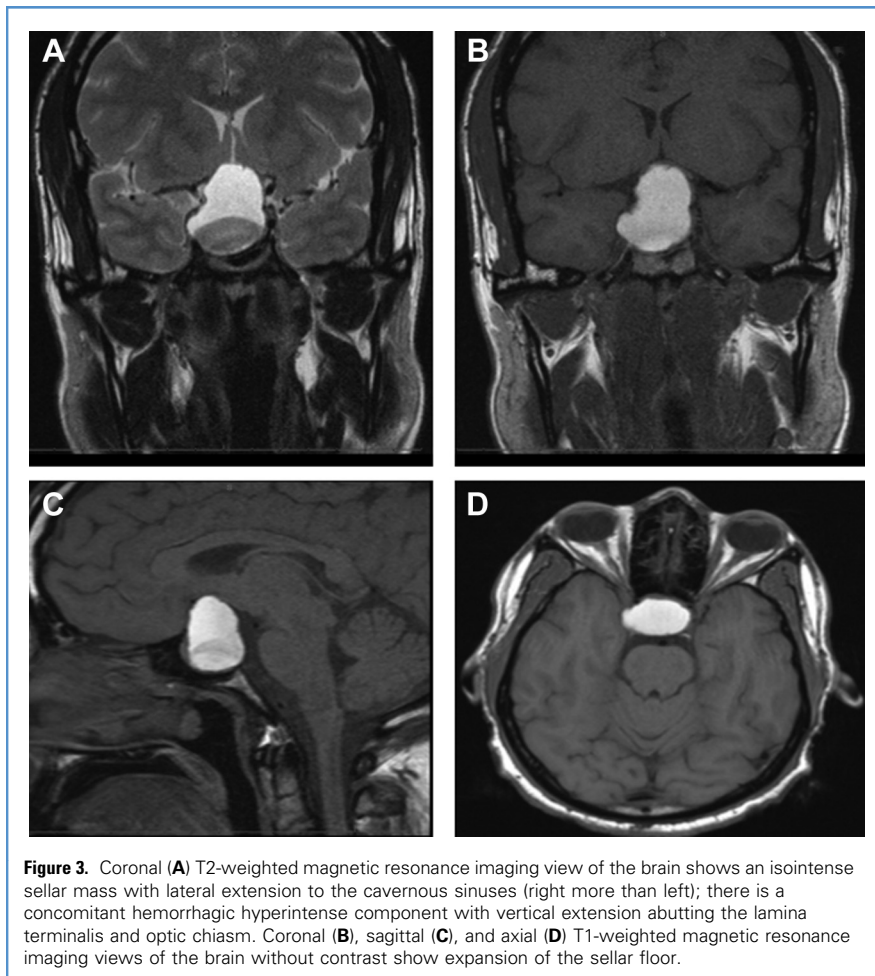
#### Literature Review Results

We included 28 published case reports of cerebral infarction associated with pituitary apoplexy published in the English literature between 1990 and 2019 (**Table 1**). In addition, we include the data from the patient presented in this case report. Of the 29 patients, 17 (58.6%) were reported to have compression, 9 (31.0%) had vasospasm, and 3 (11.5%) had both vasospasm and compression (**Table 2**). Patients were predominantly male (24 of 29 patients; 82.7%) with a mean (SD) age of 45 (14.4) years. There was no difference in age or sex between patients who developed compression versus vasospasm. There was also no difference in treatment strategy (surgical vs. medical) between the 2 groups (**Table 2**).

Of the 29 patients, 6 (20.7%) underwent urgent surgical decompression within the first 7 days of presentation, 4 (13.8%) patients underwent delayed surgical



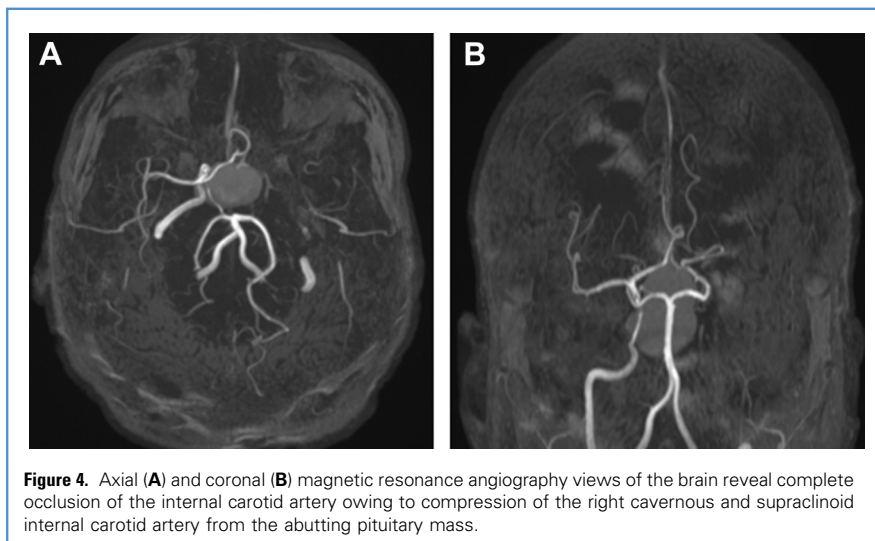
**Figure 2.** Axial diffusion-weighted imaging (A) and apparent diffusion coefficient (B) magnetic resonance imaging showing left centrum semiovale diffusion restriction, in concordance with infarction.



**Figure 3.** Coronal (A) T2-weighted magnetic resonance imaging view of the brain shows an isointense sellar mass with lateral extension to the cavernous sinuses (right more than left); there is a concomitant hemorrhagic hyperintense component with vertical extension abutting the lamina terminalis and optic chiasm. Coronal (B), sagittal (C), and axial (D) T1-weighted magnetic resonance imaging views of the brain without contrast show expansion of the sellar floor.

treatment between 14 and 30 days from presentation, 13 (44.8%) patients had surgery at an unspecified time point, and 6

(20.7%) patients were managed with medical therapy alone (Table 1). Of the 23 patients who underwent surgery, 18 (78%)



**Figure 4.** Axial (A) and coronal (B) magnetic resonance angiography views of the brain reveal complete occlusion of the internal carotid artery owing to compression of the right cavernous and supraclinoid internal carotid artery from the abutting pituitary mass.

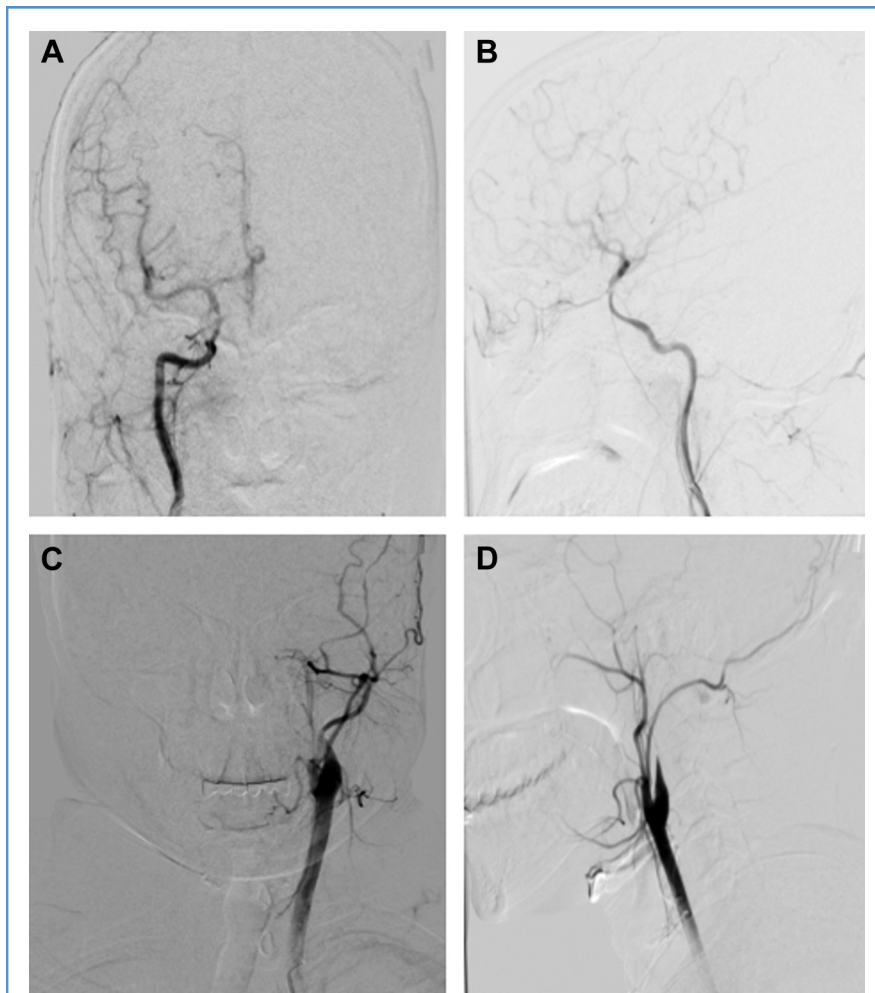
underwent a transsphenoidal approach, and 4 (17%) underwent a craniotomy, and 1 case report did not specify the approach. Of the 18 patients who underwent a transsphenoidal approach, 7 (39%) patients underwent an endoscopic endonasal approach. The remaining 11 cases did not specify endoscopic versus microscopic transsphenoidal approach. Of the 23 patients who had surgery, 16 (70%) patients improved, 2 (9%) patients worsened, and 5 (21%) patients died. Of the 6 patients who had medical therapy alone, 4 (67%) patients improved and 2 (33%) patients died.

## DISCUSSION

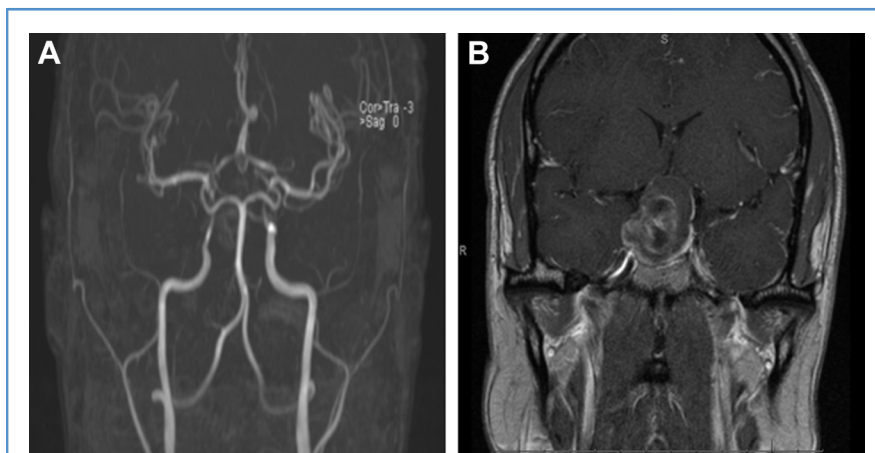
Pituitary apoplexy is an uncommon clinical condition describing acute neurologic deterioration from hemorrhage and/or infarction in the pituitary gland. Bailey<sup>31</sup> described the first case of pituitary hemorrhage in 1898, and Brougham et al.<sup>32</sup> later coined the term “pituitary apoplexy” after performing 5 autopsies with hemorrhagic and infarct transformation of the gland. Before steroid replacement, the mortality from pituitary apoplexy reached 50%.<sup>33</sup> However, modern-day series report 0%–15.3% mortality.<sup>34</sup> Risk factors include large size adenomas, hypertension, hypotension, bromocriptine initiation or withdrawal, hormonal stimulation of the pituitary gland, cavernous sinus invasion, and increased intracranial pressure.<sup>1,3,35</sup>

Pituitary adenomas are highly susceptible to infarction compared with other central nervous system tumors.<sup>36</sup> The pathogenesis of apoplexy is likely related to the high metabolic rate of these tumors, low vascular density, and high intratumoral pressure, all of which contribute to infarction and subsequent hemorrhage.<sup>36</sup> However, cortical ischemic stroke associated with apoplexy is rare, with only 29 cases reported in the English literature since 1990.

Cerebral infarction secondary to pituitary apoplexy is a severe consequence of pituitary apoplexy with an overall mortality of 24%. Infarction may be due to ICA compression by the rapidly expanding intratumoral hemorrhage, vasospasm owing to subarachnoid hemorrhage and release of vasoactive substances from the adenoma or hypothalamus, or a



**Figure 5.** (A and B) Digital subtraction angiography of the right internal carotid artery (ICA) confirms narrowing of the right cavernous and supraclinoid ICA segments without cross-filling of the left hemisphere. (C and D) Digital subtraction angiography of the left ICA shows complete occlusion.



**Figure 6.** (A) Postoperative magnetic resonance angiography of the brain shows return of blood flow in the left internal carotid artery. (B) Immediate postoperative coronal T1-weighted magnetic resonance imaging with contrast demonstrates resection of the adenoma and hematoma and postoperative changes.

combination of both.<sup>4,8,9,11,14,15,19</sup> In our case, we postulate that the inciting event was the dynamic compression of the cavernous and supraclinoid ICA bilaterally by the expanding pituitary hemorrhage. This gradual compression resulted in hypoperfusion, left subcortical infarcts, and a delayed complete occlusion with cessation of blood flow the next day. There is 1 similar case report of right cavernous and supraclinoid ICA compression from pituitary apoplexy leading to cervical ICA stenosis.<sup>37</sup> However, our case is unique with its documented delayed temporal sequence and complete left ICA occlusion the day after initial presentation.

Based on our literature review, reports of cerebral infarction owing to compression are more common than vasospasm.<sup>6,7</sup> The supraclinoid and cavernous segments of the ICA are most commonly affected in apoplexy-related vessel compression, with unilateral compression more frequent than bilateral compression (59% and 41%, respectively).<sup>7</sup> Furthermore, acute ischemic stroke owing to compression from apoplexy trended toward higher rates of mortality than apoplexy-associated vasospasm, although this difference was not significant (35% vs. 11%,  $P = 0.186$ ). Consistent with our study, Banerjee et al.<sup>7</sup> and Zou et al.<sup>6</sup> also found a higher mortality in the compression group compared with the vasospasm group (47% vs. 14%).

The most common presenting symptoms of cerebral infarction related to pituitary apoplexy are hemiparesis, headache, and loss of consciousness. The presentations were similar between the compression and vasospasm groups, although more patients in the vasospasm group presented with decreased mental status (56% vs. 24%), whereas more patients in the compression group had cranial nerve III, IV, or VI deficits (44% vs. 0%).

While the indications for surgery could not be clearly identified in many of the case reports, surgery was associated with symptom improvement in 62% of patients in the compression group and 67% of patients in the vasospasm group. Medical management alone was associated with a 50% mortality rate in the compression group versus 0% in the vasospasm group. Thus, medical management may be

**Table 1.** Summary of Case Reports Published in the Literature

Case	Reference	Age (Years)/ Sex	Vessel	Etiology	Imaging	Hemorrhage Location	Presentation	Management	Timing of Surgery (Days)	Outcome
1	Present case, 2020	31/M	Left cervical ICA, BL cavernous and supraclinoid ICA	Compression	CT, CTA, MRI, MRA, DSA	Sellar/suprasellar	Headache, right hemiparesis, left visual loss	Transsphenoidal approach (endoscopic)	2	Improved
2	Sussman et al., 2016 <sup>4</sup>	46/M	BL cavernous ICA	Compression	CT, CTA, CT perfusion, MRI	Sellar/suprasellar	Bilateral visual field loss, left CN III and IV palsy, left hemiparesis	Transsphenoidal approach (endoscopic)	Not specified	Improved
3	Kasl et al., 2015 <sup>5</sup>	14/F	Left cavernous and supraclinoid ICA	Compression	CT, CTA, MRI, DSA	Sellar/suprasellar	Right upper limb paresthesia and weakness, expressive aphasia	Transsphenoidal approach (endoscopic)	1	Worsened (new right lower limb weakness)
4	Zou et al., 2015 <sup>6</sup>	23/M	Right cavernous ICA, right M1, right A1	Vasospasm	CT, MRI, MRA, TCD	Sellar/suprasellar	Headache, nausea, decreased visual acuity, loss of consciousness	Craniotomy	7	Improved
5	Banerjee et al., 2015 <sup>7</sup>	56/F	BL cavernous ICA	Compression	CT	Sellar/suprasellar	Headache, loss of consciousness	Transsphenoidal approach (endoscopic)	Not specified	Death
6	Cho et al., 2014 <sup>8</sup>	40/F	Left lacerum ICA	Compression	MRI, MRA	Sellar/suprasellar	Headache, right hemiparesis, expressive aphasia, left CN VI deficit	Conservative management	N/A	Improved
7	Rebeiz et al., 2014 <sup>9</sup>	81/F	BL ACA	Compression	MRI, MRA	Sellar/suprasellar	Loss of consciousness, left hemiparesis	Conservative management	N/A	Death
8	Navarro-Bonnet et al., 2014 <sup>10</sup>	30/M	Right cavernous ICA, right M1, right A1	Compression	CT, MRI, MRA	Sellar/suprasellar	Headache, right CN III and IV deficits, right visual acuity loss	Craniotomy	Not specified	Improved
9	Zhang et al., 2014 <sup>11</sup>	42/M	Right supraclinoid ICA, left A1	Compression (right ICA), vasospasm (left ACA)	CT, MRI, TCD, MRA	Sellar/suprasellar	Left hemiparesis, right CN III deficit, loss of consciousness	Transsphenoidal approach (method not mentioned)	28	Improved
10	Hassan et al., 2013 <sup>12</sup>	44/M	Right MCA	Vasospasm	CT, MRI	Sellar and SAH	Headache, loss of consciousness	Craniotomy	Not specified	Death
11	Chokyu et al., 2011 <sup>13</sup>	50/M	Right cavernous ICA	Compression	CT, MRI, MRA	Sellar/suprasellar	Right CN III deficit, left hemiparesis	Transsphenoidal approach (method not mentioned)	30	Improved
12	Cerese et al., 2010 <sup>14</sup>	70/M	Diffuse intracranial arteries	Vasospasm	CT, MRI, MRA	Sellar/suprasellar	Left upper limb weakness, left facial droop (upper motor neuron), dysarthria	Conservative management	N/A	Improved
13	Lill et al., 2009 <sup>15</sup>	59/M	BL cavernous ICA	Compression	CT, MRI	Sellar/suprasellar	Bilateral amaurosis fugax, left hemiparesis, right CN III and IV deficits	Conservative management	N/A	Death

M, male; ICA, internal carotid artery; BL, bilateral; CT, computed tomography; CTA, Computed tomography angiography; MRI, magnetic resonance imaging; MRA, magnetic resonance angiography; DSA, digital subtraction angiography; CN, cranial nerve; F, female; M1, M1 segment of middle cerebral artery; A1, A1 segment of anterior cerebral artery; TCD, transcranial Doppler; N/A, not applicable; ACA, anterior cerebral artery; MCA, middle cerebral artery; SAH, subarachnoid hemorrhage; GCS, Glasgow Coma Scale; NLP, no light perception.

Continues

Table 1. Continued

Case	Reference	Age (Years)/ Sex	Vessel	Etiology	Imaging	Hemorrhage Location	Presentation	Management	Timing of Surgery (Days)	Outcome
14	Yang et al., 2008 <sup>16</sup>	43/M	BL cavernous ICA	Compression	CT, MRI, MRA, DSA	Sellar/suprasellar	Left CN III deficit, right hemiparesis	Transsphenoidal approach (method not mentioned)	4	Improved
15	Ahmed and Semple, 2008 <sup>17</sup>	51/M	BL ACA	Compression	CT, MRI	Sellar/suprasellar	Headache, left hemiparesis, loss of consciousness (GCS 8)	Transsphenoidal approach (endoscopic)	Not specified	Improved
16		31/F	BL ACA	Vasospasm	CT, MRI	Sellar/suprasellar	Headache, BL vision loss	Transsphenoidal approach (endoscopic)	Not specified	Improved
17	Das et al., 2008 <sup>18</sup>	46/M	Right supraclinoid ICA, left A1	Compression	CT, MRI, MRA	Sellar/suprasellar	Headache, BL vision loss, seizures	Transsphenoidal approach (method not mentioned)	7	Improved
18	Ben-Nakhi et al., 2008 <sup>19</sup>	40/M	Left MCA	Vasospasm	CT, MRI, TCD	Sellar/suprasellar	Transient right hemiparesis, receptive aphasia, loss of consciousness	Conservative management	N/A	Improved
19	Dogan et al., 2008 <sup>20</sup>	50/M	Left supraclinoid ICA	Compression	CT, MRI	Sellar/suprasellar	Headache, BL visual deficit, loss of consciousness	Craniotomy	Not specified	Death
20	Jeon et al., 2007 <sup>21</sup>	41/M	Left cavernous ICA	Vasospasm	CT, MRI	Sellar and SAH	Headache	Transsphenoidal approach (endoscopic)	7	Improved
22	Lomban et al., 2006 <sup>22</sup>	57/M	Left cavernous ICA	Compression	CT, CTA, MRI, MRA, DSA	Sellar/suprasellar	Headache, loss of consciousness	Conservative management	N/A	Improved
23	Kurschel et al., 2005 <sup>23</sup>	61/M	BL cavernous ICA	Compression	CT, MRI, MRA	Sellar/suprasellar	Headache, right CN III deficit	Transsphenoidal approach (method not mentioned)	Not specified	Expired
24	Bhansali et al., 2005 <sup>24</sup>	30/M	BL A1	Vasospasm	CT, CTA, MRI, DSA	Sellar and interhemispheric SAH	Frontal disinhibition, BL blindness (NLP)	Transsphenoidal approach (method not mentioned)	Not specified	Improved (except right eye blindness)
25	Akutsu et al., 2004 <sup>25</sup>	29/M	Left A1	Compression and vasospasm	CT, MRI, DSA	Sellar/suprasellar	Decreased level of consciousness, left CN III deficit, left hemifacial hypoesthesia	Transsphenoidal approach (method not mentioned)	Not specified	Improved (except right homonymous hemianopsia)
26	Rodier et al., 2003 <sup>26</sup>	35/M	BL cavernous, supraclinoid ICA, BL A1, right M1	Compression and vasospasm	CT, MRI, DSA	Sellar/suprasellar	Headache, left hemianesthesia and hemiparesis	Surgery (not mentioned if craniotomy or transsphenoidal)	Not specified	Worsened
27	Lath and Rajshekhkar, 2001 <sup>27</sup>	40/M	Right cavernous ICA	Compression	CT	Sellar/suprasellar	Headache, vomiting, BL visual loss, left hemiplegia	Transsphenoidal approach (method not mentioned)	Not specified	Death
28	Sanno et al., 1999 <sup>28</sup>	55/M	Right MCA	Vasospasm	CT, MRI, DSA	Sellar and SAH in right sylvian fissure	Loss of consciousness, transient left hemiparesis, diabetes insipidus	Transsphenoidal approach (method not mentioned)	14	Improved

29	Yaghmai et al., 1996 <sup>29</sup>	47/M	Right supraclinoid ICA	Compression	CT, MRI, MRA, DSA	Sellar/suprasellar	Headache, right vision loss	Transsphenoidal approach (method not mentioned)	Not specified	Improved
30	Itoyama et al., 1990 <sup>30</sup>	45/M	Left A1 and M1	Vasospasm	CT, DSA	Sellar and left sylvian fissure SAH	Left hemiparesis, confusion	Transsphenoidal approach (method not mentioned)	28	Improved

M, male; ICA, internal carotid artery; BL, bilateral; CT, computed tomography; CTA, Computed tomography angiography; MRI, magnetic resonance imaging; MRA, magnetic resonance angiography; DSA, digital subtraction angiography; CN, cranial nerve; F, female; M1, M1 segment of middle cerebral artery; A1, A1 segment of anterior cerebral artery; TCD, transcranial Doppler; N/A, not applicable; ACA, anterior cerebral artery; MCA, middle cerebral artery; SAH, subarachnoid hemorrhage; GCS, Glasgow Coma Scale; NLP, no light perception.

**Table 2. Clinical Presentation, Management, and Outcome in Relation to Arterial Pathology Type**

Patient Characteristics	Compression (n = 17)	Vasospasm (n = 9)	Vasospasm and Compression (n = 3)	Total (n = 29)
Age, years, mean (±SD)	47 (±14.7)	41 (±15.1)	45 (±11.5)	45 (±14.4)
Male sex	13 (77%)	8 (89%)	3 (100%)	24 (83%)
Decreased level of consciousness	4 (24%)	5 (56%)	2 (67%)	16 (55%)
Decreased muscle strength	10 (59%)	4 (44%)	3 (100%)	22 (85%)
Visual field and acuity deficits	8 (47%)	3 (33%)	0 (0%)	15 (52%)
CN III, IV, or VI deficit	7 (41%)	0 (0%)	2 (67%)	9 (31%)
Headache	11 (65%)	4 (44%)	1 (33%)	16 (55%)
Surgery	13 (76%)	7 (78%)	3 (100%)	23 (79%)
Transsphenoidal	11 (65%)	5 (56%)	2 (67%)	18 (62%)
Craniotomy	2 (12%)	2 (22%)	0 (0%)	4 (14%)
Conservative management	4 (24%)	2 (22%)	0 (0%)	9 (31%)
Improved	10 (59%)	8 (89%)	2 (67%)	20 (69%)
Worsened	1 (6%)	0 (0%)	1 (33%)	2 (7%)
Died	6 (35%)	1 (11%)	0 (0%)	7 (24%)

Values are reported as number (%) except for age.  
CN, cranial nerve.

appropriate only when cerebral infarction is clearly related to vasospasm and may include replacement steroids as well as crystalloids and permissive hypertension.<sup>38,39</sup>

This study has significant limitations owing to the small sample size and selection bias. Because of the rarity of this complication, there are insufficient cases to identify predictors of good outcomes. Furthermore, many studies did not state how they distinguished between compression and vasospasm, although compression was assumed when the hematoma was directly against the narrowed vessel. Moreover, the decision to treat as well as the surgical and medical management of patients varied among providers. There may have been a selection bias with patients treated with surgery presenting with more severe symptoms, large hemorrhages, or larger strokes.

The decision to surgically manage cases of pituitary apoplexy—associated acute ischemic stroke may be difficult owing to unclear etiology. However, as with all

cases of apoplexy, steroid replacement should be initiated early, and emergent surgery should be pursued in cases of deteriorating level of consciousness or visual field or acuity changes.<sup>40</sup> Otherwise, stable or improving symptoms may warrant conservative management only.

**CONCLUSIONS**

Cerebral infarction associated with pituitary apoplexy is a rare but devastating complication that can be related to cerebral vasospasm or compression. Vascular compression from the acute expansion of the tumor is more common and has higher mortality than vasospasm and should be managed with urgent surgical decompression if the patient presents with progressive symptoms.

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*Conflict of interest statement: The authors declare that the article content was composed in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.*

*Received 31 March 2020; accepted 30 May 2020*

*Citation: World Neurosurg. (2020) 141:73-80.  
https://doi.org/10.1016/j.wneu.2020.05.276*

*Journal homepage: [www.journals.elsevier.com/world-neurosurgery](http://www.journals.elsevier.com/world-neurosurgery)*

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