



Case report

Emergency endoscopic surgery for pituitary apoplexy presenting as cerebral infarction in a limited resources condition: A case report

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ABSTRACT

Introduction and importance: Pituitary apoplexy is defined as a sudden onset of neurologic deficit due to infarction or hemorrhage of the pituitary tumor. We report a case of emergency endoscopic surgery for pituitary apoplexy presenting as cerebral infarction due to ICA compression in a limited resources condition.

Case presentation: A 38-year-old female presented with acute onset of severe headache, decreased level of consciousness, decreased visual acuity bilaterally, aphasia, and right hemiparesis. Computed tomography angiography showed a hyperdense sellar mass with stenosis of the left ICA. The patient underwent emergent endoscopic transsphenoidal surgery for sellar decompression.

Clinical discussion: The epidemiology, presentation and diagnosis and strategy of treatments as well as their outcomes were discussed.

Conclusion: Pituitary apoplexy should be taken into consideration in a patient with increasing headache and neuro-ophthalmic symptoms. Pituitary apoplexy presenting as cerebral infarction is rare. The aim of surgery in emergency setting was sellar decompression. Endoscopic transsphenoidal surgery was an effective treatment.

1. Introduction and importance

Pituitary apoplexy is a clinical syndrome presenting as headache, acute decreased visual acuity, acute visual field deficit, and decreased in level of consciousness owing to pituitary gland infarction or hemorrhage [1]. Acute ischemic stroke due to internal carotid artery compression is extremely rare. In emergency setting in some limited resources center, navigation system is unavailable. We report a case of emergent endoscopic transsphenoidal surgery for pituitary apoplexy manifesting as cerebral infarction in a limited-resources condition. We believe this is the first case report on this issue in Vietnam.

2. Case presentation

2.1. Past medical history and physical examination

This case reports a 38-year-old female patient. She suffered from headache, blurred vision in her left eye and amenorrhea for a year. The patient and her family denied either history of surgery, obstetrics and medical diseases or any previous clinical examination.

2.2. Local hospital

While staying at home for 2 days, her headache got worse, and she was admitted to the local hospital. On the local hospital admission, her GCS was 15 points. Her left visual acuity was to count finger in the distance of 20 cm, while her right visual acuity was 10/20 (Snellen chart

Abbreviations: 3D-TOF, 3 dimensions time-of-flight magnetic resonance angiography; ADC, apparent diffusion coefficient; C5–C6, segments of internal carotid artery; clinoid and supra-clinoid segments of internal carotid artery; CT, computed tomography; CTA, computed tomography angiography; DWI, diffusion-weighted imaging; FLAIR, fluid-attenuated inversion recovery; FSH, follicular stimulating hormone; FT3, free triiodothyronine; FT4, free thyroxine; GCS, Glasgow Coma Scale; ICA, internal carotid artery; LH, luteinizing hormone; MRI, magnetic resonance imaging; N/A, not available; PAS, pituitary apoplexy score; SpO₂, oxygen saturation; T3, triiodothyronine; T4, thyroxine; TSH, thyroid stimulating hormone.

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[2,3]), with a left temporal visual field defect was recorded. The PAS was 2 point (Table 2: GCS 15 – 0 point, visual acuity unilateral – 1 point, visual field defect unilateral – 1 point, no ocular paresis – 0 point) [4]. MRI imaging of the brain at the local hospital demonstrated a pituitary tumor (Fig. 1).

After 2 more days in the local hospital, her visual acuity acutely decreased as light perception in both eyes. Visual field examination was unavailable. She had acute weakness in her left upper and lower extremities. She had increasingly difficulty in speaking. Her level of consciousness gradually decreased; and she was transferred to our hospital.

2.3. On examination

On admission to our hospital, she was lethargic with Glasgow Coma Scale of 8 points. Both her pupils were 2 mm, reactive to light. No ptosis was observed. She had right hemiparesis. The PAS was 6+ points (Table 2: GCS 8 – 4 points, visual acuity bilateral decreased – 2 points, visual field defect: N/A, no ocular paresis – 0 points). Her heart rate was 70 beats per minute, her blood pressure was 110/80 mmHg. Her respiratory rate was 20 breaths per minutes, her SpO2 was 90%. Emergency endotracheal intubation was indicated with affirmation from the parent's family members. Her vital signs became stable, SpO2 was 95% with mechanical ventilation.

2.4. Blood count

Her full blood count, her liver and renal function, and sodium level was in normal range. In emergency setting in our center, baseline blood hormone levels and MRI imaging were unavailable.

2.5. Imaging

She was indicated a multi-slice CT Angiography with contrast enhancement. The CTA showed an intrasellar lesion, which was 45 mm × 25 mm × 40 mm in size, infarction inside, anteriorly compressing the optic chiasm, developing into the left middle cranial fossa, directly compressing left internal carotid artery. The clinoid and supragenoid segments (C5–C6 segments) of the left internal carotid artery were narrowed in almost its perimeter. No thrombus was found in the cerebral arteries system. Her left hemisphere appeared hypoattenuation on CT

scan (Fig. 2). She was diagnosed pituitary apoplexy grade 5 (low GCS not allowing testing of visual acuity or field deficit [5]) causing left hemisphere cerebral infarction. An emergency endoscopic transsphenoidal surgery to decompress the sellae was indicated. A bolus dose of 100 mg intravenous hydrocortisone was administered,

2.6. Operation

In the operation, endoscopy transsphenoidal surgery was performed. After the dura mater of the Sella floor was cut, the tumor appeared soft and grey in color macroscopically, infarction inside the tumor was observed. The tumor was removed using pituitary curette and micro suction cannulas (Fig. 3A). In our center, navigation system was unavailable in emergency setting. Without the aid of intraoperative navigation system, the relationship between the tumor and the internal carotid arteries bilaterally was hard to identify. The surgery aim was to decompress the pituitary region, total tumor removal was optional. The sellar diaphragm was observed (Fig. 3B). The procedure was performed by Dr. H.V.D. and his team.

The histologic result confirmed the diagnosis of pituitary adenoma with large areas of necrotic tissues (Fig. 4).

2.7. Post-operation

Post operation, the patient was treated in the Intensive Care unit for 2 weeks. The diagnosis of cerebral infarction due to stenosis of the left ICA was confirmed by post-op images (Figs. 5 & 6). She was administered hydrocortisone (10 mg daily). On discharge, she was alert with the GCS of 15 points. Her right upper and lower limb muscle strength was 0/5. Her right visual acuity recovered to 10/20 as before the acute period. Her right visual field was normal. Her left visual acuity was counting fingers at the distance of 20 cm. Her left visual field was defected at temporal field [6]. No ocular paresis was observed. Her comprehension was intact, while her speech was severely diminished. The pituitary apoplexy was 2/10 points (Table 2: Glasgow coma scale of 15 – 0 point, visual acuity reduced unilateral – 1 point, visual field defect unilateral – 1 point, ocular paresis absent – 0 point). She was administered hydrocortisone 10 mg per day continuously.

On follow-up at 7 weeks post-operation, her mRS was 3 [7]. She was alert. She could walk without assistant. She could take care of herself

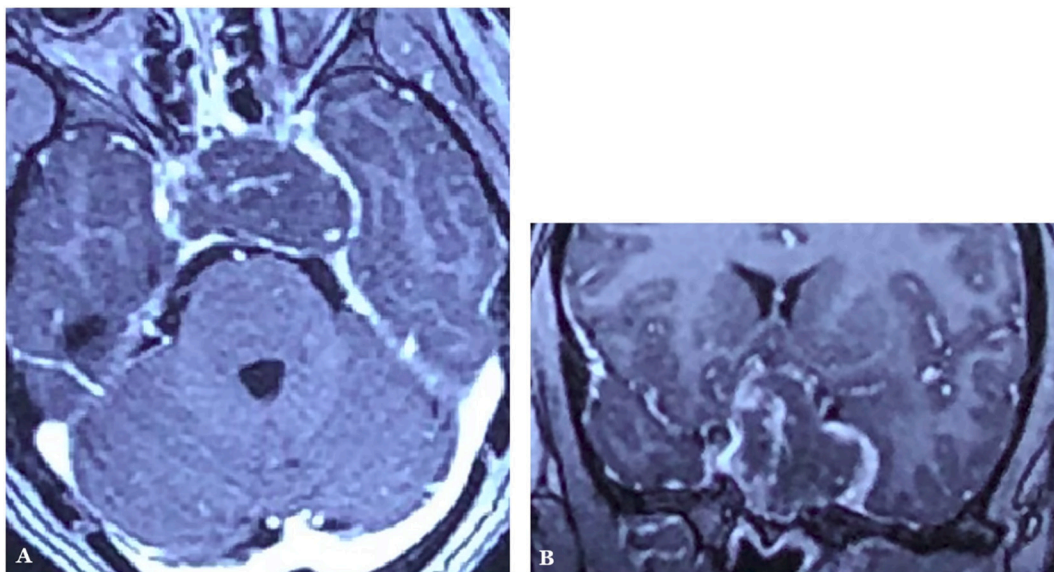


Fig. 1. MRI image at the local hospital 1 day before the operation.
A: The axial T1-weighted image with gadolinium showed a pituitary tumor with left lateral extension.
B: The coronal T1-weighted image with gadolinium showed a pituitary tumor with upper extension.

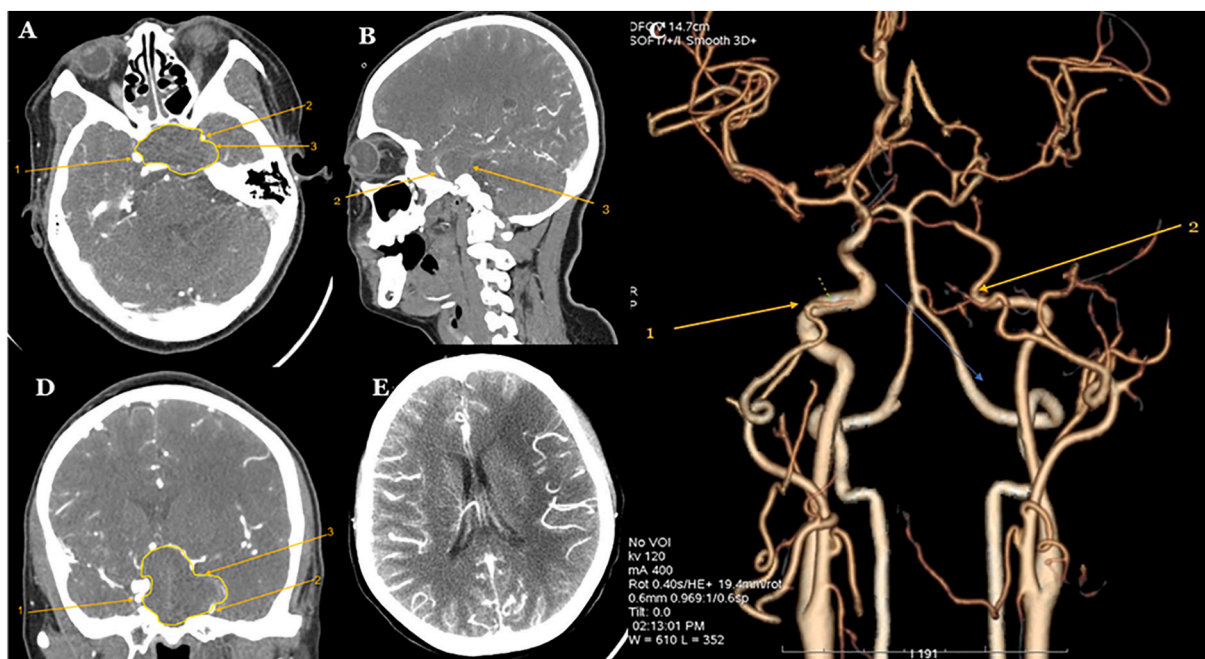


Fig. 2. The computed topography angiography imaging before the operation.

- 1: The right internal carotid artery.
- 2: The left internal carotid artery.
- 3: Pituitary tumor.

A: Axial image with marked tumor. Notice the compression by the tumor of the clinoid and supraclinoid segments (C5–C6 segments) of the left ICA as its comparison to the right ICA.

B: Sagittal image showed the compression by the tumor of the clinoid and supraclinoid segments (C5–C6 segments) of the left ICA.

C: 3-D reconstruction image of the cerebral artery system.

D: Coronal image with marked tumor. Notice the compression by the tumor of the clinoid and supraclinoid segments (C5–C6 segments) of the left ICA as its comparison to the right ICA.

E: Axial image showed hypoattenuation in the left hemisphere. Notes in Fig. 2: The compression of the left ICA by the tumor; the diameter of the left ICA in comparison with the right ICA; no thrombus was found in the cerebral arteries.

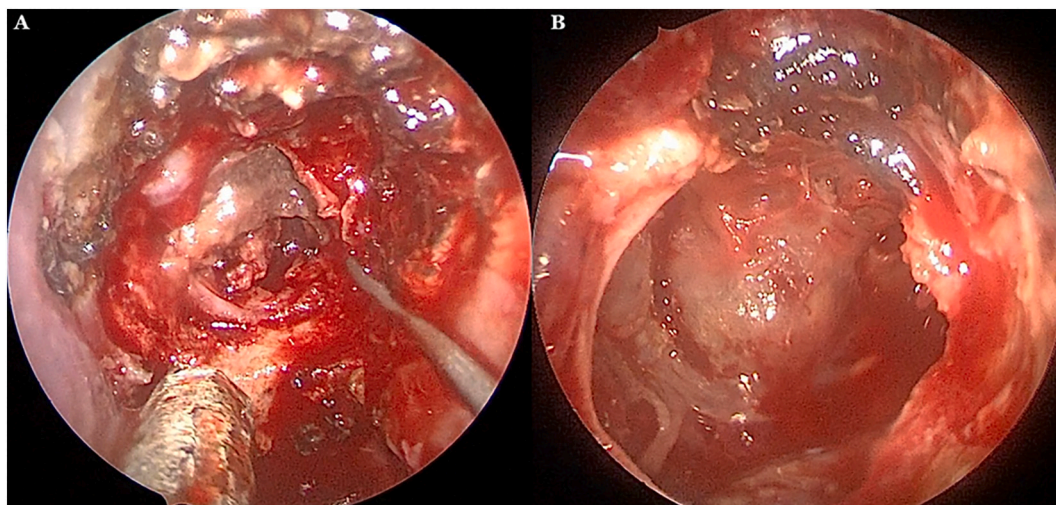


Fig. 3. Intraoperative endoscopic images.

- A: The pituitary tumor. Notice the infarction inside the tumor.
- B: The sellar diaphragm. No residual tumor was observed.

with some assistance from her family. The pituitary apoplexy score was 2/10 points (Table 2). She suffered from mass synergy patterns as flexion of her right upper limb and extension of her lower limb with hyperreflexia in upper and lower limb. Her Broca's aphasia and her vision unvaried [8]. Her cortisol level was 23.71 nmol/L (Table 1). She

was administered oral hydrocortisone 10 mg per day for 1 more month. On follow-up at 4 months post operation, her cortisol level was 93.05 nmol/L (Table 1). Oral hydrocortisone was discontinued permanently. No residual tumor was found on follow-up images (Fig. 7).

This paper has been reported in line with the SCARE 2020 criteria

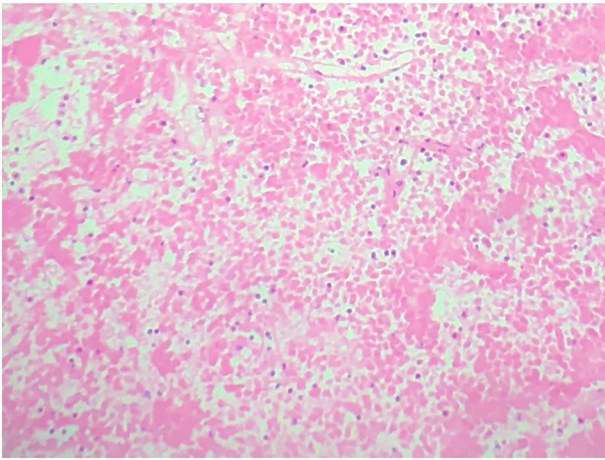


Fig. 4. Microscopic image of the tumor in H&E stain in 40 \times . Notice the mass infarction of the adenoma cells.

[9].

3. Clinical discussion

3.1. Pathophysiology of pituitary apoplexy

Pituitary apoplexy presents as acute pituitary infarction or hemorrhage. Pituitary adenomas have oddly high metabolic request for glucose and amino acid while its vascularity is inadequate. These native features make them susceptible to infarction more common than any other central nervous system tumor [10]. The clinical manifestations of pituitary apoplexy results from a swift enlargement of the intrasellar content, consequently gendering an elevation in the intrasellar pressure [4]. The typical clinical manifestations are headache, decrease in visual acuity, visual field defects, and decreased level of consciousness [1]. Several articles mentioned on the risk factors, including fluctuation in blood pressure, bromocriptine initiation or withdrawal, hormonal stimulation of the pituitary gland, increased intracranial pressure, and head trauma [1,11–13], while tumor of almost any size can undergo apoplexy [13]. In this patient, pituitary apoplexy resulted from pituitary infarction, as shown in intra-operative images (Fig. 3) and histology

image (Fig. 4). No clear risk factor had been found.

3.2. Cerebral infarction and pituitary apoplexy

Acute cerebral infarction originated from pituitary apoplexy is rare. Most common affected artery was the internal carotid artery, followed by anterior cerebral artery, middle cerebral artery and basilar artery [14]. The two primary mechanisms was intracranial blood vessels compression caused by the tumor and blood vessel spasm resulted from tumor bleeding [14]. Angiogram is needed to differentiate between those two mechanisms. Bilateral cerebral arteries are presumably involved in vasospasm mechanism. Emergency surgery was defined as within 7 days of presentation, while delayed surgery was at or more than 7 days after presentation [14]. The optimal timing of surgery was under debate [15]. Some articles were in favor of early decompression surgery, as it may counter visual impairment, ICA blood flow and brain edema [16–18]. Endoscopic transsphenoidal surgery and craniotomy were among surgery options. Endoscopic transsphenoidal surgery patients had a lower mortality rate and improved better [17]. In our patient, the left ICA was compressed by the pituitary tumor causing cerebral infarction. The operation was regarded as emergency as it occurred 4 days following the first sign of the acute period.

3.3. Treatment of pituitary apoplexy in our center

In emergency settings, MRI images and navigation systems were unavailable. The CT Angiography with contrast to assess the tumor and the cerebral vascular system was our best choice to confirm the diagnosis and to guide the endoscopic surgery. Aspirin was not used in treatment of our patient. As shown in Computed Angiography Imaging before the operation, no thrombus was found (Fig. 2). Besides, aspirin uses come along with risk of post-operation hemorrhage. Endoscopic transsphenoidal surgery for pituitary tumor removal was effective. The treatment objective was to decompress the sellar region. Complete pituitary removal by endoscope would be challenging without the aid of navigation system. In this case, without navigation, we decompressed the sellar region and completely removed the pituitary tumor. Post-operative and at follow-ups, the patient recovered as the GCS was 15 points, PAS decreased from 6+/8 points to 2 points. Cerebral infarction was irreversible as her hemiparesis was unvaried.

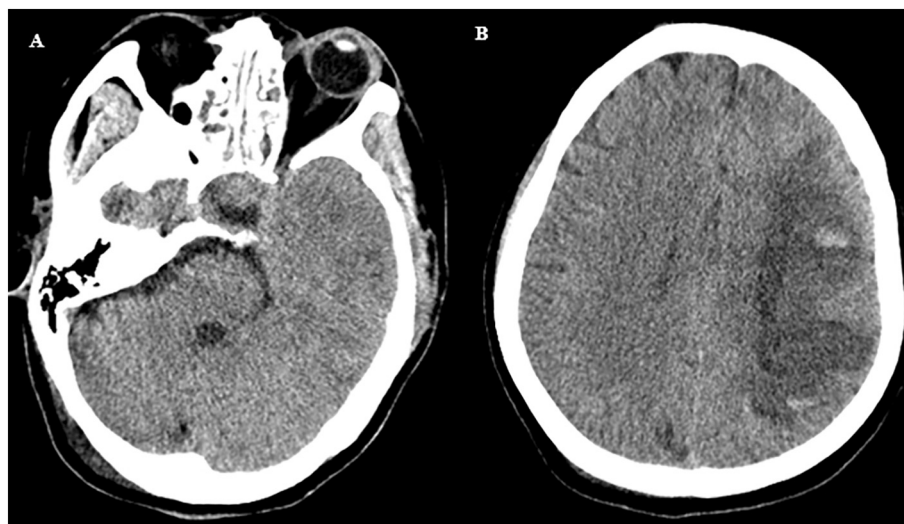


Fig. 5. The CT scan at 5 days post operation.

A: No hemorrhage in the sellar region was observed.

B: The hemorrhage inside the infarction region of the left hemisphere.

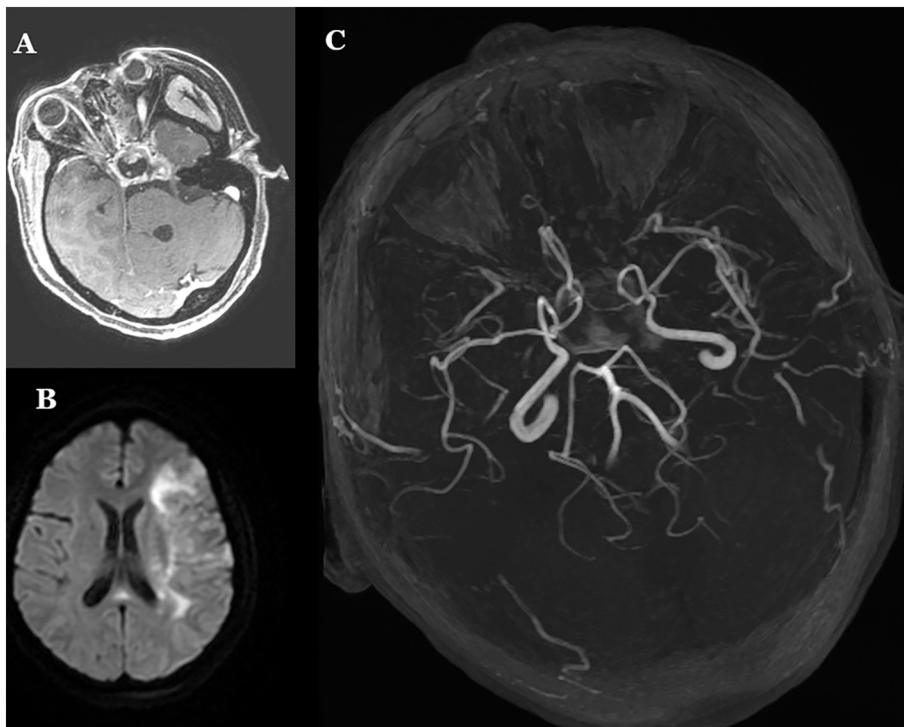


Fig. 6. 10 days post operation MRI images.
 A: T1 with gadolinium images shows no residual tumor.
 B: DWI image showed the left hemisphere infarction.
 C: 3D-TOF image showed the left ICA stenosis.

Table 1
 The level of cortisol, TSH, T3, T4, FT3, FT4 pre-operation, post-operation and at follow-up.

	Preop	1 day postop	5 days postop	10 days postop	7 weeks postop	4 months postop	Unit	Normal level
Cortisol (at 8 AM)	N/A	3484	N/A	N/A	23.71	93.05	nmol/L	AM: 172–497 PM: 74–286
TSH	N/A	0.1993	0.0374	0.0805	0.784	1.4674	mIU/L	0.35–5.5
T3	N/A	0.43	0.78	0.74	1.9	1.37	nmol/L	0.9–2.8
FT3	N/A	<2.30	2.32	N/A	3.81	3.40	pmol/L	3.5–6.5
T4	N/A	5.06	6.89	N/A	6.1	4.34	µg/dL	4.87–11.72
FT4	N/A	12.10	12.26	13.95	8.09	6.53	pmol/L	11.5–22.7

Bold signifies that the TSH level dropped most at 5 to 10 days post-operation.

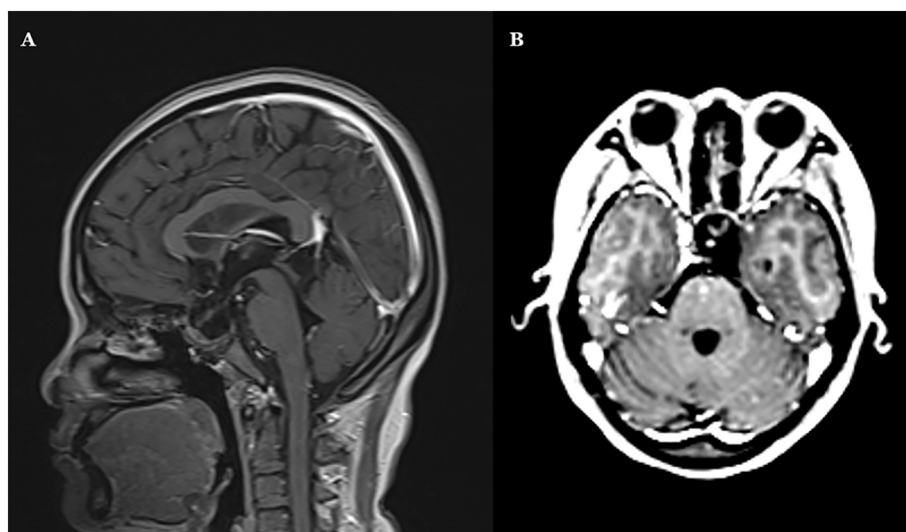


Fig. 7. 4 months post operation.
 A, B: T1 with gadolinium images shows no residual tumor.

Table 2
Pituitary apoplexy score.

Variable	Points	Preop	2-week postop	4 months postop
1. Level of consciousness	• GCS 15	0	0	0
	• GCS 8–14	2		
	• GCS <8	4	4	
2. Visual acuity	• Normal	0		
	• Reduced – unilateral	1	1	1
3. Visual field	• Bilateral	2	2	
	• Normal	0		
	• Defects – unilateral	1	N/A	1
4. Ocular paresis	• Defects – bilateral	2		
	• Absent	0	0	0
	• Present – unilateral	1		
	• Bilateral	2		
PAS total	10	6+/8	2	2

3.4. Hormone level

In our patient, there is a severe decrease in level of TSH at day 5 and day 10 post-operation, while the level of FT4 was in normal range at day1, day 5 and day 10 post-operation (Table 1). This may be due to the storage of thyroid hormones in the blood before the acute period. TSH level became normal at 7 weeks and 4 months follow-up while no treatment was applied (Table 1). Our patient suffered from amenorrhea for one year before our admission. In our treatment course, her level of FSH, LH, estrogen and progesterone were inside normal range. The patient was administered a bolus dose of 100 mg hydrocortisone intravenously pre-operation, a dose of 10 mg hydrocortisone orally 3 months post-operation. After discontinuing the use of oral hydrocortisone for 1 month, at 4 months follow-up, her cortisol level was normal. The use of oral hydrocortisone was ceased permanently. Our patient did not suffer from diabetes insipidus in the treatment course.

4. Conclusion

Pituitary apoplexy should be taken into consideration in a patient with increasing headache and neuro-ophthalmic symptoms. Pituitary apoplexy presenting as cerebral infarction is rare. The aim of surgery in emergency setting was sellar decompression. Endoscopic transphenoidal surgery was an effective treatment.

Provenance and peer review

Not commissioned, externally peer-reviewed.

Consent

Written informed consent was obtained from the patient and her family members for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Availability of data and material

Data is available upon reasonable request and with permission of Viet Duc Hospital. No patient or author details are included in the figures.

Ethical approval

The study was approved by the Research Ethics Committee of Hanoi Medical University. The procedures used in this study adhere to the

tenets of the Declarations of Helsinki.

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Guarantor

Hung Thanh Chu.

Research registration number

Not applied. This was not a first time a new surgical technique or new equipment/technology was used.

CRediT authorship contribution statement

He Van Dong: Conceptualization, Methodology, Investigation, Supervision.

Dat Tran: Conceptualization, Methodology, Investigation, Writing - review & editing, Supervision.

Hung Thanh Chu: Conceptualization, Methodology, Investigation, Writing - original draft, Writing - review & editing, Visualization.

Anh Hoang Pham: Visualization, Writing - original draft, Writing - review & editing.

Xuan Thanh Nguyen: Visualization, Writing - original draft, Writing - review & editing.

Ha Dai Duong: Conceptualization, Resources, Supervision.

All authors contributed to the interpretation of the results, discussed the results. All authors read and approved the final manuscript to submit.

Declaration of competing interest

The authors declared no conflict of interest.

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