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Hypopituitarism and associated demographic and clinical factors in patients with pituitary adenoma in Ghana: a hospital-based retrospective study

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Abstract

Background: Pituitary adenoma is considered one of the main causes of hypopituitarism, which is characterised by a deficiency of one or more pituitary hormones. Clinical features of hypopituitarism may be non-specific, leading to diagnostic delays and an increased risk of morbidity and mortality in affected patients.

Objective: This study aimed to determine the prevalence and associated factors of hypopituitarism among pituitary adenoma patients attending the Korle Bu Teaching Hospital, a tertiary hospital in southern Ghana.

Methods: In this retrospective study, the clinical records of patients diagnosed with pituitary adenoma were reviewed to obtain demographic and clinical data. Pretreatment hypopituitarism, the main outcome measure, was defined as a deficiency of one or more anterior pituitary hormones prior to any therapeutic intervention. Data was analysed using Stata 16.1, and the level of significance was set at $p < 0.05$.

Results: The prevalence of pretreatment hypopituitarism was 54.51% (95% CI, 47.92 - 60.35) and was associated with older age (Odds Ratio (OR) 2.79; 95% CI 1.41 - 5.50), male sex (OR 2.51; 95%CI 1.47 - 4.28), referral from a primary care facility (OR 4.50; 95% CI 1.39 - 14.47), non-functional pituitary adenoma (OR 4.87; 95% CI 2.78 - 8.56), macroadenoma (OR 9.75; 95%CI 4.15-22.9) and visual field defects (OR 6.35; 95% CI 3.19 - 12.62. Non-functional pituitary adenoma (Adjusted Odds Ratio (aOR) 3.03; 95% CI 1.35 - 6.76) and macroadenomas (aOR 9.62; 95% CI 2.76 - 33.39) were determinants of pretreatment hypopituitarism in patients with pituitary adenoma.

Conclusion: The prevalence of pretreatment hypopituitarism was found to be 54.51% among pituitary adenoma patients attending the Korle Bu Teaching Hospital, a tertiary hospital in southern Ghana, from 1997 to 2019. Non-functional pituitary adenoma and macroadenoma were determinants of pretreatment hypopituitarism. Increasing awareness of the associated factors of hypopituitarism among clinicians could facilitate an early diagnosis and appropriate management to avoid the attendant adverse outcomes in patients with pituitary adenoma.

Keywords: Hypopituitarism, pituitary adenoma, Ghana

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INTRODUCTION

Hypopituitarism is defined as the partial or complete failure of secretion of pituitary hormones and presents as a deficiency in one or more pituitary hormones. It is caused by pituitary adenomas (PA) and its associated radiotherapy and surgical treatment [1]. Pituitary tumours are the third most common brain tumour, accounting for 10% - 15% of all primary brain tumours [2]. In pooled autopsy and radiological series, the frequency of occurrence of PA has been reported to be 14.4% and 22.5%, respectively [3]. Of the limited data on pituitary adenoma available in sub-Saharan Africa, hospital-based studies have found 16.8% - 21% of intracranial tumours to be pituitary adenomas [4,5]. The prevalence of hypopituitarism in patients with nonfunctioning PA

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(NFPA) and macroadenomas has been reported to be between 54% to 71% and 34 to 89%, respectively [6-8].

The clinical features of hypopituitarism are variable and range from subclinical disease to overt panhypopituitarism [9]. The manifestation depends on the extent and severity of hormone deficiencies, the duration of the disease and the age of onset [10]. The onset of hypopituitarism in patients with pituitary adenoma is often insidious and typically develops sequentially. It starts with a deficiency of growth hormone, followed by deficiencies in gonadotrophins, thyroid-stimulating hormone (TSH), and adrenocorticotropic hormone (ACTH), with prolactin deficiency being rare [1]. Recognised mechanisms of hypopituitarism include the direct pressure or damage to the normal pituitary tissue surrounding the tumour, mechanical compression of the portal veins, raised intrasellar pressure, and focal necrosis due to prolonged portal vein interruption [1].

Hypopituitarism is associated with increased morbidity and mortality [1]. The morbidity associated with hypopituitarism may be due to the underlying disease, inadequate long-term hormone replacement therapy, or hypopituitarism-related co-morbidities [9]. Hypopituitarism is associated with an increased risk of developing diabetes, metabolic syndrome, sepsis, fractures, cardiovascular diseases including myocardial infarction [11,12], reduced quality of life (QoL) and a depressed mood [13-15]. Indeed, despite the normalisation of hormonal disturbances, patients with hypopituitarism may continue to experience a reduction in their psychological well-being [16]. Individuals with hypopituitarism have a higher health-related expenditure, take more sick leave days and are more likely to claim a disability pension compared to the general population [17]. In patients with acromegaly, hypopituitarism has been shown to predict worse surgical outcomes with age, sex, body mass index, tumour size, invasiveness, prolactin staining, and growth hormone levels significantly correlated with preoperative hypopituitarism [18].

Up to a two-fold increase in mortality has been documented among patients with hypopituitarism compared with age- and sex-matched cohorts [9]. The slow but progressive loss of pituitary function coupled with the mild and non-specific symptoms experienced by a significant proportion of patients results in many remaining undiagnosed for prolonged periods. This increases the likelihood of morbidity and mortality in this population [10]. This study aimed to determine the prevalence of pretreatment hypopituitarism (with a focus on anterior pituitary hormone deficiencies) and the associated factors in patients with PA at a tertiary hospital in southern Ghana. The findings are expected to highlight the burden of hypopituitarism among this population and to facilitate early diagnosis for appropriate management to forestall the associated adverse outcomes.

MATERIALS AND METHODS

This was a retrospective study conducted at the endocrinology unit of the Korle Bu Teaching Hospital (KBTH), which involved reviewing the records of pituitary adenoma patients in the database from 1997 to 2019. The data of only those who had baseline results of both anterior pituitary hormones (with the peripheral hormones from their target organs) and radiological imaging (magnetic resonance imaging or computed tomography scan) were included in the study.

The records of all those with incomplete baseline investigation results (hormonal and/ or radiological) were excluded from the study. Baseline hormonal results of follicle-stimulating hormone (FSH), Luteinising hormone (LH), testosterone (males), oestrogen (females), thyroid-stimulating hormone (TSH), free thyroxine (FT4), free triiodothyronine (FT3), adrenocorticotropic hormone (ACTH), 9 am cortisol, and prolactin were included. Results of insulin-like growth factor-1(IGF-1) used to assess growth hormone deficiency were not considered as a baseline investigation since the assay for IGF-1 was previously not readily available in our setting.

The relevant demographic and clinical data of eligible individuals were documented on a data extraction form. This included the age, gender, ethnicity, baseline anterior pituitary hormone assay results, reports of radiological imaging and visual fields test, and final diagnosis. Presenting blood pressure (BP), weight, height, and body mass index (BMI) were documented. Normal weight was defined as a BMI of 18.5 - 24.9 kg/m^2, with overweight and obesity defined by BMI between the ranges 25 - 29.9 and greater than 30 kg/m^2, respectively [19]. Blood pressure was categorised into low (< 90/60 mmHg), normal (90 - 129/60-80 mmHg), prehypertension (130 - 139/80 - 89 mmHg) and hypertension (> 140/90 mmHg) [20]. Patients with a deficiency in one or more anterior pituitary hormones prior to any therapeutic interventions were classified as having pretreatment hypopituitarism. Deficiency of specific anterior pituitary hormones was also identified.

Data analysis

Data was analysed using Stata16.1. Categorical variables have been presented as percentages. Categorical (demographic and clinical) variables of PA patients with and without hypopituitarism were compared using a Chi-squared test. Clinical and demographic factors associated with hypopituitarism were determined using univariate logistic regression, and predictors were identified after multivariate logistic regression. The level of significance was set at a p-value < 0.05.

RESULTS

Out of a total of 309 pituitary adenoma patients in the clinic's database, data from 244 eligible patients were

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analysed. The records of 65 patients were excluded because the results of incomplete baseline (hormonal and/or radiological) investigations. Analysis of the records of the 244 eligible patients found the age range to be 13 years to 76 years, with the majority being 40 years or older (131, 53.7%). One hundred and forty-five (59.4%) were female. Overall, 54.51% (95% CI, 45.64 - 64.6) of individuals with pituitary adenoma had pretreatment hypopituitarism. The proportion of individuals with hypogonadotropic hypogonadism (HH), secondary hypothyroidism (SH), and secondary adrenocortical insufficiency (SAI) were 44.26% (95% CI, 36.11 - 53.44), 38.11% (95% CI, 30.76 - 46.69) and 27.87% (95% CI, 21.64 - 35.33) respectively. Individuals with no anterior pituitary hormone deficiency were 45.49% (95% CI, 37.42 - 54.78). The proportion of individuals who were deficient in all three hormones was 17.21% (Figure 1).

Pretreatment hypopituitarism was associated with age, sex, source of referral, systolic BP, size of PA, functional status, and visual field (VF) defects ($p < 0.05$) Table 1. Univariate analysis revealed that male sex (OR 2.51 [1.47 - 4.28] 0.0007), age greater than 40 years (OR 2.79 [1.41 - 5.50] 0.003), referral from primary care facility (OR 4.50 [1.39 - 14.47] 0.012), macroadenoma (OR 9.75 [4.15 - 22.93] < 0.0001), NFPA (OR 4.87 [2.78 - 8.56] < 0.0001), and abnormal VF test (OR 6.35 [3.19 - 12.62] < 0.0001) were associated with higher odds of pretreatment

Table 1: Demographic and clinical factors associated with pre-treatment hypopituitarism

Variable	Pre-treatment hypopituitarism				
	Yes N(%)	No N(%)	Chi (p-value)	OR [95%CI]p-value	Adjusted OR [95%CI]p-value
Age			9.13 (0.01)		
≤29	18(36.7)	31(63.3)		Ref	Ref
30-39	34(53.1)	30(46.9)		1.95[0.91-4.17]0.09	1.27[0.42-3.89]0.671
40+	81(61.8)	50(38.2)		2.79[1.41-5.50]0.003	1.54[0.54-4.34]0.417
Sex			11.65(0.001)		
Female	66(45.5)	79(54.5)		Ref	Ref
Male	67(67.7)	32(32.3)		2.51[1.47-4.28]0.0007	1.50[0.66-3.39]0.329
Ethnicity			0.68(0.879)		
Northern Ghana	11(47.8)	12(52.2)		Ref	
Akan	74(56.2)	56(43.8)		1.44[0.59-3.51]0.42	
Ga-Adangbe	22(53.7)	19(46.3)		1.26[0.45-3.51]0.65	
Ewe	26(56)	24(44.0)		1.18[0.44-3.18]0.74	
Source of Referral (229)			8.93(0.011)		
Tertiary	4(25.0)	12(75.0)		Ref	Ref
Secondary	10(43.5)	13(56.5)		2.31[0.57-9.35]0.24	3.15[0.42-23.28]0.262
Primary	114(60.0)	76(40.0)		4.50[1.39-14.47]0.012	2.97[0.51-17.24]0.226
Size			35.29(<0.001)		
Microadenoma	7(15.2)	39(84.8)		Ref	Ref
Macroadenoma	126(63.6)	72(36.4)		9.75[4.15-22.93]<0.0001	9.62[2.76-33.39]<0.001
Functional status			32.37(<0.001)		
Functioning	55(39.7)	86(60.3)		Ref	Ref
Non-functioning	78(75.7)	25(24.3)		4.87[2.78-8.56]<0.0001	3.03[1.35-6.76]0.007
VFT (193)			31.01 (<0.001)		
normal	15(24.6)	46(75.4)		Ref	Ref
abnormal	89(67.4)	43(32.6)		6.35[3.19-12.62]<0.0001	2.30[0.87-6.08]0.06
BMI (182)			1.65 (0.648)		
Normal	25(59.5)	17(40.5)		Ref	
Underweight	1(50.0)	1(50.0)		0.68[0.04-11.6]0.79	
Overweight	27(46.6)	31(53.4)		0.59[0.26-1.32]0.20	
Obesity	42(52.5)	38(47.5)		0.75[0.35-1.60]0.46	
Systolic BP			8.97(0.03)		
Normal	85(52.8)	76(47.2)		Ref	
Low BP	9 (81.8)	2 (18.2)		4.02[0.84-19.20]0.08	
Prehypertension	2(20)	8(80)		0.22[0.05-1.08]0.06	
Hypertension	37(59.7)	25(40.3)		1.32[0.73-2.4]0.36	
Diastolic BP			6.90(0.075)		
Normal	91(58.7)	64(41.3)		Ref	Ref
Low BP	1(50.0)	1(50.0)		0.70[0.04-11.45]0.81	
Prehypertension	4(25)	12(75.0)		0.23[0.07-0.76]0.016	0.10[0.01-0.79]0.029
Hypertension	37(50.7)	34(49.3)		0.77[0.44-1.35]0.35	0.52[0.20-1.31]0.165

VFT=visual field test; BMI=body mass index; BP= blood pressure; Ref= Reference category used for inferences

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Table 2. Demographic and clinical factors associated with secondary hypothyroidism

Variable		Secondary hypothyroidism (SH)	
	N(%)	P-value*	OR [95%CI] p value
Age		0.013	
≤29	10(21.7)		Ref
30-39	25(41.0)		2.50[1.06-5.89]0.036
40+	58(46.0)		3.09[1.43-6.73]0.004
Sex		0.006	
Female	45(31.0)		Ref
Male	48(46.5)		2.09[1.23-3.55]0.006
Size		<0.001	
Microadenoma	2(4.3)		Ref
Macroadenoma	91(46)		18.71[4.41-79.3]0.0001
Functional status		<0.001	
Functional	34(24.1)		Ref
Non-functional	59(57.3)		4.22[2.44-7.31]<0.0001
VFT		<0.0001	
normal	7(11.5)		Ref
abnormal	66(50.0)		7.71(3.27-18.20)<0.0001
BMI		0.423	
Normal	18(42.9)		Ref
Underweight	0(0.00)		-
Overweight	18(31.0)		0.60[0.26-1.37]0.23
Obesity	26(32.5)		0.64[0.30-1.39]0.26
Systolic BP		0.025	
Normal	55(34.2)		Ref
Low BP	8(72.7)		5.13[1.31-20.15]0.02
Prehypertension	2(20.0)		0.48[0.1-2.34]0.37
Hypertension	28(45.2)		1.58[0.87-2.88]0.13
Diastolic BP		0.421	
Normal	61(39.4)		Ref
Low BP	1(50.0)		1.54[0.09-25.1]0.76
Prehypertension	3(18.8)		0.36[0.1-1.3]0.12
Hypertension	28(39.4)		1.00[0.56-1.78]0.99

*P-value from Chi-squared test; VFT=visual field test; BMI=body mass index; BP= blood pressure; Ref= Reference category used for inferences

Table 3. Demographic and clinical factors associated with secondary adrenocortical insufficiency

Variable		Secondary adrenocortical insufficiency (SAI)	
	N(%)	P-value*	OR [95%CI] p-value
Age		0.13	
≤29	8(16.3)		Ref
30-39	20(31.3)		2.33[0.92-5.86]0.07
40+	40(30.5)		2.25[0.97-5.24]0.06
Sex		0.06	
Female	34(23.4)		Ref
Male	34(34.3)		1.71[0.97-3.0]0.06
Size		<0.001	
Microadenoma	3(6.5)		Ref
Macroadenoma	65(32.8)		7.01[2.09-23.4]0.002
Functional status		<0.001	
Functional	20(14.2)		Ref
Non-functional	48(46.6)		5.28[2.86-9.73]<0.0001
VFT		<0.001	
normal	4(6.6)		Ref
abnormal	46(34.8)		7.62(2.60-22.34)0.0002
BMI		0.014	
Normal	17(40.0)		Ref
Underweight	1(50.0)		1.47[0.09-25.16]0.79
Overweight	17(29.3)		0.61[0.26-1.41]0.25
Obesity	12(15.0)		0.26[0.11-0.61]0.003
Systolic BP		0.115	
Normal	46(28.6)		Ref
Low BP	6(54.5)		3.00[0.87-10.32]0.08
Prehypertension	1(10.0)		0.27[0.03-2.25]0.23
Hypertension	15(24.2)		0.80[0.41-1.57]0.51
Diastolic BP		0.421	
Normal	48(31.0)		Ref
Low BP	1(50.0)		2.23[0.14-36.39]0.57
Prehypertension	3(18.8)		0.51[0.14-1.87]0.32
Hypertension	16(22.5)		0.65[0.34-1.24]0.19

*P-value from Chi-squared test; VFT=visual field test; BMI=body mass index; BP= blood pressure Ref= Reference category used for inferences

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hypopituitarism. Those with diastolic BP in the prehypertension range (OR 0.23 [0.07 - 0.76] 0.016) were less likely to have pretreatment hypopituitarism (Table 1). Abnormal VF tests, macroadenoma and NFPA were associated with a higher likelihood of SH (Table 2), SAI (Table 3) and HH ((Table 4) (p < 0.001). Males (OR 2.09 [1.23 - 3.55] 0.006) delete older than 40 years (OR 3.09 [1.43 - 6.73] 0.004), and those with a low systolic BP (OR 5.13 [1.31 - 20.15] 0.02) had a higher probability of having SH (Table 2). The diagnosis of SAI was less probable in individuals with BMI within the obesity range (OR 0.26 [0.11 - 0.61] 0.003) (Table 3). HH was more likely in individuals aged 40 years or older (OR 2.03[1.02 - 4.04] 0.04) but less likely in those with diastolic BP within the prehypertension range (OR 0.16 [0.03 - 0.71] 0.02) (Table 4). Multivariate analysis identified NFPA (aOR 3.03 [1.35 - 6.76] 0.007) and macroadenoma (aOR 9.62 [2.76 - 33.39] < 0.001) as the only two factors that predicted the presence of pretreatment hypopituitarism. On the other hand, diastolic BP in the prehypertension range (aOR 0.10 [0.01 - 0.79] 0.029) was independently associated with a lower likelihood of having pretreatment hypopituitarism (Table 1).

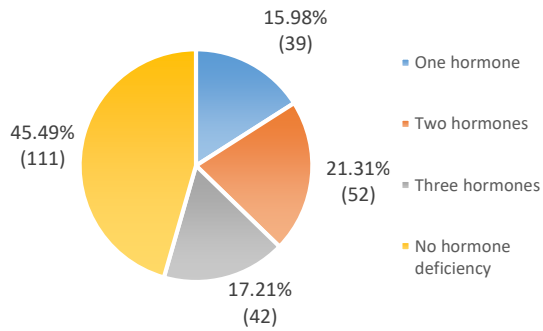

Figure 1. Proportions of deficient anterior pituitary hormones

DISCUSSION

The results from the current study show that the majority (54.51%) of patients with PA have pretreatment hypopituitarism, which is consistent with findings from previous studies [21]. Hypogonadotropic hypogonadism (HH) was the most common anterior pituitary hormone deficiency, followed by SH and then SAI. This pattern of hormone deficiency is similar to that from other studies and

Table 4. Demographic and clinical factors associated with hypogonadotropic hypogonadism

Variable		Hypogonadotropic hypogonadism (HH)	
	N(%)	p - value*	OR [95% CI] p-value
Age		0.116	
≤29	16(32.6)		Ref
30-39	27(42.2)		1.51[0.69-3.27]0.3
40+	65(49.6)		2.03[1.02-4.04]0.04
Sex		0.105	
Female	58(40)		Ref
Male	50(50.5)		1.53 [0.91-2.56] 0.11
Size		<0.001	
Microadenoma	7(15.2)		Ref
Macroadenoma	101(51.0)		5.80 [2.47-13.59] 0.0001
Functional status		<0.001	
Functional	43(30.5)		Ref
Non-functional	65(63.1)		3.90 [2.28-6.67] < 0.0001
VFT		<0.001	
normal	11(18.0)		Ref
abnormal	76(57.6)		6.17 (2.95-12.91) < 0.0001
BMI		0.768	
Normal	21(50.0)		Ref
Underweight	1(50.0)		-
Overweight	23(39.7)		0.65 [0.29-1.46] 0.3
Obesity	34(42.5)		0.74 [0.35-1.56] 0.43
Systolic BP			
Normal	71(44.1)		Ref
Low BP	8(72.7)		3.38 [0.86-13.2] 0.08
Prehypertension	1(10.0)		0.14 [0.02-1.14] 0.07
Hypertension	28(45.2)		1.04 [0.58-1.88] 0.89
Diastolic BP		0.062	
Normal	74(47.7)		Ref
Low BP	1(50.0)		1.09 [0.07-17.81]0.95
Prehypertension	2(12.5)		0.16 [0.03-0.71]0.02
Hypertension	31(43.7)		0.85 [0.48-1.49]0.57

*P-value from chi-squared test; VFT=visual field test; BMI=body mass index; BP= blood pressure Ref= Reference category used for inferences

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reflects the classical sequential order of development of hypopituitarism associated with PA [1,22-24]. The proportion of patients with deficiencies of one, two or three anterior pituitary hormones is comparable to that previously documented [25]. The proportion of individuals with a deficiency of only one anterior pituitary hormone was less than that of individuals with a deficiency of more than one hormone, and this finding is corroborated by other studies [26].

In the current study, male sex and age greater than 40 years were each associated with a 2-fold increase in the odds of having hypopituitarism. The diagnosis of PA and the attendant hypopituitarism may be delayed especially in older adults, since they are slow-growing tumours which often present with non-specific symptoms [27]. Indeed, the incidence of PA tends to increase with age, with older adults generally reporting late and being more prone to hypopituitarism [27,28]. The increased likelihood of hypopituitarism among males may be due to delayed hospital presentation, which reflects the extensively reported low healthcare utilisation in men [29,30]. In addition to older age and male sex, other factors such as pituitary stalk deviation, contrast enhancement, and optic chiasma compression have also been associated with hypopituitarism [25].

The majority of patients in our setting have been found to present in advanced stages of PA with large tumours and often with hypopituitarism and compression of surrounding structures [31]. Compression of the optic chiasm by an enlarging adenoma causes visual impairment, the most frequently encountered neuro-ophthalmological symptom in patients with PA [32]. In a hospital-based study in Ghana, PA was the most common brain tumour (62.9%) associated with visual impairment [33]. Generally, bitemporal hemianopia tends to be the commonly diagnosed VF defect [34]. The majority of the participants with VF defects in this study had hypopituitarism, and the odds of hypopituitarism in such individuals were high. Therefore, it is imperative that patients with tumours abutting the optic chiasm undergo visual field perimetry to assess for VF defects [31,34].

Hypopituitarism among individuals with macroadenoma is reported to range between 37% to 85%, and the proportion found in this study falls within this range [22]. Also, in this present study, individuals with macroadenomas were 9 times more likely to have hypopituitarism compared with those with microadenoma. Additionally, macroadenomas predicted the presence of hypopituitarism. The pituitary dysfunction associated with macroadenoma can be caused by the involvement of the normal gland or pituitary stalk compression, with the risk of hypopituitarism being directly related to tumour volume [35,36]. Large adenomas, suprasellar extension and stalk compression on MRI have been shown to be the best predictors of hypopituitarism, and different tumour diameter cut-offs have been proposed as predictors of specific pituitary hormone deficiencies [25, 37]. Microadenomas larger than 5mm may cause hypopituitarism, with the prevalence of hypopituitarism in patients with nonfunctioning microadenoma reported to be 33% [38]. For this reason, routine testing for hypopituitarism is recommended in patients with macroadenoma and those with large microadenomas [35,37]. The inability of NFPA to secrete biologically active hormones often results in patients being asymptomatic, leading to diagnostic delays [39]. At diagnosis, most are macroadenomas and present with hypopituitarism and features of mass effect on surrounding tissues [23,39].

Generally, the frequency of hypopituitarism among NFPA increases with increasing size of the tumour [25,37]. The reported prevalence of hypopituitarism in patients with NFPA ranges between 41 - 89% and this is consistent with the finding in this current study [23,40]. Obesity has been found to be prevalent in adults with hypopituitarism, especially among male patients and those who have hypothalamic dysfunction, TSH and GH deficiency [41]. In this study, participants who had a BMI in the obesity range were less likely to have SAI. This finding is expected since anorexia and weight loss are well-established manifestations of chronic adrenocortical insufficiency [42]. Thus, in PA patients with obesity, deficiency in anterior pituitary hormones other than adrenocortical insufficiency should be actively investigated.

Individuals with hypopituitarism have an increase in morbidity and mortality compared to the general population [11,12]. Hypopituitarism has a negative influence on multiple aspects of QoL of patients with PA [13–15,43], particularly among those with hypopituitarism co-existing with acromegaly or Cushing syndrome [44]. Factors associated with high mortality rates include female sex, young age, high BMI, uncontrolled diabetes, hypogonadism, GH excess, and a long duration between symptom onset and diagnosis [45,46]. Cardiovascular diseases are the most common cause of death [45,47] due to the high prevalence of cardiovascular risk factors among patients with hypopituitarism [47, 48]. An early diagnosis and replacement of deficient hormones in patients with PA, before and after pituitary surgery, are essential to avoid the life-threatening complications and death associated with hypopituitarism [50]. Hormone replacement may be lifelong, and often, patients are inclined to resist long-term therapy because of the publicity about the possible adverse effects of medications [26]. To overcome this challenge, it is crucial that patients are adequately educated.

The findings from this hospital-based study are not generalisable to the general population. Being retrospective study data, some variables were not available due to incomplete documentation. To the best of our knowledge, however, this is the first study that has focused on the burden of hypopituitarism among patients with PA in Ghana and, indeed, in the subregion. It can serve as a baseline for further study.

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Conclusion

The majority of patients with PA attending the KBTH, a tertiary facility in southern Ghana, had pretreatment hypopituitarism with the associated factors being male sex, older age, referral from primary care, macroadenoma, nonfunctioning PA, VF defects and low systolic BP. Nonfunctioning PA and macroadenomas were determinants of hypopituitarism, whereas individuals with diastolic BP in prehypertension range were less likely to have hypopituitarism. Hypopituitarism is associated with significant morbidity and mortality; hence, an increase in the awareness of its burden and associated factors among clinicians is essential. This is expected to facilitate early diagnosis, appropriate management, and adequate patient education to improve outcomes in patients with PA.

DECLARATIONS

Ethical consideration

Ethical approval was obtained from the Ethical and Protocol Review Committee of the College of Health Sciences, University of Ghana (Reference number – CHS/EPRC/MAR; protocol identification number CHS-Et/M.7-P1.5/2017-2018)

Consent to publish

All authors agreed on the content of the final paper.

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Competing Interest

The authors declare that there is no conflict of interest regarding the publication of this article.

Author contributions

JA, YA, and EY conceptualised and designed the study. JT, JA, and MR analysed the data, JA drafted the initial manuscript, all authors made significant intellectual contributions, and read and approved the final manuscript.

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Availability of data

Data is available upon request to the corresponding author.

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