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Diagnostic Value of the QoL-AGHDA Questionnaire in Assessing Growth Hormone Deficiency in Patients with Chiasmal-Sellar Region Tumors Before and After Surgical Treatment

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ABSTRACT

Abbreviation's: CSR: Chiasmal-Sellar Region; NF-Pit NETs: Non-Functioning Pituitary Neuroendo; WHO: World Health Organization; HRQoL: Health-Related Quality of Life; QoL-AGHDA: Quality of Life — Assessment of Growth; RHGH: Recombinant Human Growth Hormone; TSA: Trans Sphenoidal Adenomectomy; GH: Growth Hormone; IGF-1: Insulin-Like Growth Factor-1 (IGF-1); RSSPMCE: Republican Specialized Scientific and Practical Medical Center of Endocrinology; RRCEM: Republican Research Center of Emergency Medicine; CAD: Coronary Artery Disease; FC: Functional Class; NFPA: Non-Functioning Pituitary Adenoma

Background and Relevance

Tumors of the chiasmal-sellar region (CSR) account for up to 15–20% of all primary intracranial neoplasms, with non-functioning pituitary neuroendocrine tumors (NF-PitNETs) occupying the leading position in their structure [1]. According to the modern World Health Organization (WHO) classification, the transition to PitNET terminology reflects complex cellular differentiation (predominantly along the SF1, TPIT, and PIT1 transcription factor lineages) and the potentially invasive growth pattern of these neoplasms [2]. Due to the absence of hypersecretory endocrine syndromes at early stages, NF-PitNETs remain clinically latent for a prolonged period and are verified predominantly at the macro- and giant adenoma stage (exceeding 10–30 mm), when neurological and neuro-ophthalmological impairments manifest due to compression of the optic chiasm and cavernous sinus structures [3].

Pathophysiology of Hypopituitarism and Systemic Consequences of Growth Hormone Deficiency

Chronic mass effect of the tumor on the hypophyseal stalk portal system and elevated intrasellar pressure lead to adenohypophyseal ischemia and sequential loss of its tropic functions [4]. The somatotropic axis exhibits the highest vulnerability to mechanical and vascular injury; consequently, adult growth hormone deficiency (AGHD) develops earliest and is recorded in the vast majority of patients with CSR macroadenomas already at the preoperative stage [5]. Contemporary international clinical studies consider AGHD not as an isolated laboratory phenomenon, but as a severe multisystem metabolic syndrome associated with increased cardiovascular morbidity and premature mortality [6]. Chronic deficiency of growth hormone (GH) and insulin-like growth factor-1 (IGF-1) induces a complex of pathological alterations in the body:

- Body composition and metabolic disturbances: progressive accumulation of visceral fat alongside loss of lean muscle mass, development of insulin resistance, atherogenic dyslipidemia, and endothelial dysfunction [7].
- Musculoskeletal impairment: accelerated bone resorption, decreased bone mineral density (osteopenia and osteoporosis), and pronounced sarcopenia, markedly reducing exercise tolerance [8].
- Neurocognitive and psychoemotional disorders: reduced energy potential, disrupted sleep architecture, emotional lability, anxiety-depressive states, and social maladjustment [9].

Impact of Surgical and Radiation Treatment on Somatotropic Status

Endoscopic endonasal transsphenoidal adenomectomy (TSA) is the primary treatment modality for symptomatic CSR neoplasms, supplemented by stereotactic radiosurgery in cases of incomplete resection or recurrence [1]. Despite advancements in minimally invasive neurosurgical techniques, decompression of the sellar region does not always restore somatotropic function. Conversely, intraoperative traction of the pituitary stalk, resection of tumors with high invasion grades according to the Knosp scale (grades 3–4), and delayed post-radiation fibrosis frequently aggravate hypopituitarism, bringing the prevalence of persistent postoperative AGHD to 75–90% [10,11].

Diagnostic Barriers and the Role of the QoL-AGHDA Questionnaire

Verification of somatotropic insufficiency in routine clinical practice involves considerable challenges. Basal GH levels are uninformative due to pulsatile secretion, while serum IGF-1 concentrations remain within age-adjusted reference ranges in a substantial proportion of adults with confirmed AGHD, limiting its diagnostic sensitivity [5,12]. Provocative stimulation tests (insulin tolerance test, glucagon test, or macimorelin test) require strict inpatient conditions, carry risks of severe hypoglycemia or cardiovascular complications, and have extensive contraindications in comorbid and elderly patients [12]. Under these circumstances, health-related quality of life (HRQoL) assessment using the disease-specific psychometric questionnaire QoL-AGHDA (Quality of Life — Assessment of Growth Hormone Deficiency in Adults) becomes a key instrument for primary screening and dynamic monitoring [13]. Unlike generic scales, QoL-AGHDA specifically evaluates domains most sensitive to GH deficiency (vitality, memory, concentration, emotional control, and social interaction). Recent prospective cohort studies confirm that QoL-AGHDA scores correlate with hypopituitarism severity, objectify postoperative psychosomatic deterioration even at borderline IGF-1 levels, and serve as a primary criterion for selecting patients for recombinant human growth hormone (rhGH) replacement therapy and evaluating its efficacy [14,15].

Objective of the Study

To investigate the diagnostic value of the Quality of Life — Assessment of Growth Hormone Deficiency in Adults (QoL-AGHDA) questionnaire in patients with non-functioning pituitary adenomas and other chiasmal-sellar region tumors before and after surgical treatment.

Materials and Methods

Between 2024 and 2026, we examined 136 adult patients with AGHD caused by functionally inactive neoplasms of the chiasmal-sellar region (NFPAs). The mean age of men was 44.2 years, and that of women was 46.4 years. Disease duration ranged from 2 months to 25 years. Surgical treatment (transnasal selective hypophysectomy — TSA) was performed in 36 (26.5%) patients at the Neurosurgery Departments of the Republican Research Center of Emergency Medicine (RRCEM) and the Republican Specialized Scientific and

Practical Medical Center of Endocrinology (RSSPMCE) of the Ministry of Health of the Republic of Uzbekistan, while 1 patient underwent bifrontal craniotomy at the N.N. Burdenko National Medical Research Center of Neurosurgery (Moscow). The control group comprised 20 apparently healthy individuals matched for sex and age.

Examination Methods Included

(1) general clinical assessment (evaluation of endocrine and neurological status, anthropometry — height, body weight, waist circumference, hip circumference, body mass index, and AGHD questionnaire assessment);

(2) instrumental investigations (full-color perimetry, ophthalmoscopy, visometry, ECG, bone densitometry, CT/MRI of the sella turcica and adrenal glands, and pelvic ultrasound);

(3) hormonal blood assays (GH, IGF-1, LH, FSH, PRL, TSH, ACTH, testosterone, estradiol, progesterone, cortisol, free T3, and free T4) performed by immunochemiluminescence in the Hormonal Research Laboratory of RSSPMCE (Head of Lab: S.Kh. Saidova); and

(4) biochemical blood tests (LDL, HDL, total cholesterol, triglycerides, calcium, etc.). To assess patients' quality of life, the specialized QoL-AGHDA (Quality of Life — Assessment of Growth Hormone Deficiency in Adults) questionnaire developed within the KIMS project (Wallymahmed M.E. et al., 1999) and comprising 25 dichotomous questions was utilized. A cumulative score of more than 11 positive answers was interpreted as a clinical marker of probable AGHD in adults [13].

Statistical data processing was performed using Microsoft Excel and STATISTICA 6.0 software packages. Mean values (M) and standard errors of the mean (m) were calculated. The Mann–Whitney U test / Wilcoxon test was applied to assess statistical significance of differences for quantitative variables, and Fisher's exact test was used for small samples and qualitative parameters. Correlation analysis was conducted using Spearman's rank correlation coefficient. Differences were considered statistically significant at $p < 0.05$.

Results

Among the 136 examined patients, the following sellar region lesions were identified: non-functioning pituitary adenoma (NFPA) in 67 (49.3%) patients, acromegaly (with AGHD developing during therapy) in 17 (12.5%), prolactinoma in 12 (8.8%), craniopharyngioma in 10 (7.4%), Cushing's disease in 5 (3.7%), pinealoma in 1 (0.7%), astrocytoma in 1 (0.7%), meningioma in 1 (0.7%), germinoma in 1 (0.7%), and other CSR lesions in 21 (15.4%) patients. Depending on treatment strategy and comorbidities, patients were stratified into 5 clinical groups:

- Group 1 — 67 (49.3%) patients with various pituitary adenomas receiving conservative therapy (bromocriptine, cabergoline);
- Group 2 — 36 (26.5%) patients with pituitary adenomas who underwent surgical treatment (TSA, etc.);
- Group 3 — 4 (2.9%) patients with pituitary adenomas who received radiation therapy;
- Group 4 — 8 (5.9%) patients with pituitary adenomas who received combined therapy (TSA + radiation therapy);
- Group 5 — 21 (15.4%) patients with coronary artery disease (CAD) and AGHD, subdivided into two subgroups: Subgroup 5A — patients with exertional angina functional class (FC) II (n = 4; 19.0%), and Subgroup 5B — patients with exertional angina FC III (n = 17; 81.0%).

According to CT/MRI of the chiasmal-sellar region, the size distribution of pituitary adenomas (n = 72) was as follows: microadenomas (< 10 mm) were detected in 11 (15.3%) patients, mesoadenomas (11–20 mm) in 11 (15.3%), macroadenomas (21–30 mm) in 16 (22.2%), and giant adenomas (> 30 mm) in 34 (47.2%) patients. According to the topographic-anatomical classification of pituitary adenoma growth direction by B.A. Kadashev (2007), tumors with endosellar-suprasellar extension predominated in 27 (37.5%) patients and total growth pattern in 21 (29.2%); laterosellar (n = 3; 4.2%), infrasellar (n = 2; 2.8%), antesellar (n = 1; 1.4%), and retrosellar (n = 1; 1.4%) variants were substantially less frequent. The sex and age distribution of examined patients according to the WHO classification is presented in Table 1. Women predominated among the examined cohort (58.09%), and the largest proportion of patients belonged to the working-age group of 30 to 59 years (63.24%).

Table 1: Distribution of Patients by Sex and Age (WHO Classification).

Age, years	Men, abs. (%)	Women, abs. (%)	Total, abs. (%)
16–29	13 (9.56%)	11 (8.09%)	24 (17.65%)
30–44	16 (11.76%)	29 (21.32%)	45 (33.09%)
45–59	17 (12.50%)	24 (17.65%)	41 (30.15%)
60–74	11 (8.09%)	11 (8.09%)	22 (16.18%)
75 and older	–	4 (2.94%)	4 (2.94%)
Total (n = 136)	57 (41.91%)	79 (58.09%)	136 (100%)

Table 2 and Figure 1 present the results of QoL-AGHDA surveying of 136 patients across clinical groups at the preoperative stage (prior to treatment initiation). As shown in the presented data, in all primary groups (Groups 1, 2, 3, 4, and Subgroup 5B), mean QoL-AGHDA scores statistically significantly exceeded the normal threshold (< 11 points) and control group values ($p < 0.05$). The sole exception was Subgroup

5A (patients with CAD FC II), where the mean score remained within normal limits (9.75 ± 0.4 points; $p > 0.05$). Next, we investigated the relationship between QoL-AGHDA quality-of-life indicators and basal plasma concentrations of GH and IGF-1 in the 136 examined patients (Table 3, Figure 2). Across all clinical groups, basal GH levels were statistically significantly reduced compared to the control group ($p < 0.05$). IGF-1 levels were also significantly lower than control values in Groups 1, 2, 3, 4, and Subgroup 5B ($p < 0.05$), whereas in Subgroup 5A they remained within the reference range ($p > 0.05$).

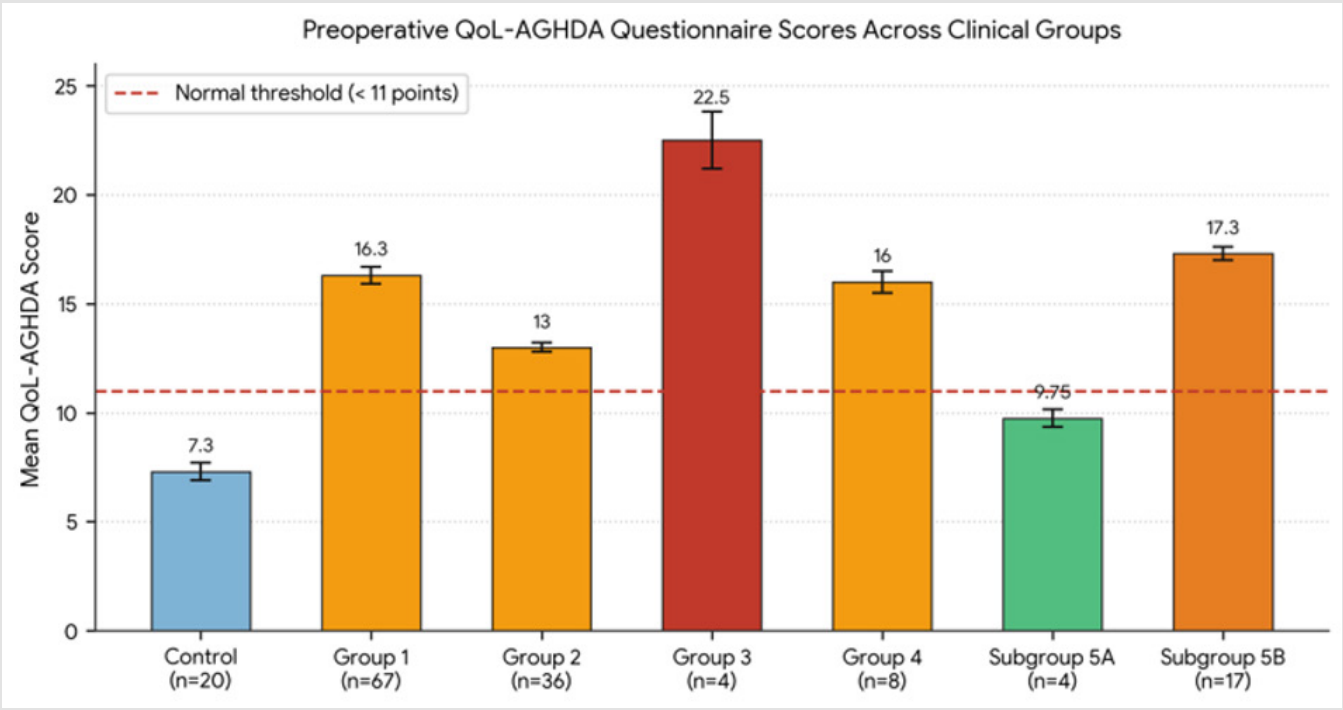


Figure 1: Mean QoL-AGHDA scores across clinical groups at the preoperative stage.

Table 2: Preoperative QoL-AGHDA Questionnaire Results by Clinical Group.

Clinical Group (Number Examined)	Mean Score, M ± m	Statistical Significance vs. Control (p)
Group 1 (n = 67)	16.3 ± 0.4	$p < 0.05$
Group 2 (n = 36)	13.0 ± 0.2	$p < 0.05$
Group 3 (n = 4)	22.5 ± 1.3	$p < 0.01$
Group 4 (n = 8)	16.0 ± 0.5	$p < 0.05$
Group 5: Subgroup 5A (n = 4)	9.75 ± 0.4	$p > 0.05$
Group 5: Subgroup 5B (n = 17)	17.3 ± 0.3	$p < 0.05$
Control Group (n = 20)	7.3 ± 0.4	–
Reference Norm	< 11 points	–

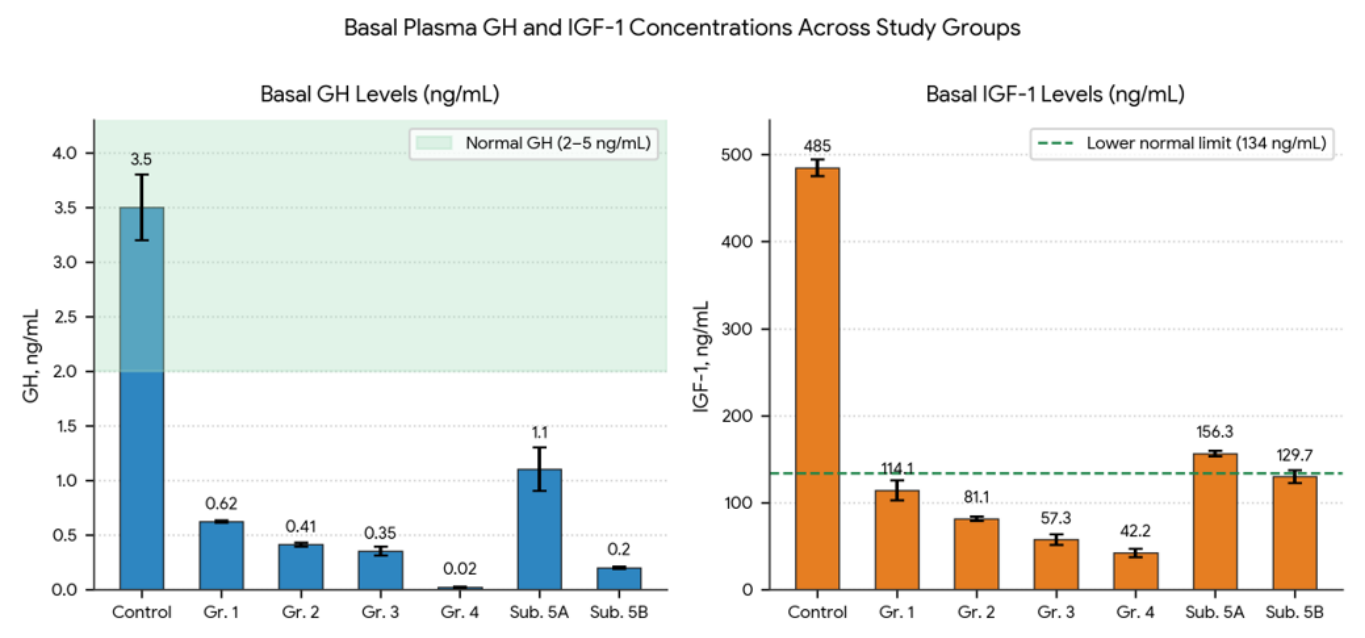


Figure 2: Basal plasma concentrations of GH (left) and IGF-1 (right) across study groups.

Table 3: Comparative Characteristics of QoL-AGHDA Scores and Basal Plasma GH and IGF-1 Levels by Group.

Group (n)	Mean QoL-AGHDA Score	GH, ng/mL (M ± m)	p_1 / p_3 (GH)	IGF-1, ng/mL (M ± m)	p_2 / p_3 (IGF-1)
Group 1 (n = 67)	16.3 ± 0.4	0.62 ± 0.01	< 0.05 / < 0.05	114.12 ± 11.5	< 0.05 / < 0.05
Group 2 (n = 36)	13.0 ± 0.2	0.41 ± 0.02	< 0.05 / < 0.05	81.10 ± 2.6	< 0.01 / < 0.01
Group 3 (n = 4)	22.5 ± 1.3	0.35 ± 0.04	< 0.05 / < 0.05	57.32 ± 6.3	< 0.01 / < 0.01
Group 4 (n = 8)	16.0 ± 0.5	0.02 ± 0.003	< 0.01 / < 0.01	42.16 ± 4.8	< 0.01 / < 0.01
Subgroup 5A (n = 4)	9.75 ± 0.4	1.1 ± 0.2	< 0.05 / > 0.05	156.3 ± 3.3	> 0.05 / > 0.05
Subgroup 5B (n = 17)	17.3 ± 0.3	0.2 ± 0.01	< 0.05 / < 0.05	129.7 ± 7.5	< 0.05 / < 0.05
Control (n = 20)	7.3 ± 0.4	3.5 ± 0.3	—	485.0 ± 9.8	—
Norm	< 11 points	2.0–5.0	—	134–836	—

Note:

- p_1 -statistical significance of GH level differences compared to the control group;
- p_2 -statistical significance of IGF-1 level differences compared to the control group;
- p_3 - statistical significance of correlation between QoL-AGHDA score and GH/IGF-1 concentrations.

A significant inverse correlation between cumulative QoL-AGHDA scores and low basal GH and IGF-1 levels was established in Groups 1, 2, 3, 4, and Subgroup 5B ($p < 0.05$), confirming the high diagnostic validity of the questionnaire in somatotropic deficiency of diverse etiology. At the next stage, we evaluated the dynamics of QoL-AGHDA quality-of-life scores and hormonal profiles in 36 operated Group 2 patients 3 months after transsphenoidal adenomectomy (Table 4, Figure 3). Three months post-TSA, the mean QoL-AGHDA score significantly increased from 13.0 ± 0.2 to 19.4 ± 0.4 points ($p < 0.05$) alongside a significant decline in basal GH (from 0.41 ± 0.02 to 0.11 ± 0.03 ng/mL; $p < 0.05$) and IGF-1 (from 81.10 ± 2.6 to 74.3 ± 2.4 ng/mL; $p < 0.05$) concentrations. These findings indicate an exacerbation of somatotropic insufficiency in the early postoperative period and substantiate the need for timely initiation of recombinant growth hormone replacement therapy.

Table 4: Dynamics of QoL-AGHDA Scores and GH, IGF-1 Levels in 36 Operated Group 2 Patients 3 Months After TSA.

Group (n)	QoL-AGHDA Before TSA	QoL-AGHDA 3 Mo Post-TSA	GH Before TSA, ng/mL	GH3 Mo Post-TSA, ng/mL	IGF-1 Before TSA, ng/mL	IGF-13 Mo Post-TSA, ng/mL
Group 2 (n = 36)	13.0 ± 0.2	19.4 ± 0.4*	0.41 ± 0.02	0.11 ± 0.03*	81.10 ± 2.6	74.3 ± 2.4*
Control (n = 20)	7.3 ± 0.4	7.3 ± 0.4	3.5 ± 0.3	3.5 ± 0.3	485.0 ± 9.8	485.0 ± 9.8
Norm	< 11 points	< 11 points	2.0–5.0	2.0–5.0	134–836	134–836
p (vs. Control)	< 0.05	< 0.05	< 0.05	< 0.01	< 0.01	< 0.01

Note:

- *- statistically significant difference 3 months after TSA compared to preoperative baseline ($p < 0.05$);
- P - statistical significance of differences compared to the control group.

Conclusions

1. Quality-of-life assessment using the QoL-AGHDA questionnaire in 136 patients with chiasmal-sellar region tumors revealed a statistically significant exceedance of the normal threshold (< 11 points) across all primary clinical groups at the preoperative stage ($p < 0.05$), with the most pronounced quality-of-life impairment observed in patients following radiation therapy (22.5 ± 1.3 points) and in those with concomitant CAD FC III (17.3 ± 0.3 points).
2. A significant inverse correlation was established between cumulative QoL-AGHDA scores and low basal plasma GH and IGF-1 concentrations in Groups 1, 2, 3, 4, and Subgroup 5B ($p < 0.05$), confirming the high diagnostic value of the questionnaire as a screening tool for adult growth hormone deficiency.
3. Three months after transsphenoidal adenomectomy (n = 36), a significant increase in mean QoL-AGHDA score from 13.0 ± 0.2 to 19.4 ± 0.4 ($p < 0.05$) was observed alongside further reductions in GH and IGF-1 levels, reflecting progression of postoperative hypopituitarism and substantiating the appropriateness of initiating rhGH replacement therapy.

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