

# A Case Report on Pseudodepression: 37-year-old Male with Suprasellar Mass

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**Abstract:** Craniopharyngioma is a rare benign epithelial tumor that typically occurs in a bimodal age distribution and intracranial tumors commonly present with signs of increased intracranial pressure due to the mass effect. Additionally, craniopharyngiomas often manifest as pituitary and/or hypothalamic dysfunction as well as visual impairment. This case report will discuss an unusual case of craniopharyngioma in a middle-aged male who presented with pseudodepression for 4 months prior to diagnosis. Eventually, the patient developed headache and decreasing sensorium. He underwent excision of tumor during admission and histopathologic diagnosis showed adamantinomatous craniopharyngioma which has a relatively good prognosis but with a high recurrence rate. Hypothetically, the disruption of the neural connections between the hypothalamus and limbic system in craniopharyngiomas cause the psychiatric symptoms. The gradual onset of psychological symptoms and mass effect may be attributed to the benign nature of craniopharyngiomas. The salient features of this case emphasize the importance of having a high index of suspicion for an organic neurological etiology in cases of new onset psychiatric symptoms without the presence of lateralizing signs.

**Keywords:** Craniopharyngioma, Pseudodepression, Sella or Suprasellar Tumors

## 1. Introduction

Craniopharyngiomas is a rare benign epithelial tumor that typically occurs in a bimodal age distribution. Intracranial tumors commonly present with signs of increased intracranial pressure due to the mass effect. Additionally, craniopharyngiomas often manifest as pituitary and/or hypothalamic dysfunction as well as visual impairment. This case report will discuss an unusual case of craniopharyngioma in a middle-aged male who presented with pseudodepression.

## 2. Presentation

### 2.1 History

A 37-year-old male presented with a 4 months history of being socially withdrawn, anhedonic. He had lack of motivation, delay in responses, and memory lapses of recent events. Progressively, he complained of headaches with blurring of peripheral vision. He had decreasing appetite and weight loss over 2 months and increasing drowsiness. He was unresponsive to escitalopram and fluoxetine. Mini-mental status exam was 26/30, Montreal Cognitive Assessment 24/30, Frontal Assessment Battery 16/18, Hamilton Depression Scale 13/52 (mild depression), Geriatric Depression Scale 10/15 (moderate depression). Glabellar and palmomental reflexes were noted. He was well-groomed with blunted affect, neutral mood, slow, monotonous and fluent speech, organized thought process, no suicidal ideation, oriented to person and place but not to time, poor recent and immediate memory. Physical examination was otherwise unremarkable with no noted lateralizing signs.

### 2.2 Management

MRI of the brain with contrast showed a 4.9 x 3.7 x 3.9 cm T1-weighted mixed intensity, T2-weighted hyperintense lobulated suprasellar mass with minimal anterior peripheral contrast enhancement. The mass compresses the floor of the third ventricle and obscures the hypothalamus. The mass extends posterior into the interpeduncular and right prepontine cisterns with compression of the midbrain. The supraclinoid segments of both ICA are inseparable with the mass. MR features suggestive of a craniopharyngioma. See Figures 1-4.

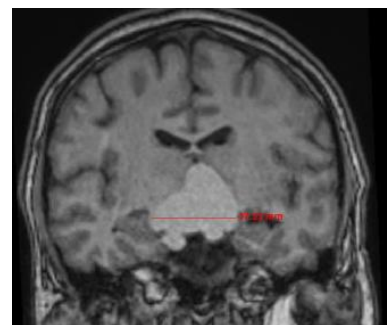


Figure 1: Coronal view, T1-weighted image



Figure 2: Sagittal view, T1-weighted image

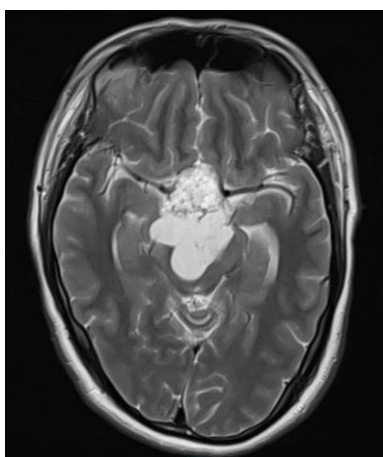


Figure 3: Axial view, T2-weighted image

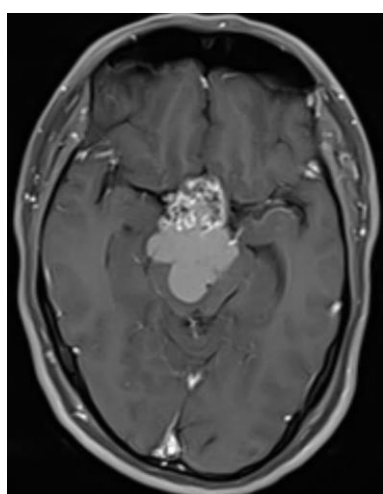


Figure 4: Axial view, T1-weighted contrast image

Electroencephalogram recording done showed focal theta and delta waves on the left frontotemporal region with no epileptiform discharges. Hormonal work-up was unremarkable. Medical decompression through intravenous dexamethasone was done and he underwent craniotomy, excision of the suprasellar mass. Gross findings showed multiple cream, tan, soft tissue fragments. Histopathology showed solid and cystic area. Solid areas had palisading epithelium surrounded by stellate reticulum and nodular areas consisting of wet keratin. Findings were consistent with adamantinomatous craniopharyngioma. See Figure 5.

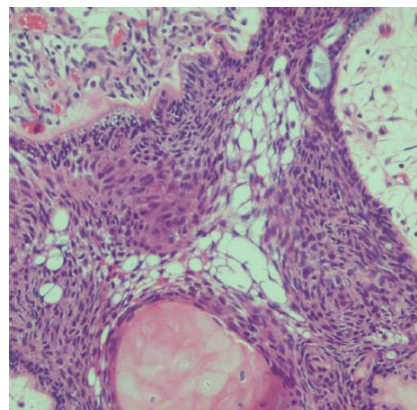


Figure 5: Histopathology. Black arrow-palisading epithelium. Yellow arrow-stellate reticulum. White arrow-wet keratin

### 3. Discussion

#### 3.1 Epidemiology and Clinical Manifestations

Craniopharyngioma is a benign epithelial intracranial tumor with an incidence rate of 0.2-2 cases per million persons per year with a bimodal age distribution; and in this case we are presented with a 37 year old male. Due to its sellar/parasellar location, clinical manifestations primarily present as hypothalamic or pituitary dysfunction in 52-87% of cases which was not seen in this case. Visual impairment in 62-84% of cases was seen similarly to this case. As a space occupying lesion, it may show signs of increased intracranial pressure usually at the time of diagnosis which was also seen in this case presenting as headache and decreasing sensorium. Symptoms in this case were present 4 months and studies show that median time of diagnosis of craniopharyngioma is 6 months.<sup>2</sup>

Craniopharyngioma in 50-78% of cases occur with psychiatric symptom such as mood symptoms (41%) and psychotic symptoms (19%). Specific psychiatric symptoms are as follows: mood (depression, mania), psychosis (delusion, hallucination), memory, personality, anorexia and anxiety. Intracranial tumors are said to cause diaschisis which affects connections to distant areas of the brain, thus, psychiatric symptoms generally have no localizing value. Anorexia symptoms was strongly associated with hypothalamic tumors whereas psychotic symptoms are probably related to pituitary tumors, memory symptoms to thalamic tumors and mood symptoms to frontal tumors.<sup>4</sup>

About 40-87% of patients with craniopharyngioma have at least one hormonal deficit. Among adults, gonadotrophin deficiency (amenorrhea or loss of libido and erectile dysfunction) appears in 40% of cases, growth hormone deficiency in 85% of cases and adrenocortical hormone deficiency and TSH deficiency in 25% of cases each.<sup>1</sup>

ACP contain a solid part consisting of nests of squamous epithelium bordered by palisading columnar epithelium ("picket-fence") which are surrounded by stellate reticulum. ACP also contain cystic parts consisting of wet keratin. PCP is characterized as well-circumscribed than ACP and contains squamous epithelium that is well-circumscribed.

There are infrequent goblet cells, ciliated epithelium and calcifications in PCP.<sup>1</sup>

### 3.2 Etiopathogenesis and Histology

Craniopharyngiomas are WHO Grade I tumors with two subtypes: adamantinomatous (ACP) and papillary (PCP). They originate from the Rathke's pouch. ACP is due to somatic mutations at CTNNB1 causing impaired degradation and accumulation of  $\beta$ -catenin, hence over activating the WNT/ $\beta$ -catenin pathway leading to cell proliferation, invasion, and development of a tumor. PCP, on the other hand is due to somatic BRAF-V600E mutations.<sup>2, 5</sup>

### 3.3 Diagnostics

Neuroimaging is advised in patients with first-onset psychiatric symptoms and anorexia without body dysmorphism.<sup>4</sup> Craniopharyngiomas are predominantly cystic (90%), calcifications (90%), contrast-enhancing on cystic walls (90%). Usual characteristic on T2 is hypo- and hyperintense.<sup>2</sup> MRI is the gold standard of imaging and CT scan may aid in visualizing the calcifications.

Hormonal work-up must be done in all patients suspected of craniopharyngioma including fasting morning cortisol, ACTH, thyroid stimulating hormone, free T4, follicle-stimulating hormone, estradiol (females), testosterone (males), growth hormone, insulin-like growth factor-I, prolactin, serum sodium, and urine specific gravity and osmolality.<sup>1</sup>

### 3.4 Management

Treatment of craniopharyngioma requires multi-disciplinary approach involving Neuro-oncology, Neurosurgery and Endocrinology. Treatment modalities are as follows: pharmacologic/hormonal therapy, complete or partial resection, radiotherapy or a combination of these. Goal of treatment is to have the least long-term morbidity. Surgical intervention depends largely on tumor size, location, invasiveness, proximity to nearby neurovascular structures, hence it must proceed with great caution to avoid neurovascular injury and hypothalamic damage. Radiation therapy may be given after partial/near-total resection or upon recurrence after total resection. Forms of radiotherapy include radiosurgery, conventional/fractionated radiotherapy, and photon beam therapy. Intracystic therapy is performed for purely cystic craniopharyngioma wherein sclerosing substances such as interferon-alpha.<sup>1,2</sup>

### 3.5 Prognosis

Craniopharyngioma is known to have high recurrence rate of ~50% but has a high survival rate (83-96% five-year survival and 65-100% 10-year survival). Prognosis depends on the size, histology, surgical intervention and extent of endocrine dysfunction. Prognosis is good among young age group than ages 65 and above. Females were also said to have worse prognosis than males.<sup>5</sup>

## 4. Conclusion

It is thought that the disruption of the neural connections between the hypothalamus and limbic system in craniopharyngiomas cause the psychiatric symptoms. The gradual onset of psychological symptoms and mass effect may be attributed to the benign nature of craniopharyngiomas. The salient features of this case

emphasize the importance of having a high index of suspicion for an organic neurological etiology in cases of new onset psychiatric symptoms without the presence of lateralizing signs.

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