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## Case Report

# Unveiling postpartum pituitary apoplexy through atypical presentation: A case report and review of literature ☆☆☆

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## ABSTRACT

Our case details the atypical presentation of postpartum pituitary apoplexy in a 20-year-old female, who exhibited general weakness, dizziness, and brief loss of consciousness following an uncomplicated vaginal delivery. Despite normal vital signs except for bradycardia, imaging revealed a pituitary hemorrhage, leading to the diagnosis of pituitary apoplexy. Managed conservatively with IV hydrocortisone and intensive care, the patient experienced persistent bradycardia and severe abdominal pain, requiring transfer to another ICU. This case highlights the diagnostic challenges posed by the rarity and complexity of pituitary apoplexy during pregnancy and the postpartum period, emphasizing the importance of early diagnosis and tailored treatment strategies. The discussion further contrasts this case with existing literature, particularly in the context of postpartum pituitary apoplexy, and explores the broader implications for managing such rare cases, reinforcing the viability of conservative management in the absence of visual field disturbances.

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## Introduction

Pituitary apoplexy is a rare but serious condition resulting from infarction or hemorrhage within the pituitary gland. The incidence ranges from 0.6% to 9% among patients with pre-existing pituitary tumors, particularly adenomas, which are

benign growths within the gland [1]. This acute event typically occurs when there is a sudden increase in pressure within the pituitary sella, often due to bleeding into or around the tumor. This sudden expansion can compress nearby structures, leading to range of clinical symptoms including a sudden and severe headache, visual disturbances such as vision loss or double vision, altered mental status ranging from confusion

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**Table 1 – Comparison of pretreatment and post-treatment status.**

Lab test	Normal range	Pretreatment value	Post-treatment value	Interpretation
Hemoglobin	12-16 g/dL	11.8 g/dL	13.4 g/dL	Improved
Sodium, serum	135-145 mEq/L	142 mEq/L	139 mEq/L	Within normal range
Potassium, serum	3.5-5.3	4.1 mEq/L	4.2 mEq/L	Within normal range
CRP	< 10 mg/L	10.1 mg/L	3 mg/L	Significant reduction
ESR	< 20 mm/hr	50 mm/hr	13 mm/hr	Significant reduction
Prolactin	5.18-26.53 ng/mL	58 ng/mL	22 ng/mL	Normalized after treatment

to unconsciousness, and endocrine dysfunction due to pituitary failure. The severity is generally proportional to the extent of bleeding and the resulting pressure on adjacent brain structures, such as the optic chiasm and hypothalamus [2,3].

Diagnosis requires clinical suspicion, laboratory tests, and imaging studies. CT and MRI are crucial for confirming the diagnosis by revealing bleeding or infarction within the pituitary region. Treatment involves surgical intervention to relieve pressure and medical management to address hormonal imbalances and prevent further complications [4,5].

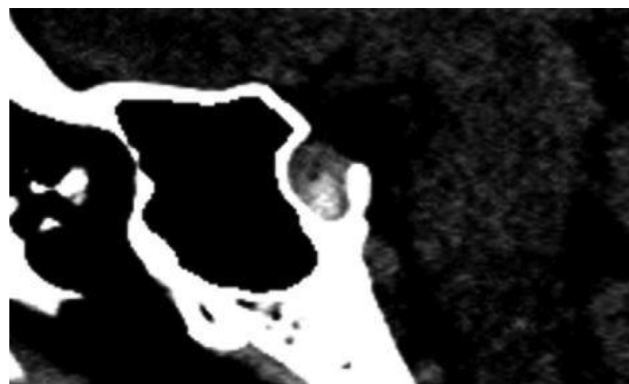
In this case, we are reporting a 20-year-old female presented with weakness, dizziness, and a brief loss of consciousness following an uncomplicated vaginal delivery. Despite normal vital signs, except for bradycardia, a brain CT revealed a pituitary hemorrhage, leading to a diagnosis of pituitary apoplexy. She was treated with IV hydrocortisone and monitored in the ICU. Despite treatment, she experienced persistent bradycardia and severe abdominal pain, necessitating further evaluation and transfer to another ICU for continued critical care.

## Case presentation

A 20-year-old female presented to the emergency room with general weakness, dizziness, and a 30-minute episode of loss of consciousness. She had recently undergone an uncomplicated vaginal delivery with epidural anesthesia. A week after Delivery, she experienced persistent dizziness, chills, and abdominal and back pain but had no history of seizures. Vital signs were largely normal, except for bradycardia (60 beats per minute). Her blood pressure was 123/72 mmHg, oxygen saturation 98%, and she was afebrile. Initial lab investigations were conducted (see Table 1).

Given her bradycardia, hypotension, and severe abdominal pain, differential diagnoses included adrenal insufficiency secondary to pituitary apoplexy (Sheehan's syndrome), gastrointestinal conditions, and cardiovascular issues like neurocardiogenic syncope and myocardial infarction. A brain CT revealed an abnormal hypodense focus in the left posterior pituitary gland, suggesting hemorrhage consistent with pituitary apoplexy, leading to the final diagnosis (see Fig. 1).

The neurosurgery team recommended against immediate surgery. Further imaging with a brain MRI showed a small, well-defined hypointense area in the left posterior pituitary gland on T2-weighted imaging, with no contrast enhancement, confirming pituitary apoplexy (see Fig. 2). A full en-



**Fig. 1 – Head CT scan: Sagittal plane with a craniocaudal view shows a hyperdensity within the left posterior aspect of the pituitary gland, suggesting hemorrhage.**

docrine workup was initiated to assess pituitary function, including thyroid and gonadal axes (see Table 1). Given the risk of adrenal insufficiency, early administration of stress-dose corticosteroids was considered.

The patient was transferred to the ICU for close monitoring and received IV hydrocortisone. Despite these interventions, she continued to experience episodes of bradycardia, with heart rates as low as 40 beats per minute. An ECG showed sinus bradycardia, prompting a thorough cardiac evaluation, including echocardiography. However, she did not exhibit neurological deficits such as headaches, diplopia, or visual impairment, and her muscle power remained normal.

As her condition progressed, she developed severe headaches, diplopia, and worsening bradycardia, with blood pressure rising. Pain management included morphine and IV Paracetamol. Given persistent severe abdominal pain, further diagnostic imaging, including an abdominal ultrasound and CT scan, was recommended, but these did not reveal any acute surgical pathology, suggesting a non surgical cause related to her endocrine dysfunction.

Over the next few days, her symptoms improved with continued ICU care, IV hydrocortisone, and supportive medications. Bradycardia and hypotension resolved, and subsequent imaging confirmed resolution of the pituitary hemorrhage. A full endocrine workup indicated normal pituitary function. As her condition stabilized, she transitioned to oral hydrocortisone. Abdominal pain resolved without surgical intervention, and by discharge, she was fully ambulatory, with stable vital



**Fig. 2 – Brain MRI, Sagittal Plane: (A) T1-weighted imaging without contrast shows an isointense pituitary gland with no visible lesions. (B) T2-weighted imaging reveals a small, well-defined hypointense area in the left posterior aspect of the pituitary gland. (C) After IV contrast administration, T1-weighted imaging shows no enhancement of this area, which is highly suggestive of apoplexy.**

signs and no neurological deficits. She was discharged with a follow-up plan for periodic endocrine and neurosurgical evaluations to monitor her recovery.

## Discussion

Pituitary apoplexy (PA) is an uncommon and potentially perilous disorder, particularly rare during pregnancy, and is characterized by ischemic infarction or hemorrhage of the pituitary gland [6]. Importantly, in many cases, PA occurs spontaneously, as in our case, or is associated with pituitary tumors. Moreover, several risk factors for PA have been identified, including pregnancy, which significantly increases the risk due to pituitary gland enlargement caused by the massive hyperplasia of lactotroph cells driven by elevated estrogen levels [7]. Other factors include head trauma, hypotension, major surgery, anticoagulants, a history of irradiation, and hypertension [8,9]. Additionally, the most frequent clinical presentation of PA includes the abrupt onset of retro-orbital headache, vomiting, visual disturbances, and altered levels of consciousness, with the second, third, fourth, and sixth cranial nerves most commonly affected [10]. Furthermore, MRI is the definitive diagnostic tool for pituitary apoplexy, particularly in pregnant patients, as it is safe and has minimal harmful effects on the fetus, confirming more than 90% of cases. Specifically, hyperintensity on T1-weighted images is indicative of hemorrhage [11,12].

Herein, there are 2 scenarios regarding pituitary apoplexy mechanisms. The most frequent is that the intrinsic vascularization of the adenoma is more fragile and less mature compared to the normal pituitary gland which is supplied by the portal system. As the tumor's metabolic requirements increase, it may outstrip its blood supply. Any reduction in systemic blood pressure may lead to reduced blood flow to the pituitary adenoma. The second scenario involves compression of the superior hypophyseal vessels by tumor expansion or pregnancy-induced pituitary gland hyperplasia [13,14].

Depending on the location and extent of infarction, patients may develop deficiencies in 1 or more anterior pituitary hormones, most commonly gonadotropins, followed by ACTH

and TSH, and less commonly prolactin. Since gonadotropins are physiologically suppressed during pregnancy, their deficiency is challenging to evaluate [15]. Adrenal insufficiency is a fatal complication and a high-risk condition [6]. Therefore, whenever pituitary apoplexy is considered, a full endocrine evaluation (free T4, TSH, cortisol, ACTH, prolactin, FSH, LH, insulin-like growth factor 1) and blood assessment (CBC, electrolyte panel, and renal and liver function tests) should be urgently ordered [16].

Initial management of pituitary apoplexy consists of administering fluids, correcting electrolytes, and hormonal replacement. When pituitary apoplexy is suspected, glucocorticoids must be administered to avoid the risk of adrenal insufficiency. UK guidelines recommend an intravenous bolus of 100-200 mg hydrocortisone, followed by a 2-4 mg/h intravenous continuous infusion or intramuscularly 50-100 mg every 6 hours. Once the acute episode is overcome, a standard maintenance dose of 20-30 mg is used. Surgery is indicated if there is no clinical improvement or deterioration [10,16].

When comparing PA with Sheehan's syndrome, it is important to note that Sheehan's syndrome typically arises from hypotension or hypovolemic shock resulting from massive hemorrhage during or after childbirth. The prevalence of PA is estimated to be approximately 6.2 cases per 100,000 people. In contrast, the prevalence of Sheehan's syndrome varies significantly based on the quality of maternal healthcare. This condition is more commonly observed in regions with suboptimal delivery conditions and in countries where adequate medical follow-up for pregnant women is lacking [17].

Based on a review of the literature, 43 cases of pregnancy-related pituitary apoplexy were identified. The average gestational age at the time of admission was 27.9 weeks, according to statistical analysis of Table 2. With the exception of four cases, including ours, all occurred postpartum. As mentioned by Janssen et al., [18] pituitary apoplexy can occur in the first trimester, though this is very rare. It is worth noting that, the first case occurred after spinal anesthesia, as mentioned by Mathur et al., [19] and was managed conservatively. The second case, published by Perotti and Dexter, was diagnosed following spontaneous delivery and treated with transphenoidal craniotomy [20]. The third case, described by Paech et al., [21] was diagnosed after delivery and managed conser-

**Table 2 – Review of over 42 published articles related to our study.**

References	GA	Presenting symptoms	Treatment	Outcome
1 [23]	22	Headache, nausea and vomiting	Surgery	Hypothyroidism
2 [25]	32	severe headache and deterioration of the visual field	Surgery	Hypothyroidism
3 [26]	36 + 5	headaches and left-sided visual loss	Surgery	Full recovery
4 [27]	37 + 4	severe headaches	Conservative	Full recovery
5 [28]	29	Headache	Surgery	Diabetes insipidus
6 [29]	28	Headache, nausea and vomiting	Conservative	N/M
7 [30]	23	right-sided headache, associated with photophobia and right-sided numbness	Surgery	N/M
8 [31]	39	severe headache	Conservative	Full recovery
9 [32]	37 + 4	sudden onset of visual disturbance and severe occipital headache	Conservative	Full recovery
10 [24]	24	Headache, nausea and vomiting	Surgery	Panhypopituitarism
11 [33]	28	Severe headache	Conservative	Partial hypopituitarism
12 [34]	N/M	Headache	N/M	
13 [7]	35	Headache	Surgery	Central hypothyroidism
14 [35]	18	visual disturbance and headache	Surgery	Full recovery
15 [36]	36	severe onset headache and acute left-sided vision loss	Surgery	Temporary diabetes insipidus
16 [37]	19	headaches with visual disorders (blindness of the right eye)	Conservative	Resolution of visual symptoms
17 [19]	Postpartum	intense headache	Surgery	Reversible cerebral constrictive syndrome
18 [38]	26	sudden onset of headache and bitemporal hemianopsia	Surgery	Diabetes insipidus
19 [39]	19	Headache, nausea and vomiting	Surgery	Full recovery
20 [18]	10	continuous headache and visual complaints of the left eye	Conservative	Adrenal insufficiency
21 [40]	19	severe headache, nausea, vomiting, and blurred vision	Conservative	Full recovery
22 [41]	36	headache, vision disturbance	Conservative	Panhypopituitarism
23 [42]	39	nausea, vomiting, and general fatigue	Conservative	Panhypopituitarism
24 [43]	23	nausea, vomiting	Conservative	Full recovery
25 [44]	29	Headache, nausea and vomiting	Conservative	Full recovery
26 [45]	39	headache and nausea	Surgery	Full recovery
27 [20]	Postpartum	Glasgow Coma Scale score of 3, fixed dilated pupils, complete ophthalmoplegia	Surgery	Panhypopituitarism
28 [46]	20	headache and vision loss	Surgery	Full recovery
29 [22]	33	worsening blurred vision and generalized headache	Conservative	Death
30 [47]	22	severe headache and nausea	Conservative	Miscarriage at 9th week, diabetes insipidus
31 [48]	7	sudden coma	Conservative	Panhypopituitarism, diabetes insipidus
32 [49]	29	Headache, nausea and vomiting	Conservative	Panhypopituitarism, diabetes insipidus
33 [21]	29	partial oculomotor nerve palsy	Surgery	Panhypopituitarism
34 [50]	Postpartum	headache and bitemporal hemianopsia	Conservative	Full recovery
35 [6]	34	sudden onset of severe headache, vomiting, disturbed consciousness and photophobia	Conservative	Adrenal insufficiency
36 [51]	23	unilateral visual loss and intermittent headaches	Conservative	Panhypopituitarism
37 [52]	30	sudden vision loss and headache	Surgery	Full recovery
38 [53]	28	Polyuria	Surgery	Full recovery
39 [54]	32	visual acuity disturbances	Surgery	Diabetes insipidus
40 [55]	24	Headache, nausea and vomiting	Surgery	Full recovery
41 [56]	8	transient paresis of the left abducens nerve	Surgery	Left sided cranial nerve palsy
42 [16]	23	headaches, nausea, and photophobia	Surgery	Resolution of visual symptoms
43 (our case)	Postpartum	Headache, nausea and vomiting	Surgery	Panhypopituitarism
	Postpartum		Conservative	Full recovery

Table 1: GA (Gestational age).

vatively. As mentioned by Lucian Gheorghe Pop, the fourth case occurred after a cesarean section and showed no improvement with conservative treatment, requiring endoscopic trans-sphenoidal resection [16]. We encountered only 1 case that ended in patient death after the development of hypertension, encephalopathy, and cardiac arrest [22]. According to statistical analysis of Table 2, headache was the most frequent symptom, present in 76.7% of cases, followed by visual disturbances (48.8%), nausea and vomiting (41.8%), and, rarely, altered level of consciousness (6.9%), as in our case [16].

As the patient stabilized, the primary decision was whether to pursue conservative treatment or surgery. An analysis of 22 cases from Table 2 demonstrates that surgical decompression during pregnancy, particularly in the second trimester, can be performed safely without teratogenic effects or alterations in the pregnancy course, as reported by Oguz et al. [23] and Querol Ripoll et al. [24]. In contrast, an analysis of conservatively managed cases confirmed that this approach is viable and safe, especially in patients without visual field disturbances.

Among the 43 cases reviewed, 55.3% were treated conservatively, while 44.7% underwent surgical intervention. Overall, 36.5% of patients experienced a full recovery, whereas 63.5% developed some degree of pituitary dysfunction or complications, such as diabetes insipidus, panhypopituitarism, or even death. Specifically, 17 cases, including the current 1, achieved complete recovery without endocrinological disturbances, whereas 21 cases developed varying degrees of insufficiency, including diabetes insipidus and panhypopituitarism.

## Conclusion

To the best of our knowledge, our case is the fifth documented case of postpartum pituitary apoplexy. To date, there are no specific guidelines for the optimal management of pituitary apoplexy during pregnancy. However, our case confirms the effectiveness of conservative treatment as a successful trial without relapse. We believe this underscores the importance of early diagnosis, as it enables timely intervention and alleviates potential morbidity and mortality. Although headaches are commonly benign in pregnancy and postpartum, they may indicate a critical issue.

## Ethics approval

Our institution does not require ethical approval for reporting individual cases or case series.

## Patient consent

Written informed consent was obtained from the patient's herself for her anonymized information to be published in this article.

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