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The Forgetten Axis-Hidden Neuroendocrine Sequelae Of Aneurysmal Subarachnoid Hemorrhage

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ABSTRACT

Background and Aim: In the treatment of Aneurysmal Subarachnoid Hemorrhage (aSAH), endocrine dysfunction is rarely given adequate attention. Nevertheless, neuroendocrine disturbances, particularly involving the pituitary gland, occur frequently after aSAH and can cause lasting issues such as impaired cognition and diminished quality of life. While hypopituitarism following SAH may present with vague or subtle signs, missing the diagnosis can lead to serious health risks.

Methods and Materials/Patients: This prospective study aimed to investigate the frequency and characteristics of neuroendocrine changes in patients with acute aneurysmal subarachnoid hemorrhage (aSAH). It included 35 patients who presented within seven days of symptom onset. Comprehensive endocrine evaluations were conducted to assess potential dysfunction across various pituitary axes, including the somatotropic, gonadotropic, corticotropic, thyrotropic axes, as well as prolactin levels.

Results: A total of 35-SAH cases (10 males and 25 females; mean age was 56.24 years) were included in the study. Aneurysms were more commonly found in the anterior circulation (n=30) than in the posterior circulation (n=5). Most of the patients presented with the Hunt-Hess grade of 1, followed by grades 3, 2, and 4, respectively. Growth hormone deficiency (48%) was the most common pituitary dysfunction, followed by adrenocorticotrophic hormone (24%), gonadotropins (FSH & LH) (24%), and thyroid stimulating hormone (16%) deficiencies and prolactin deficiency (10%) respectively. Single pituitary axis neuroendocrine dysfunction was noted in 14 patients (40%) and multiple pituitary axes dysfunction was observed in 11 patients (31.5%). Overall, 25 patients (71.5%) had neuroendocrine dysfunction in single or multiple pituitary hormone axes.

Conclusion: Neuroendocrine dysfunction in acute aSAH is 71.5%. Accordingly, 40% of the participants had single-axis pituitary dysfunction and 31.5% had multiple axes pituitary dysfunction. The most common endocrine dysfunction was growth hormone deficiency (48%), followed by adrenocorticotrophic hormone, gonadotropins (LH & FSH), and thyroid stimulating hormone. Therefore, it is suggested to include hormonal evaluation in the management of acute SAH for better clinical outcomes.

HIGHLIGHTS: There is an increased incidence of neuroendocrine dysfunction following aneurysmal SAH (aSAH), mostly during the acute phase. Multiple pituitary hormone deficiencies and single-axis pituitary dysfunction were noted in 36% and 32% of patients, respectively. The most common pituitary hormone deficiency following aSAH was growth hormone (48%), followed by adreno-corticotrophic hormone, gonadotropins (LH/FSH), and thyroid stimulating hormone and prolactin deficiency. In the clinical management of aSAH, clinical outcome benefits from the evaluation of hormonal status.

PLAIN LANGUAGE SUMMARY

This study aims to investigate the pattern of hormone dysfunction in the case of subarachnoid haemorrhage (SAH) following the rupture of aneurysms. The pituitary hormones, including growth hormone, gonadotropins (FSH/LH), adrenocorticotrophic hormone, thyroid stimulating hormone and prolactin were studied for possible dysfunction. A total of 35 patients, comprising 10-males and 25 females with a mean age of 56.24 years, were included in this study. Most cases presented with the Hunt-Hess grade 1, followed by grade III (20%). The most common neuroendocrine deficiency was the growth hormone in 48% of the patients, followed by the adrenocorticotrophic hormone (24%), the gonadotropins (FSH/LH) (24%), prolactin deficiency and thyroid stimulating hormone (16%) deficiency, respectively. Single pituitary hormone deficiency was observed in 14 cases (68%). Endocrine dysfunction is an important complication of SAH. This study concludes that pituitary hormone dysfunction is highly prevalent in aneurysmal SAH. This leads to residual symptoms, such as decreased cognition and quality of life. Although hypopituitarism following SAH presents with subtle nonspecific symptoms, it can lead to serious consequences if left undiagnosed. Therefore, it is suggested to include hormonal evaluation in the management of aneurysmal SAH for better clinical outcomes.

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INTRODUCTION

Subarachnoid haemorrhage (SAH) is a life-threatening condition caused by bleeding into the space between the arachnoid and pia mater. While the majority of SAH cases are traumatic in origin, aneurysmal subarachnoid haemorrhage (aSAH) represents a smaller but more serious subset. Aneurysmal rupture is typically suspected in cases of spontaneous SAH, and aSAH is considered the most concerning form of the condition. Globally, aSAH carries a significant health burden due to its high fatality rate and the risk of long-term disability. The incidence of aSAH ranges from 6 to 10 cases per 100,000 individuals annually, with most patients falling within the 40 to 60-year age range. Non-traumatic SAH is most commonly caused by ruptured cerebral aneurysms, accounting for approximately 75% to 85% of such cases. Mortality following aSAH reaches about 50% within six months. Because aSAH often affects individuals during their most productive years, it has profound social, functional, and economic consequences. The period within six months post-aSAH is referred to as the acute phase, while the period beyond six months is termed the chronic phase.

Endocrine dysfunction is a relatively rare and often overlooked aspect of aSAH management. Only a limited number of studies—mostly small case series and individual reports—have examined the relationship between aSAH and neuroendocrine dysfunction. The few systematic investigations available have produced inconsistent findings. Notably, most of these studies have been conducted at least one year after the initial haemorrhage, with very few focusing on the acute phase. This study seeks to explore the incidence, patterns, and extent of neuroendocrine changes occurring during the acute phase of aSAH.

METHODS AND MATERIALS/PATIENTS

We aimed to evaluate the pattern and incidence of neuroendocrine dysfunction in cases of acute aSAH. This prospective observational hospital-based study was conducted in the Department of Neurosurgery. Gauhati Medical College, India. All-adult patients (age >18 years) admitted with aSAH in the Department of Neurosurgery, Gauhati Medical College, Gauhati, India from June-2024 to May 2025 were included in this study. Patients with ictus of SAH for more than 7 days, on drugs affecting hypothalamic-pituitary function, or with a pre-existing pituitary disorder were excluded from this study, The patients were included after giving an informed consent letter. The data on age, sex, clinical severity of SAH at the time of admission (by the Hunt-Hess grading system), location of the aneurysm, and the modality of treatment (embolization or surgery) were noted. A Computed Tomography (CT) scan and Digital Subtraction Angiography (DSA) of the cerebral circulation were used for radiological evaluation. On the day after admission, the serum hormone level assessment was done during morning hours (8:00 to 9:00) and the following hormones were measured: thyroid stimulating hormone (TSH), free thyroxine, cortisol, growth hormone (GH), prolactin, insulin-like growth factor 1 (IGF-1), follicle-stimulating hormone (FSH), luteinizing hormone (LH), and testosterone (males)/ estrogen(females). The endocrine evaluation was done on the next day after admission in the morning between 8:00 and 9:00 and it included the measurement of the following hormones: TSH, free thyroxine, cortisol, GH, prolactin, IGF-1, FSH, LH, and testosterone (males)/estrogen (females). The statistical analysis was done using the SPSS software version 20 and the Chi-square test was also utilized in the analysis.

The posterior pituitary deficiency was not evaluated. The endocrine assessment was performed through laboratory tests using commercially available diagnostic kits, which included the following items:-

Serum thyroxine (normal range: 4.8-12.7 µg/dL);
Serum TSH (normal range: 0.5-5 µU/mL);
Cortisol (0800 h: 171-536 nmol/L);
IGF-1 (age- and gender-specific normative data were used);
Prolactin (men: 4.0-15.2 ng/mL and women: 4.79-23.3 ng/mL);
LH (men: 1.7-8.6 mIU/ml, and women: 2.4-12.6 mlU/mL);
FSH (men: 1.5-12.4mlU/ml and women: 3.5-12.5 mIU/mL, postmenopausal >30 mIU/L)
Testosterone (9.9-27.8 nmol/L);
Estradiol (12.5-166 pg/mL) (18).

Meanwhile, the definitions of endocrine abnormalities were done using certain criteria:

Secondary hypothyroidism was defined as T4 level <4.8 µg/dL with low/normal TSH level;
Central hypogonadism was defined as serum testosterone <9 nmol/L in the presence of low or normal levels of LH (men); 17 ß estradiol levels <12 pg/mL in the presence of low or normal gonadotropins (women) and, low or inappropriately normal gonadotropins for age (FSH <30 mlU/L) in postmenopausal women.
Serum prolactin levels <5 ng/mL in either sex were considered low.

Relevance of the study

This study aims to evaluate hormonal dysfunction in patients after acute aneurysmal subarachnoid hemorrhage (aSAH). It provides insight into the incidence and patterns of neuroendocrine abnormalities following aSAH, while also examining various factors linked to the condition. Additionally, the research seeks to predict disease progression and prognosis in relation to hormonal disturbances post-SAH. Although some prior studies have addressed this topic, most have originated from Western populations. Overall, this study highlights the importance of hormonal assessment in SAH management, which could improve clinical outcomes and enhance quality of life for patients recovering from aSAH.

RESULTS

A total of 35 cases admitted with aSAH in the Department of Neurosurgery, Gauhati Medical College, India from June-2024 to May 2025-were included in this study. Cases were selected according to the predetermined inclusion and exclusion criteria. These cases were subjected to thorough clinical and endocrine assessments. The participants included 10 males and 25 females with a male/female ratio of 2:5. The overall Mean ± SD age was 56.24 ± 4.39 years.

The most common location of aneurysms was found to be in the anterior circulation (n=30) rather than the posterior circulation (n=5). The distribution of aneurysms based on the involved blood vessels in the decreasing order of frequency is as follows: Anterior communicating artery (A.COM)=10; anterior cerebral artery (ACA)=8; middle cerebral artery (MCA)=8; internal carotid artery (ICA)=4; superior cerebellar artery (SCA)=3; basilar artery (BA)=2; and vertebral artery.

Table 1. Patients' demographics

Table 1A. Mean ± SD age and gender distribution of aSAH cases

Gender	NO.(%)	Mean ± S.D.
Male	10 (28%)	54.2 ± 5.30
Female	25 (72%)	56.60 ± 4.07
Total	35	56.24 ± 4.39

Table 1B. Grades of SAH

SAH	No. (%)
1.00	13(52.0)
2.00	2(8.0)
3.00	9(36.0)
4.00	1(4.0)

The grading of SAH was done using the Hunt-Hess classification. The distribution SAH grades is as follows:
grade 1-52%, grade 3-36%, grade 2-8%, and grade 4-4%.
The endocrine evaluation showed the deficiency of certain hormones during the acute phase of aSAH. Among 35 patients, 25 cases had one or more pituitary
hormone deficiencies; that is, the incidence of pituitary dysfunction during the acute phase of aSAH in our study was 68%. Among this population, 14 cases (40%) had single pituitary axis deficiency and 11 cases (31%) had deficiencies in multiple pituitary axes. In addition, 6 cases (17%) showed ACTH deficiency. GH deficiency was noted in 12 cases (34%) and cortisol was normal in all the cases and no cortisol deficiency was observed. LH deficiency was reported in 6 cases (17%) and FSH deficiency in 6 cases (17%). Prolactin deficiency was 10%. 4 patients (11.5%) had TSH deficiency. Accordingly, GH deficiency (48%) was the most common pituitary dysfunction in our study, followed by ACTH (24%), FSH/LH (24%), and TSH (16%).

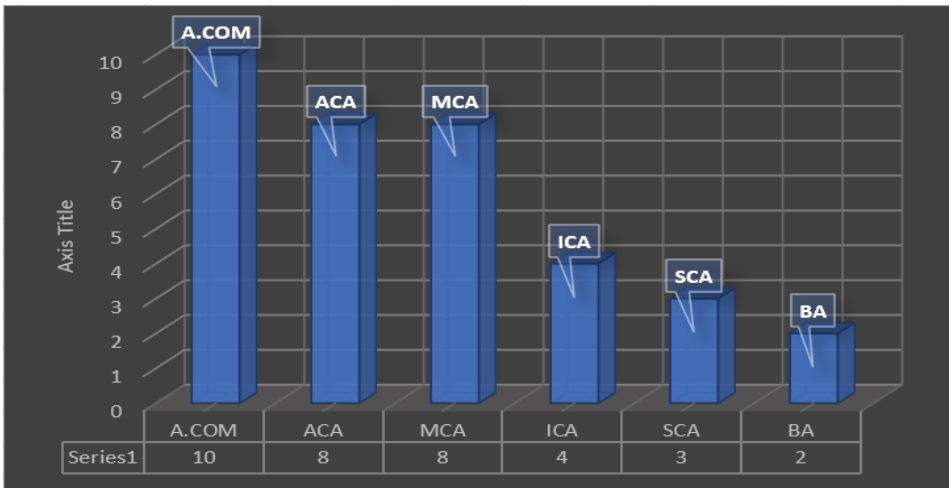

Figure 1. Distribution of aneurysms based on the involved vessels

A.COM: Anterior communicating artery; MCA: Middle cerebral artery; ICA: Internal carotid artery; SCA: Superior cerebellar artery; BA: Basilar artery; VA: Vertebral artery.

Table displays the days from the SAH admission to the investigation. The Mean ± SD period of investigation was 2.64 ± 0.86 days after admission.
According to the data, it can be inferred that the most common hormonal deficiency during the acute phase of aSAH was GH(48%), followed by ACTH (24%), LH (24%), FSH(24%), and TSH(16%). The table shows the association between the grades of SAH and hormones. There is no correlation between the grades of SAH and hormonal deficiencies. Although the hormonal deficiencies of ACTH, GH, LH, FSH, TSH and Prolactin can be observed in grade 1 and grade 3 SAH, this association is insignificant as the P>0.05.

Hormones	Hormone Status	NO. (%)
ACTH	Normal	19(76.0)
	Decreased	6(24.0)
GH	Normal	13(52.0)
	Decreased	12(48.0)
Cortisol	Normal	25(100)
	Decreased	0(0)
TSH	Normal	21(84.0)
	Decreased	4(16.0)
LH	Normal	19(76.0)
	Decreased	6(24.0)
FSH	Normal	19(76.0)
	Decreased	6(24.0)
Prolactin	Normal	25(100)
	Decreased	0(0)

Table 2. Hormonal disturbance of ACTH, GH, Cortisol, TSH, LH, FSH, and Prolactin

Figure 2 presents the association between gender and hormones among aSAH cases. Among the cases with ACTH deficiency, 33.3% were female and 66.7% were male (P = 0.14) Of all patients with GH deficiency, 58.3% were female and 41.7% were male (P = 0.59). Among the patients with gonadotropin (LH/FSH) deficiency, half were female and half were male (P = 0.45). Moreover, 75% of the patients with TSH deficiency were female and the rest (25%) were male (P=0.46). This association between gender and hormonal deficiency in aSAH is considered insignificant as the P>0.05.

SAH	No. (%)													
	ACTH		GH		Cortisol		LH		FSH		Prolatin		TSH	
	N	D	N	D	N	D	N	D	N	D	N	D	N	D
1.00	11(57.9)	2(33.3)	8(61.5)	5(41.7)	13(52)	0	10(52.6)	3(50)	10(52.6)	3(50)	13(52)	0	10(47.6)	3(75)
2.00	2(10.5)	0	0	2(16.7)	2(8)	0	2(10.5)	0	2(10.5)	0	2(8)	0	2(9.5)	0
3.00	5(26.3)	4(66.7)	4(30.8)	5(41.7)	9(36)	0	6(31.6)	3(50)	6(31.6)	3(50)	9(36)	0	8(38.1)	1(25)
4.00	1(5.3)	0	1(7.7)	0	1(4)	0	1(5.3)	0	1(5.3)	0	1(4)	0	1(4.8)	0
P	0.31		0.28		-		0.71		0.71		0		0.74	

Table 3. Association between grades of SAH and hormones

ACTH: Adrenocorticotropic hormone; GH: Growth hormone; TSH: Thyroid stimulating hormone; LH: Luteinizing hormone; FSH: Follicle stimulating hormone.

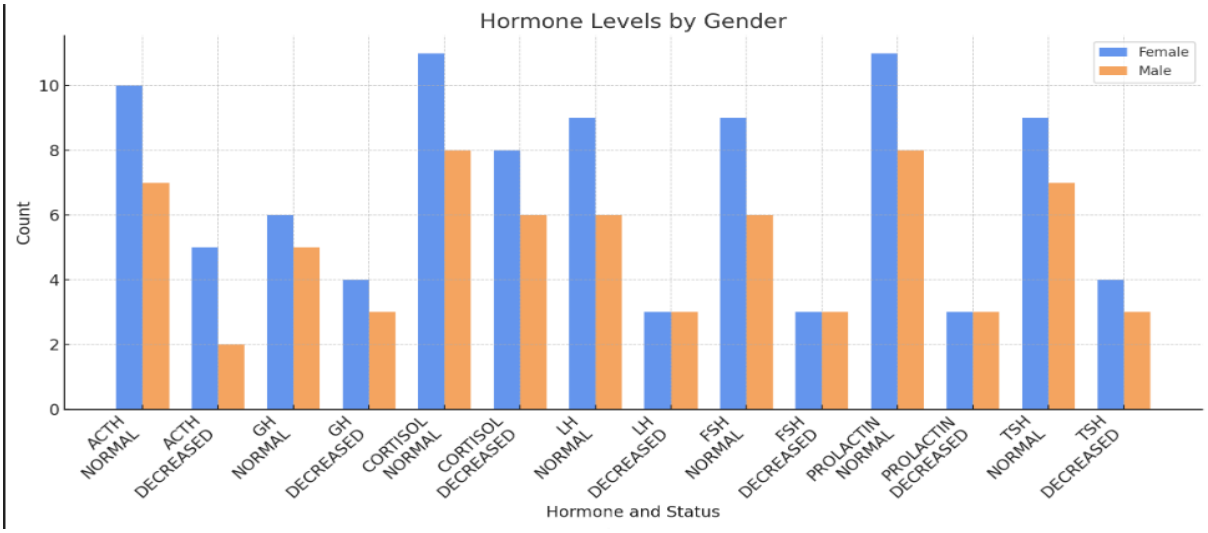

Figure 2. Association between gender and hormones among aSAH cases.

ACTH: Adrenocorticotropic hormone; GH: Growth hormone; TSH: Thyroid stimulating hormone; LH: Luteinizing hormone; FSH: Follicle stimulating hormone; aSAH: Aneurysmal subarachnoid haemorrhage.

Table 3 indicates the association between the blood vessels involved in aSAH and hormonal disturbances. ACTH deficiency was noted in 6 aSAH cases involving anterior circulation, and none involving posterior circulation. ACTH deficiency was noted in 3 A.COM, 2 MCA, 1-ICA and 2 SCA involvement (P = 0.88). GH deficiency was noted in 10 aSAH cases involving anterior circulation and 2 aSAH cases involving posterior circulation; meanwhile, GH deficiency was noted in 3 A.COM, 4 MCA, 3 ICA, 1 BA, and 1 VA involvement (P = 0.48) Gonadotropin (LH/FSH) deficiency was observed in 6 aSAH cases involving anterior circulation and none involving posterior circulation. Additionally, gonadotropin (LH/FSH) deficiency was seen in 4 A.COM, 2 MCA, 1 ICA, and 1 SCA involvement (P = 0.88) , TSH deficiency was reported in 4 aSAH cases involving anterior circulation (3 AL.COM and 1 MCA), and none involving posterior circulation (P = 0.47) Figure 3). However, this association between the involved blood vessels and hormonal disturbances was not significant as the P>0.05.

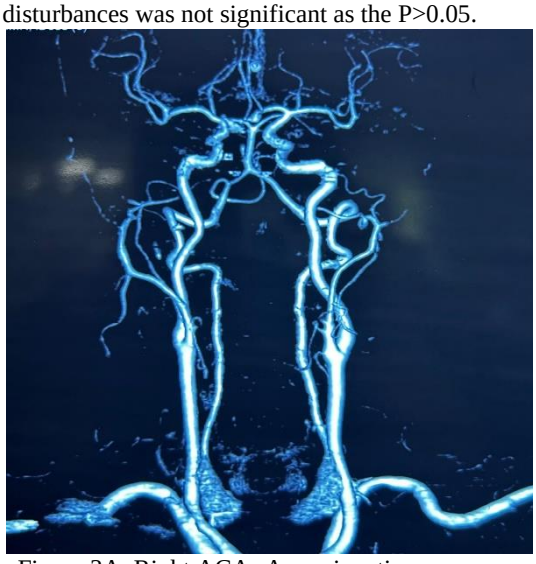

Figure 3A. Right ACA- Acom junction aneurysm

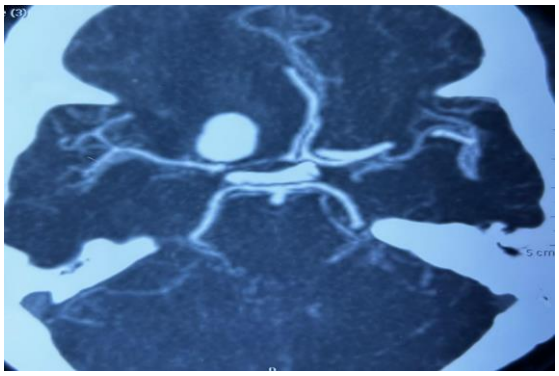

Figure 3B. Right ICA Aneurysm

DISCUSSION

Research examining the link between aneurysmal subarachnoid hemorrhage (aSAH) and neuroendocrine dysfunction remains limited, with existing evidence largely drawn from small case series and individual reports. This lack of extensive investigation has led to mixed and often contradictory findings.

One of the earlier studies in this area was conducted by Osterman in 1975, involving 50 patients evaluated approximately 3.5 months after experiencing SAH. To assess the hypothalamic-pituitary-adrenal (HPA) axis, researchers measured the circadian rhythm of plasma 11-hydroxycorticosteroids and administered the Metyrapone test. Gonadal and thyroid function were evaluated through both clinical examination and baseline hormonal levels. The study concluded that hormonal disturbances were relatively rare: 6% of participants had disrupted cortisol circadian rhythm, 11% showed abnormal results in the Metyrapone test, and 2% exhibited mild thyroid dysfunction. No cases of hypogonadism were identified.

In a separate study, Kreitschmann-Andermahr and colleagues evaluated 21 patients between 14 and 43 months following aSAH. They used a series of dynamic tests—including thyrotropin-releasing hormone, LH-releasing hormone, the Arginine test, and the insulin tolerance test—to investigate pituitary function. Endocrine disturbances were found in 43% of the patients, including isolated deficiencies in ACTH (n=4) and GH (n=3), as well as combined deficiencies in both ACTH and GH (n=2).

Another investigation by Brandt et al. assessed 10 individuals one year after aSAH using standard laboratory tests and dynamic hormonal assessments. Half of the patients (50%) demonstrated some form of pituitary hormone deficiency. These included

isolated or combined somatotroph and gonadotroph deficits, although all patients retained normal thyroid and adrenal gland function.

In a study by Dimopoulou et al., 30 patients were assessed between 12 and 24 months following aneurysmal subarachnoid hemorrhage (aSAH). The results showed that endocrine dysfunction was relatively common, affecting 47% of those screened. Growth hormone (GH) deficiency emerged as the most prevalent hormonal abnormality, indicated by reduced levels of insulin-like growth factor 1 (IGF-1) in a significant portion of the cohort. While dynamic testing is generally recommended for evaluating GH reserve and somatotroph function, accumulating evidence suggests that low IGF-1 levels alone are a reliable indicator of GH deficiency. In this study, hypogonadism was the next most frequently observed endocrine issue after GH deficiency.

To assess adrenal function, the study employed a low-dose (1-μg) ACTH stimulation test. This method is widely accepted as a standard for evaluating the hypothalamic-pituitary-adrenal (HPA) axis, as its results strongly align with those obtained from the insulin-induced hypoglycemia test. Among the participants, only three exhibited a reduced cortisol response. Despite the low incidence, this finding is clinically significant, as undiagnosed cortisol deficiency can have severe consequences—particularly when the patient is exposed to physical or emotional stress and unable to mount an adequate physiological response.
In parallel, experimental studies using porcine models of SAH have demonstrated that the endocrine stress response initiates rapidly—within 15 minutes—via the HPA axis. These studies have shown early surges in ACTH, cortisol, and aldosterone levels. Additionally, prompt activation of the sympathetic nervous system was also observed.

A prospective observational study conducted by Khajeh et al. (2014) examined the prevalence and progression of anterior pituitary dysfunction in patients following aneurysmal subarachnoid hemorrhage (aSAH). Endocrine function was assessed in the fasting state at three different time points: baseline, six months, and fourteen months. The study involved 84 patients, with a mean age of 55.8 ± 11.9 years. Findings revealed that pituitary dysfunction affected 39% of patients at baseline, decreased to 26% at six months, and further declined to 7% at fourteen months. The most frequently observed hormonal deficiencies were related to gonadotropins (34%) and growth hormone (GH) (31%). Baseline assessments were conducted within an average of 32 days post-hemorrhage. Among those with gonadotropin deficiency, all female participants were postmenopausal, with a mean age of 58 ± 10 years. GH deficiency was present in 31% of the cases, while deficiencies in TSH and ACTH were each observed in 1% of the patients.

The prevalence of hypopituitarism in patients after SAH was found to be higher compared to that in the general population. Additionally, the occurrence of pituitary dysfunction appeared to decrease over time and was associated with the clinical severity and complications of SAH, suggesting a potential causal relationship. The study concluded that pituitary dysfunction represents a notable complication in SAH survivors, with hydrocephalus identified as an independent clinical predictor of long-term hormonal disturbances.

Multiple studies have reported that GH and gonadotropin deficiencies occur more frequently than TSH or ACTH deficiencies. This trend implies that the anterior pituitary lobe may be more susceptible to damage following SAH, potentially due to its cellular composition, which is rich in somatotrophs, gonadotrophs, and thyrotrophs. The increased vulnerability might also be explained by anatomical or vascular factors. Specifically, the anterior lobe is supplied by long pituitary portal vessels that pass over the diaphragma sellae, whereas the rest of the gland receives blood from the middle and inferior pituitary arteries.

Acute hormonal deficiencies may result from mechanical pressure, vasospasm, hemorrhage, or ischemia, and in some cases, these disruptions are reversible with treatment. Consequently, some hormone levels may normalize over time. However, during the later phases, infarctions or structural damage to the hypothalamic-pituitary axis may lead to the development of new or permanent hormone deficiencies. These mechanisms could account for both the recovery of some functions and the emergence of new deficiencies.

Although many patients show recovery of pituitary function during follow-up, a subset continues to experience persistent dysfunction. Some evidence suggests the adult pituitary gland may have regenerative potential after injury, which could explain the partial restoration of hormonal function. Therefore, early identification of pituitary dysfunction may be beneficial in improving long-term outcomes after SAH. Notably, GH deficiency has been linked to symptoms such as fatigue, low energy, and cognitive impairments—including issues with memory and planning—which can interfere with rehabilitation and recovery.

A study conducted by Pinaki Dutta et al. (2012) investigated whether the location of an aneurysm—in the middle cerebral artery (MCA) versus the anterior communicating artery (A.COM)—influences the occurrence of hormonal deficiencies following aneurysmal subarachnoid hemorrhage (aSAH). The results indicated no significant difference in the prevalence of endocrine abnormalities between the two groups, suggesting that hormonal disturbances are not directly correlated with aneurysm location. Additionally, the study found no association between the severity of hormonal deficiencies and the clinical grade of SAH, as measured by the Hunt-Hess scale, or the radiological severity of hemorrhage.

The research included 60 aSAH patients, comprising 37 males and 23 females, with a mean age of 44.9 ± 13.1 years. Among the female participants, 12 were postmenopausal. Of the total cohort, 23 cases resulted from MCA aneurysm rupture, while 37 were due to A.COM aneurysm rupture. Growth hormone (GH) status was evaluated using insulin-like growth factor 1 (IGF-1) levels. At the time of ictus, IGF-1 levels were within normal limits (mean ± SD: 149.00 ± 16.30 ng/dL), but declined significantly to 96.10 ± 56.35 ng/mL at the six-month follow-up (P = 0.05).

At initial presentation, hypogonadism was observed in eight patients. Interestingly, by the six-month mark, four cases of new-onset hypogonadism were identified, despite resolution in the initial cases. GH deficiency, based on age- and gender-adjusted IGF-1 values, was identified in seven patients at the time of ictus. After six months, five individuals were found to have GH deficiency—two of whom developed it during follow-up, while the remaining three had persistent GH deficiency since ictus. Central hypothyroidism, indicated by low T4 levels, was seen in three patients initially; two of these cases resolved, while one case persisted after six months.

At the six-month follow-up, 31.6% of the participants exhibited one or more pituitary hormone deficiencies. Central hypogonadism was the most prevalent (36.6%), followed by GH deficiency (15%). Although the differences between MCA and A.COM aneurysm groups were not statistically significant, there was a notable trend: prolactin deficiency and the syndrome of inappropriate antidiuretic hormone secretion (SIADH) appeared more frequently in patients with A.COM-related aSAH.

The study emphasized the importance of endocrine evaluation not only at the initial stage but also at follow-up intervals—particularly between six and twelve months post-aSAH—to ensure the timely identification and management of hormonal dysfunction.

Pereira et al. (2013) conducted a study involving 66 patients diagnosed with aneurysmal subarachnoid hemorrhage (aSAH), assessing their pituitary hormone levels within the first 15 days of hemorrhage. The average duration for endocrine evaluation was 7.4 ± 6.6 days post-aSAH, which contrasts with 2.64 ± 0.86 days in our study. In their cohort, 66.7% of participants were female and 33.3% were male, with a mean age of 48.3 ± 13.8 years—demographics that closely resemble our study, which had 60% female and 40% male participants and a mean age of 55.24 ± 12.39 years. Pereira's findings showed that 59.1% of patients exhibited pituitary dysfunction. The most frequently observed deficiency was in gonadotropins (LH/FSH, 34.8%), followed by growth hormone (GH) deficiency (28.7%), adrenocorticotropic hormone (ACTH) deficiency (18.1%), and thyroid-stimulating hormone (TSH) deficiency (9%). In contrast, our study identified GH deficiency (48%) as the most prevalent hormonal abnormality, followed by ACTH and FSH/LH deficiencies (24% each), and TSH deficiency (16%). Moreover, multiple pituitary axis involvement was reported in 25.7% of Pereira's patients, compared to 32% in our study. Their analysis also indicated a higher prevalence of hormonal dysfunction in patients with a Glasgow Coma Scale (GCS) score below 13, Hunt-Hess grade above 4, and Fisher grade 4. However, no significant relationship was found between hormone abnormalities and the duration of hospitalization or patient outcomes. Similarly, our data did not reveal any significant association between endocrine dysfunction and variables such as sex, SAH grade, or aneurysm location.

Another relevant comparison is the study by Klose et al. (2010), which evaluated pituitary function in 26 aSAH patients approximately 7 days after onset. The incidence of pituitary dysfunction was 58%, with FSH/LH deficiency present in 93.3% of cases. Additionally, 35% of patients showed low triiodothyronine (T3) levels, 12% had reduced cortisol, and 15% had diminished GH/IGF-1 levels. Posterior pituitary dysfunction, typically indicated by sodium imbalance due to antidiuretic hormone (ADH) irregularity, was not evaluated in our study, as such disturbances are widely recognized and anticipated in both SAH patients and those undergoing neurosurgical procedures.

Our results are also consistent with those reported by Jaiswal et al. in a prospective investigation of 100 aSAH patients who underwent comprehensive clinical and hormonal evaluations during the acute phase. Their study population consisted of 62% females and 38% males, mirroring the gender distribution seen in our data. The average patient age was 43.6 years. Most aneurysms in Jaiswal's study were located in the anterior circulation (n=95), specifically in the A.COM (n=49), MCA (n=15), P.COM (n=16), ICA (n=9), anterior choroidal artery (n=1), and distal anterior cerebral artery (n=5). Aneurysms in the posterior circulation (n=5) were limited to the basilar artery (n=4) and posterior cerebral artery (n=1). Our study similarly noted higher aneurysm frequency in the anterior circulation, with distribution as follows: A.COM (8), MCA (8), ICA (4), SCA (2), BA (2), and VA (1).

Jaiswal et al. identified GH deficiency as the most common hormonal abnormality, followed by deficiencies in gonadotropins, corticotropins, and thyrotropins. This order aligns with our findings, except that ACTH (corticotropin) deficiency was more prominent than gonadotropin deficiency in our cohort. While hyperprolactinemia was reported in 10 patients in their study, it was not observed in ours. Regarding the number of hormonal axes involved, Jaiswal et al. reported single-axis dysfunction in 26% of cases and multi-axis dysfunction in 67%, compared to our findings of 36% and 32%, respectively. The overall rate of pituitary dysfunction in their cohort was 93%, notably higher than the 68% seen in our patients. Their results reinforce the recommendation for routine endocrine evaluation in the acute stage of aSAH.

Additionally, a study by Kronvall et al. reported a 37% prevalence of pituitary dysfunction in the acute phase of aSAH. Their data also suggested that endocrine abnormalities may be linked to poorer clinical outcomes and are more frequently found in patients with bleeding near the hypothalamus.

CONCLUSION

Neuroendocrine dysfunction was observed in 68% of patients during the acute phase of aneurysmal subarachnoid hemorrhage (aSAH). Among these, 32% exhibited dysfunction in a single pituitary axis, while 36% showed abnormalities across multiple hormonal axes. Growth hormone (GH) deficiency emerged as the most prevalent hormonal disorder (48%), followed by deficiencies in adrenocorticotropic hormone (ACTH), gonadotropins (LH and FSH), and thyroid-stimulating hormone (TSH). These findings support the recommendation that incorporating hormonal assessment into the clinical management of acute aSAH may contribute to improved patient outcomes.

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