

4 **Death from neglected pituitary adenoma, a case report.**  
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8 **ABSTRACT:**We present a case of a patient with macro pituitary adenomyoma complicated with  
9 hydrocephalus who presented late to the referral hospital comatose and died a few hours later. He was  
10 a 33-year-old civil servant, who gave a history of highly accelerated symptoms lasting 1 week. Autopsy  
11 findings suggested that the disease could have lasted a little bit longer than suggested.

12 **Keywords:** *Pituitary,adenoma,hydrocephalus.*

13 **INTRODUCTION**

14 Pituitary adenomas (PA) are intracranial tumours that may result in death(1). Autopsy studies suggest  
15 that microadenomas(<10mm) occur in 10 to 20% of the population (2, 3),macroadenomas >10mm are  
16 quite rare(4, 5).PA are the most common intracranial tumours producing ocular symptoms, which are  
17 either ocular or endocrine in nature(6) with others relating to mass effect as well as obstruction to  
18 cerebrospinal fluid(CSF) flow.Giant PA  $\geq 4$ cm have been reported(7-10).PA occur mostly in adults,but  
19 cases in infancy(11),sudden death due to PA in an infant (12), paediatric age range and adolescents(13,  
20 14) are reported.Accounting for 10% of intracranial neoplasms, most arise from the adenohipophysis,  
21 other sites are involved by extension, infiltration or ectopic location(15, 16).They are common tumours  
22 and they, present a challenge to the clinician in determining the clinical significance, appropriate  
23 diagnosis, need for treatment and type of therapies/ therapies(17).Pituitary adenomas may be functional  
24 or non-functional(NFA).Functional types producing single hormone, growth hormone(GH) secreting  
25 somatostatinomas,prolactin(PRL)prolactinomas,adrenocorticotrophic hormone(  
26 ACTH)corticotrophinomas, thyroid stimulating hormone(TSH) thyrotropinomas (18, 19) and other rare  
27 hormone products(20) or mixed hormone secretion(21-23).Clinical presentations may range from  
28 acromegaly or gigantism(24-26), Cushing's syndrome(27-29), Nelson syndrome, hyperprolactinaemia,  
29 and other rare endocrine syndromes(20).Most cases are sporadic, but some occur as part of multiple  
30 endocrine neoplasia1 (30-33),or other inheritable tumour syndromes(34, 35),and may occur with other  
31 tumours(36-41).

32 Mortality in the NFA adenoma is attributable to hormone deficiency due to the mass, surgery, and mass  
33 effect as they often grow very large before detection(42, 43).In one series patient died of circulatory,  
34 respiratory as well as infections(42), and in another series patients suffered occult hydrocephalus, and  
35 they died of complications following repeated interventions(44).The common presentation of NFA remain,  
36 headaches visual disturbance and the hormonal deficiencies(45).Among the functioning(FA),mortality  
37 from vascular disease is the predominant cause of death(43). In ACTH producing adenomas, control of  
38 cortisol and growth hormone secretion limits the cardiovascular risk(43).Pituitary apoplexy is a sudden  
39 vascular event in a PA that can also be life threatening(46-49), and has been reported to induce severe  
40 intracerebral haemorrhage(50, 51), and death(49, 52).

41 **CASE REPORT:**

42 We present the case of a 33-year-old man with a history of headache of seven days duration.  
43 Blurring of vision of 2 days.  
44 Seizures which were intermittent.

45 Unconsciousness of a day's duration.  
46 The headache was of sudden onset, noticed on the right temporal area and later became generalized.  
47 Described as the worse headache he has ever suffered. No known aggravating or relieving factor. Pain  
48 was said to radiate to the neck. Patient was initially ambulant but later could not undertake normal  
49 activities and could not sleep. There was no fever or associated but vomited repeatedly. The vomitus was  
50 non projectile, on billous and comprised of recently ingested feeds.  
51 He then developed tonic clonic seizure with an episode lasting 2 minutes, terminating in a cry and urinary  
52 incontinence.

53 Patient lapsed into unconsciousness on the way from the referral centre to the teaching hospital.  
54 His past medical history was not contributory.  
55 He does not take alcohol or tobacco. He was not a known hypertensive or diabetic patient and had not  
56 recently travelled to the meningitis belt of the country.  
57 Physical examination revealed a young man gasping for breath, was on oxygen at the time of reception  
58 at the referral hospital.

59 **CNS EXAMINATION:** He was unconscious with a Glasgow coma scale of 3/15 distributed as  
60 spontaneous eye movement(E1), verbal response(V1), motor response(M1).

61 Pupils were 5cm dilated and fixed, non-reactive to light direct and consensual.

62 The neck was supple Kernig's and Brudzinski signs were negative.

63 Cranial nerves 11, 111,1V, V11,1X,X were non responsive.

64 Muscle tone was reduced in all the limbs. Muscle power could not be assessed objectively.

65 There was generalized depression in limbs reflexes, both plantar and flexor.

#### 66 **RESPIRATORY SYSTEM:**

67 The respiratory rate RR: was 42 cycles per minute, was gasping. The chest was resonant and breast  
68 sounds were vesicular.

69 **CARDIOVASCULAR SYSTEM:**Pulse rate was 140 bpm,small volume .The apex beat, was at the normal  
70 position, jugular venous pressure was not raised.

71 The blood pressure progressively fell from 120/60 mmHg on admission at 9am to being unrecordable at  
72 12 noon.

73 Examination of the other organ systems did not reveal any significant find.

#### 74 **PRESUMPTIVE DIAGNOSIS:**

75 (1) Subarachnoid haemorrhage secondary to ruptured aneurism.

76 (2) Space occupying lesion (Glioma)

77 Patient was admitted into the intensive care unit under the neurology unit.

#### 78 **INVESTIGATIONS AND RESULTS.**

79 (1) Random blood sugar RBS 12.4mmol/l.

80 (2) Urinalysis was normal.

81 (3) HIV screening was Negative

82 (4) Complete blood count: Total wbc 5x 10/dl, Lymphocytes 47%,Neu.50%,eos.1%

83 Platelets; in normal range.

84 (5) Serum electrolytes: Sodium:130mm/l, Potassium.3.3 mm/l calcium, magnesium and creatinine  
85 were being processed before patients' demise.

86 (6) Skull X ray was ordered but was not done because the patient was very unstable.

87 (7) Cranial CAT scan and MRI were to be urgently ordered but were not done before patient passed.

#### 88 **TREATMENT:**

89 (1) 20 % Mannitol 250 ml over 4 hours, every 8 hrs. For 72 hrs.

90 (2) Normal saline 1 litre 8 hourly for 72 hours.

91 (3) Intravenous frusemide 20 litres after each dose of mannitol.

92 (4) Monitoring of vital signs every 30 minutes.

93 (5) Strict monitoring of fluid input and output.

94 **OUTCOME:** Patients died within two hours. Autopsy was ordered for.

#### 95 **AUTOPSY FINDINGS:**

96 Patient was generally well nourished looking, male 1.62m in height and weighing 72kg.The pupil were  
97 dilated and fixed. Other physical examinations carried out were found to be in the normal range.

98 The main autopsy findings were in the central nervous system (CNS) and the bones surrounding the sella  
99 tursica. The brain weighed 1350g, The gyri were flattened bilaterally and the sulci narrowed. There was  
100 bilateral uncal and cerebellar tonsillar grooving. A large pituitary tumour measuring 3.4 x 2 x 2 cm was  
101 found completely occluding the 3<sup>rd</sup> ventricle and projecting into the lateral ventricles. The lateral ventricles  
102 were dilated on both sides, the left measuring 12 cm long and 8 cm in the widest portion. The right  
103 measured 12 cm long and 7cm in the widest portion. The bones of the sella tursica were significantly  
104 eroded, but the tumour was still covered by the diaphragma sella and the cavernous sinus had not been  
105 eroded into.



106

107 **Figure 1:** Left cerebral hemisphere showing a pituitary adenoma and markedly enlarged lateral ventricle.

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110 A diagnosis of Pituitary adenoma causing Hydrocephalus was made.

111 The cause of death was coning due to hydrocephalus, which in turn was induced by a pituitary adenoma.

112 **DISCUSSION:**

113 Although the patient history suggested a symptom duration of 1 week, the findings at autopsy suggested  
114 otherwise. It is very likely that the pituitary adenoma had been for a while inducing symptoms which were  
115 not heeded to until it became too late. Adenomas that grow very large tend to be the non-functional  
116 types(6, 42, 53) in most cases, although one series in the United States reported 5 giant prolactinomas in  
117 their series(7).Patients with the functional adenomas normally seek medical attention quicker because of  
118 the endocrine symptoms they induce (17).Our index patient had no hormonal assay or  
119 immunohistochemistry to determine if the PA was functional or not. There was however raised random  
120 blood sugar which may suggest hypercortisolism, or even raised thyroid hormones. It is also important to  
121 state that the types of resources needed to save this patient are currently not in our centre. The lessons  
122 to learn in this case is the need for early hospital consultation when the ordinary cases such as persistent  
123 headaches are encountered. In our country the alternative medical practitioners often act as a barrier  
124 between patients and orthodox medical consultations, often with disastrous consequences.

125 **CONCLUSION:**This case highlights the common practise of patients arriving late for hospital  
126 treatment.Although the patient would have still had a tough time accessing the kind of care that will bring  
127 relief in this case.Government should intervene to change the circumstances that will improve patients  
128 welfare

129 **CONSENT:** Patients personal data was anonymised and permission for the use of specimen  
130 photographs was granted by the Department of Pathology, University of Calabar Teaching hospital.  
131

132 **ETHICAL APPROVAL:** Was granted by the institutional review board  
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UNDER PEER REVIEW