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Pituitary apoplexy; a review on the etiology, management and outcomes

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Abstract

Pituitary apoplexy (PA) is an uncommon but urgent medical condition characterized by sudden hemorrhage or infarction within a pituitary adenoma, a benign tumor of the pituitary gland. This event constitutes a critical endocrine and neurosurgical emergency, demanding rapid diagnosis and intervention to prevent serious complications or death. The underlying pathogenesis of PA involves vascular compromise within the tumor microenvironment, often precipitated by a combination of systemic risk factors such as hypertension or anticoagulation therapy, and tumor-specific features including rapid tumor growth or fragile blood vessels. Clinically, PA typically presents with a sudden onset of severe headache, visual disturbances resulting from optic chiasm compression, and varying degrees of hypopituitarism leading to acute hormonal deficiencies, which can include adrenal insufficiency and hypothyroidism. These symptoms develop swiftly, making clinical suspicion crucial for early detection. Diagnosis is primarily based on neuroimaging, with magnetic resonance imaging being the gold standard to identify hemorrhage or infarction within the adenoma. Concurrent hormonal assays are essential to assess pituitary function and guide urgent endocrine management. Treatment begins with immediate stabilization, including correction of fluid and electrolyte imbalances and administration of high-dose corticosteroids to address potential adrenal crisis. The decision between surgical decompression and conservative management hinges on the severity of neurological impairment and imaging findings. Surgical intervention is reserved for patients with severe visual deficits or deteriorating consciousness, while stable cases may be managed conservatively with close monitoring. With advances in multidisciplinary approaches involving endocrinologists, neurosurgeons, and radiologists, patient outcomes have significantly improved. Visual recovery is often favorable, but many patients experience persistent endocrine dysfunction requiring long-term hormonal replacement. Ongoing research aims to refine risk stratification criteria and develop individualized treatment protocols to further improve prognosis and quality of life in patients with PA.

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Introduction

Pituitary apoplexy (PA) is an uncommon but serious medical emergency characterized by the sudden hemorrhage or infarction within the pituitary gland, typically occurring in the context of a preexisting pituitary adenoma (1). It represents a clinical syndrome with acute onset of headache, visual disturbances, ophthalmoplegia, altered mental status, and endocrine dysfunction (1,2). The condition requires rapid diagnosis and appropriate management to optimize outcomes and prevent permanent neurological or hormonal deficits (3). Etiologically, this condition most often arises within pituitary adenomas, especially non-functioning macro-adenomas, which comprise the majority of cases (4). The underlying mechanism involves sudden expansion of the tumor due to hemorrhage or ischemic necrosis within the confined space of the sella turcica (5). This condition leads to compression of the normal pituitary gland, the optic chiasm, and adjacent neurovascular structures (5,6). Risk factors identified include hypertension, diabetes mellitus, dyslipidemia, and other cardiovascular comorbidities, which have been found at higher prevalence in PA patients relative to the general population (7). Additional precipitants may include events or conditions causing rapid fluctuations in blood pressure or pituitary perfusion, such as major surgery, anticoagulation therapy, pituitary stimulation tests, head trauma, pregnancy, and infections that cause thrombocytopenia, as reported in rare cases related to dengue fever-induced thrombocytopenia (8,9). Overall, the exact trigger often remains unidentified, but it appears to be the interplay of tumor biology and systemic factors that precipitate sudden vascular events within the pituitary adenoma (4,10).

Key point

The pathophysiology of pituitary apoplexy is ill-understood; however, it is believed that rapidly growing tumors can outgrow their blood supply, leading to ischemic infarction, or compression of surrounding vessels. Pituitary adenomas generally have less vascularization, making them susceptible to ischemia when blood supply decreases, such as during an acute blood pressure fall. It is possible that, an inherent fragility of tumor vessels or compression of the superior hypophyseal artery. Moreover, it is postulated that, the high metabolic demand and marginal tumor perfusion in pituitary adenomas create a tenuous balance, making them vulnerable to ischemia and infarction with any alteration to this balance.

Search strategy

For this narrative review, we conducted a literature search across multiple databases, including PubMed, Google Scholar, the Directory of Open Access Journals (DOAJ), Web of Science, EBSCO, Scopus, and Embase, using a variety of relevant keywords such as pituitary apoplexy, brain, pituitary gland, sella turcica and neurohypophysis

Pathophysiology of pituitary apoplexy

The pathophysiology of PA begins with the unique vascular anatomy of the pituitary gland, which renders it particularly vulnerable to ischemic and hemorrhagic events (11,12). The anterior pituitary, or adenohypophysis, derives its blood supply almost exclusively from the hypophyseal portal system, a low-pressure venous network originating from the superior hypophyseal arteries (4,13). Unlike most other endocrine glands, it lacks a direct arterial supply, making it susceptible to infarction under conditions of increased metabolic demand or reduced perfusion (4). The posterior pituitary, or neurohypophysis, receives direct arterial supply from the inferior hypophyseal arteries and is thus less commonly involved in apoplectic events (12,14). Pituitary adenomas, especially macro-adenomas (those greater than 10 mm in diameter), are the most frequent substrate for apoplexy (15). These tumors often outgrow their vascular supply, leading to areas of necrosis and fragility (16). The tumor's expanding mass can compress surrounding vasculature, including the portal vessels, further compromising perfusion (11,17). Additionally, the tumor may induce mechanical distortion of the diaphragma sellae, increasing susceptibility to vascular shear stress (18). Intra-parenchymal hemorrhage can occur spontaneously or be precipitated by a variety of triggers, including systemic hypertension, anticoagulant therapy, major surgery, dynamic endocrine testing (such as insulin tolerance tests), pregnancy, head trauma, or even vigorous Valsalva maneuvers (19-21). In some cases, dopamine agonist therapy for prolactinomas has been implicated, possibly due to rapid tumor shrinkage leading to vascular disruption (22,23). Rarely, apoplexy may occur in non-adenomatous pituitary tissue, such as in Sheehan's syndrome postpartum, or in association with systemic conditions like sickle cell disease, leukemia, or lymphocytic hypophysitis (4,11,24,25). Mostly important is that, PA often arises spontaneously, and many patients present without a prior diagnosis of a pituitary tumor (12,17). However, various factors can trigger this condition in a notable proportion of cases. These precipitating elements include arterial hypertension, the use of anticoagulant medications, major surgical procedures, and pregnancy (26-30). Hypertension is frequently observed as an associated factor in patients experiencing PA (26). Similarly, antithrombotic or anticoagulant therapies are recognized as potential contributing elements (27). Alterations in blood pressure or intracranial pressure, which can result from events like head trauma, lumbar puncture, or spinal anesthesia, may also lead to apoplexy (28). Furthermore, certain medical interventions, such as pituitary stimulation tests involving GnRH or TRH, or the administration of GnRH analogues, can induce an acute increase in the size of the pituitary gland, thereby precipitating apoplexy (29). In pregnant women, gestational PA is a rare occurrence that can be linked to the cessation of dopamine agonists in those with macroprolactinomas or to the physiological changes that occur within the pituitary during gestation (30).

Clinical presentation of pituitary apoplexy

Clinically, PA presents abruptly with a constellation of symptoms. The hallmark is a sudden, severe headache described as the worst ever experienced (31). This is often accompanied by nausea, vomiting, visual deficits including visual field defects, detected as the bitemporal hemianopsia being classic due to chiasmal compression, ophthalmoplegia from cranial nerve palsies, and altered consciousness ranging from confusion to coma (5). Hormonal deficiencies are common and can be acute or develop over time, involving anterior pituitary insufficiency or posterior pituitary dysfunction manifesting as diabetes insipidus (12,32). Endocrine emergencies such as acute secondary adrenal insufficiency can arise due to corticotropic failure, necessitating prompt glucocorticoid replacement (33,34). The diagnosis is supported by neuroimaging, with magnetic resonance imaging being the gold standard due to its superior soft tissue contrast that allows detection of hemorrhage, infarction, and sellar mass effect (6,35,36). Computed tomography may be used initially in emergencies but lacks sensitivity (6). Laboratory assessment includes pituitary hormone profiling to identify deficiencies and guide replacement therapy (5).

Diagnosing of pituitary apoplexy

Diagnosing PA requires a high index of suspicion, especially given its varied clinical appearance (11,28). Imaging is crucial for confirmation, with magnetic resonance imaging (MRI) being the modality of choice (6). MRI can identify PA in up to 91% of cases, compared to 28% with CT scans (37). In PA, MRI is much more

effective at detecting hemorrhage compared to CT scans (6). While CT is useful in the acute setting (first 24-48 hours), MRI is superior for identifying blood components in the subacute setting (4 days to 1 month) and characterizing the pituitary lesion (38). Characteristic MRI findings include hyper- and hypointense areas on T1 and T2 weighted images, respectively, suggesting apoplexy (6). The presence of a pituitary ring sign with gadolinium enhancement in the peripheral rim may also be seen, though it is not specific (6,28).

Management of pituitary apoplexy

Management of PA is a medical emergency requiring immediate attention and a multidisciplinary team approach involving endocrinologists, neurosurgeons, and neuro-ophthalmologists (5,11). The initial management always includes supportive therapy, such as careful assessment of fluid and electrolyte balance and immediate administration of high-dose corticosteroids, which can be life-saving (12). Specifically, hydrocortisone 50 mg every 6 hours or 200 mg/24 hours continuously or intermittently, is recommended. After initial improvement, the glucocorticoid dose is gradually tapered (5). The decision between surgical decompression and conservative management remains controversial and depends on the patient's clinical manifestations and the presence of neuro-ophthalmic signs (39). Urgent surgical intervention is generally indicated for patients with severe or progressive visual impairment (acuity or fields), altered mental status, or hypothalamic dysfunction (40). As an example, patients with visual field defects are more likely to undergo surgery (41). In a previous study, 61 out of 87 patients (70.1%) underwent surgery, with a median time of 5 days from apoplexy (42). Conservative management, involving monitoring fluid and electrolyte levels and intravenous glucocorticoids, can be effective for selected patients, particularly those without neuro-ophthalmic signs, with mild and non-progressive signs, or isolated cranial nerve palsies (38,43). In a study of 45 patients by Sibal et al, 18 (40%) were managed conservatively (37). Another study found that about one-third of patients could be treated conservatively (5). Conservative management has been shown to work well for the majority of patients in some cohorts, with only a small percentage requiring acute surgical intervention (5). Frequent reassessment is mandatory for conservatively managed patients due to the unpredictable clinical course, and if symptoms progress, later elective surgery may be indicated (34). Scoring systems like the PA score or a grading system from grade one to five can help guide therapeutic decisions, with higher scores typically suggesting surgery (5). The outcomes of PA are generally excellent for most patients (42). Complete or near-complete resolution of visual acuity, visual field deficits, and ocular palsy occurred in over 93% of surgically treated patients in one study (37). All patients with these deficits in the conservative group also showed complete or near-complete recovery in the same study (37). Similarly, another study reported complete resolution or substantial improvement in eye movements and visual fields at follow-up for all survivors (42). However, the endocrinological outcome is less favorable, irrespective of the treatment option, with many patients remaining on long-term hormone replacement therapy (34). In PA, the majority of patients in one series required subsequent hormonal replacement therapy, and in another study, many patients needed long-term levothyroxine and cortisol replacement. Once pituitary deficiencies develop, they usually do not recover. Recurrence of pituitary adenoma has been observed in a small number of patients, emphasizing the importance of long-term surveillance (5,43-45). Overall, a multidisciplinary approach and proper evaluation with long-term follow-up are essential for optimal patient outcomes (5,46).

Conclusion

Pituitary apoplexy is a rare but potentially life-threatening clinical syndrome resulting from acute hemorrhage or infarction within the pituitary gland, most commonly occurring in the setting of a pre-existing pituitary adenoma. This abrupt vascular event leads to rapid enlargement of the tumor and compression of surrounding structures, precipitating a striking clinical presentation. Patients often experience sudden onset of severe headache, visual disturbances such as visual field defects or decreased acuity, ophthalmoplegia caused by cranial nerve involvement, and frequently altered mental status. Signs of acute pituitary hormone insufficiency, including adrenal crisis and hypothyroidism, commonly complicate the clinical picture, accentuating its medical urgency.

Though uncommon, PA requires immediate recognition and management to prevent irreversible neurological damage, permanent hypopituitarism, or death. The etiology is multifactorial; it involves vascular instability within the adenoma driven by tumor growth, vascular occlusion, or systemic factors like hypertension, coagulopathy, or major physiological stress. Mechanical factors such as rapid tumor expansion can also contribute to this acute event. Management has evolved considerably, moving from an almost exclusively surgical approach to more refined, individualized treatment strategies. Current protocols emphasize urgent stabilization with corticosteroids to treat adrenal insufficiency and reduce cerebral edema, alongside close neuroendocrine and neurological monitoring. Surgery is reserved for patients with severe or progressive neurological deficits, while select stable patients may be managed conservatively. Prognosis largely depends on the initial severity and how quickly treatment is initiated. Although visual recovery is generally good with prompt care, persistent endocrine dysfunction is frequent, necessitating lifelong hormone replacement therapy and regular follow-up. Continuous

advances in multidisciplinary care and personalized treatment aim to further improve patient outcomes and minimize long-term complications.

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Authors' contribution

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Ethical issues

Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the authors.

Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this work, the authors utilized Perplexity to refine grammar points and language style in writing. Subsequently, the authors thoroughly reviewed and edited the content as necessary, assuming full responsibility for the publication's content.

Conflicts of interest

The authors declare that they have no competing interests.

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