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ORIGINAL ARTICLE

Diagnostic Utility of Serum β-Catenin in Patients with Acromegaly: A Case–Control Study

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ABSTRACT

Background: Acromegaly is a rare endocrine disorder, most commonly caused by a growth hormone (GH)–secreting pituitary adenoma. This study aimed to assess circulating β-catenin protein levels in patients with acromegaly and to compare its diagnostic performance with routine laboratory tests.

Methods: This case–control study included 60 adults with acromegaly and 30 age- and sex-matched healthy subjects (HS). Fasting blood glucose, lipid profile, renal and liver function tests, GH, insulin-like growth factor-1 (IGF-1), uric acid, and β-catenin were measured in all participants.

Results: β-catenin levels were significantly higher in patients with acromegaly than in HS. Receiver operating characteristic (ROC) curve analysis demonstrated excellent discriminatory ability of β-catenin for acromegaly, with an area under the curve (AUC) of 0.992 ($p < 0.001$). At an optimal cutoff value of > 1.811, the sensitivity was 91.7% and the specificity was 100%. DeLong's test further confirmed the superior diagnostic performance of β-catenin compared with routine tests. Correlation analysis showed that β-catenin was significantly and positively correlated with IGF-1, total cholesterol, and total serum bilirubin (TSB), while it was significantly and negatively correlated with creatinine. Multiple linear regression identified IGF-1, GH, urea, GPT, ALP, TSB, and uric acid as independent predictors of β-catenin levels.

Conclusion: β-catenin levels are elevated in patients with acromegaly and exhibit excellent diagnostic performance. Monitoring β-catenin may provide clinically useful information and could assist in risk stratification and management of acromegaly.

Key words: β-catenin; Acromegaly; Growth hormone; Insulin-like growth factor-1; Lipid profile.

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INTRODUCTION

Acromegaly is a chronic endocrine disorder that is most commonly caused by excessive secretion of growth hormone (GH), typically due to a pituitary adenoma. The resulting elevation in insulin-like growth factor 1 (IGF-1) contributes to a wide range of systemic complications, including metabolic disturbances, cardiovascular morbidity, and musculoskeletal disorders. Because the onset of symptoms is often insidious, diagnosis is frequently delayed, which may increase disease-related complications and reduce patients' quality of life [1, 2]. Acromegaly patients may have a number of systemic complications in addition to apparent growth of the hands, feet, and facial features. Changes in excessive perspiration, skin texture, and joint discomfort are some of the symptoms. Acromegaly can result in major health problems like cardiovascular disease, hypertension, and diabetes if treatment is not received [3].

The human protein β-catenin, which is encoded by the CTNNB1 gene, consists of 781 amino acid residues. It is composed of 12 incomplete Armadillo repeats in the center, with separate N-terminal and C-terminal domains on either side [4]. β-catenin was first identified in the early 1990s as part of mammalian cell adhesion and as the protein that anchors the cadherin cytoplasmic domain [5].

A crucial transcriptional co-activator, β-catenin is involved in the Wnt signaling pathway, which controls a variety of cellular functions such as migration, proliferation, and cell fate determination. Numerous cancer forms have been linked to the onset and spread of dysregulation of the Wnt/β-catenin pathway [6].

Pituitary tumors are among the neoplasms that have been linked to aberrant β-catenin activation. In non-functioning pituitary neuroendocrine tumors (NF-PitNETs), the phosphorylation state of β-catenin, specifically at Serine 552, has been linked to tumor invasion and recurrence [7].

This study aims to estimate β-catenin levels in patients with acromegaly. Understanding these relationships might help identify possible treatment options for this complicated illness. However, the clinical significance of serum β-catenin in patients with acromegaly remains poorly defined.

PATIENTS AND METHODS

- **Population and study design:** The scientific and ethical committee of the National Diabetes Center at Mustansiriyah University authorized this case–control study, and all participants provided verbal informed consent. The study was conducted between November 2024 and January 2025. Sixty individuals with acromegaly were diagnosed according to established international guidelines based on elevated age-adjusted serum IGF-1 levels, lack of GH suppression during oral glucose tolerance testing (when applicable), and pituitary MRI evidence of a GH-secreting adenoma. All enrolled patients were receiving long-acting somatostatin analog therapy. At the time of blood sampling, patients were stratified based on biochemical control status using age-adjusted IGF-1 concentrations to distinguish active versus controlled disease. Patients aged 35–65 years with a confirmed diagnosis of acromegaly were included and age- and sex-matched with 30 apparently healthy subjects (HS). All patients received Octreotide LAR once every four weeks. Patients with pregnancy, active malignancies, thyroid disease, heart disease, alcoholism, and smokers were excluded.

- **Anthropometric measurements:** Age, height, weight, and body mass index (BMI)—calculated as weight divided by the square of height—were recorded for patients and HS [8].

- **Collection of blood samples:** Blood samples were collected using a 5 mL disposable syringe between 8:30 and 11:30 a.m. after an 8–12 hour fast. Blood (5 mL) was transferred to a gel tube and allowed to clot at room temperature. Serum was separated by centrifugation for 10 minutes at 3000 rpm. GH and IGF-1 levels were measured using the Cobas e411 analyzer (Roche). Biochemical tests (fasting blood glucose, urea, creatinine, liver function tests, lipid profile, and uric acid) were determined using Cobas c111. In addition, human β-catenin was measured in vitro using a BioSource (USA) ELISA kit (catalog number: E-EL-H0666; storage temperature: −20 °C) with an ELISA plate reader (Human Reader, Germany) and a sandwich enzyme immunoassay method (ELISA).

- **Statistical analysis:** All data were analyzed using SPSS (version 25) and MedCalc (version 20.027). The Shapiro–Wilk test was used to assess the distribution of variables. Quantitative variables were expressed as mean ± SD for normally distributed data, or median with interquartile range for skewed data. Differences between study groups were assessed using the independent *t*-test. Correlations were assessed using Pearson's correlation coefficient. Multiple linear regression analysis was used to identify independent relationships. Receiver operating characteristic (ROC) curve analysis was performed to determine the diagnostic ability of β-catenin, and the optimal cut-off value was determined using the Youden index. DeLong's test was used to compare the discriminatory power of each biomarker with β-catenin and conventional tests.

RESULTS

- **Subject characteristics:**
  There were 30 healthy subjects (HS) as a control group (43.56±0.98 years) and 60 age- and sex-matched patients with acromegaly (44.47±8.92 years). (Table 1) shows that BMI was significantly higher in patients than in HS (p = 0.001).
- **Biochemical and traditional biomarker tests:**
  The levels of FBG, GH, IGF-1, ALP, total cholesterol (TC), triglycerides (TG), very-low-density lipoprotein (VLDL), and low-density lipoprotein (LDL) were significantly higher in patients with acromegaly compared with HS. In contrast, high-density lipoprotein (HDL) and urea levels were significantly lower in the acromegaly group ($p \leq$ 0.001) compared with HS. However, no statistically significant differences were observed between the two groups in uric acid, total serum bilirubin (TSB), and glutamic oxaloacetic transaminase (GOT), as shown in (Table 1).
- **Serum β-catenin levels in study groups:**
  According to (Table 1), serum β-catenin levels were significantly higher in patients with acromegaly (4.47 ± 1.23) than in HS (1.33 ± 0.20), with a p-value ≤ 0.001.
- **Correlation of β-catenin with other parameters:**
  In the acromegaly group, serum β-catenin levels were significantly positively correlated with IGF-1 (r = 0.370, p = 0.004), total cholesterol (r = 0.491, p < 0.0001), and TSB (r = 0.332, p = 0.009). These correlations are summarized in (Table 2).
- **Multiple regression analysis of β-catenin as a predictor of acromegaly:**
  The relationship between serum β-catenin and laboratory and clinical parameters was evaluated using multivariate linear regression analysis. IGF-1, GH, urea, GPT, ALP, TSB, and uric acid were identified as independent and significant predictors of β-catenin. These findings were confirmed through multiple linear regression analysis, as summarized in (Table 3).
- **ROC Curve Analysis:**
  Receiver operating characteristic (ROC) curve analysis was used to examine how well serum β-catenin levels could differentiate between patients with acromegaly and control individuals. The ROC curve for acromegaly indicated excellent diagnostic validity, with high sensitivity (91.7%) and specificity (100.0%). The optimal prediction of acromegaly was achieved, as shown by an area under the curve (AUC) of 0.992 (p < 0.001) (Figure 1A).

The combined predictive performance of β-catenin with conventional biomarkers (GH and IGF-1) was evaluated using DeLong's test for correlated ROC curves, as shown in (Figure 1B). The differences between the areas under the curve (AUCs) were 0.0783 and 0.142, with corresponding p-values of 0.0080 and 0.0001, respectively. These findings indicate a statistically significant difference between β-catenin and GH (p = 0.0080) and between β-catenin and IGF-1 (p = 0.0001).

Table 1. Demographic characteristics, biochemical tests, and hormonal biomarkers in patients with acromegaly and healthy subjects (HS).

Parameter	Acromegaly	HS	p
Subject characteristics			
Age (years)	44.47 ± 8.92	43.56 ± 0.98	0.781
BMI (kg/m²)	33.98 ± 3.13	24.59 ± 1.98	**0.001**
Sex, n (%)			
Female	30 (50)	15 (50)	[a]
Male	30 (50)	15 (50)	[a]
Biochemical tests			
FBG (mg/dL)	132.60 ± 73.49	92.03 ± 4.79	**0.0001**
Urea (mg/dL)	22.08 ± 5.00	26.72 ± 4.26	**0.0001**
Creatinine (mg/dL)	0.69 ± 0.21	0.723 ± 0.077	0.359
Uric acid (mg/dL)	4.79 ± 1.40	4.39 ± 1.24	0.168
TSB (mg/dL)	0.60 ± 0.354	0.49 ± 0.251	0.099
GOT (U/L)	16.49 ± 3.66	16.30 ± 2.92	0.791
GPT (U/L)	14.60 ± 4.55	15.40 ± 3.30	0.348
ALP (U/L)	96.13 ± 32.55	65.13 ± 16.07	**0.0001**
Total cholesterol (mg/dL)	178.40 ± 27.89	101.81 ± 10.11	**0.0001**
Triglycerides (mg/dL)	180.36 ± 14.33	88.20 ± 26.58	**0.0001**
VLDL (mg/dL)	30.43 ± 16.22	17.50 ± 5.41	**0.001**
LDL (mg/dL)	104.05 ± 28.99	82.03 ± 9.49	**0.001**
HDL (mg/dL)	44.48 ± 7.38	61.20 ± 11.25	**0.001**
Hormonal biomarkers			
GH (ng/mL)	7.07 ± 1.81	2.58 ± 1.33	**0.001**
IGF-1 (ng/mL)	772.60 ± 260.75	443.26 ± 180.27	**0.001**
β-catenin	4.47 ± 1.23	1.33 ± 0.20	**0.001**

Data are presented as mean ± SD unless otherwise indicated; categorical data are shown as n (%).
Significant p-values (p < 0.05) are shown in bold.
[a] Sex distribution was matched by design; therefore, no statistical test was performed.
Abbreviations: ALP, alkaline phosphatase; BMI, body mass index; FBG, fasting blood glucose; GH, growth hormone; GOT, glutamic oxaloacetic transaminase; GPT, glutamate pyruvate transaminase; HDL, high-density lipoprotein; HS, healthy subjects; IGF-1, insulin-like growth factor-1; LDL, low-density lipoprotein; TSB, total serum bilirubin; VLDL, very-low-density lipoprotein.

Table 2. Correlation of serum β-catenin with selected parameters in patients with acromegaly and healthy subjects (HS).

Parameter	Acromegaly (n=60)		HS (n=30)	
	r	p-value	r	p-value
IGF-1 (ng/mL)	0.370	**0.004**	0.354	0.055
Creatinine (mg/dL)	−0.397	**0.002**	0.154	0.415
Total cholesterol (mg/dL)	0.491	**0.0001**	−0.163	0.390
TSB (mg/dL)	0.332	**0.009**	0.174	0.357

r, Pearson correlation coefficient.
Significant p-values (p < 0.05) are shown in bold.
Abbreviations: HS, healthy subjects; IGF-1, insulin-like growth factor-1; TSB, total serum bilirubin.

Table 3. Multiple linear regression analysis of independent predictors of serum β-catenin in patients with acromegaly.

Predictor	Unstandardized coefficients		Standardized	*t*	*p*-value
	B	SE	β		
Age (years)	0.0	0.0	−0.1	−2.7	0.1
BMI (kg/m²)	0.0	0.0	0.1	1.4	0.2
IGF-1 (ng/mL)	0.0	0.0	0.5	6.0	**0.0001**
GH (ng/mL)	−0.2	0.0	−0.3	−3.6	**0.0010**
FBG (mg/dL)	0.0	0.0	−0.1	−1.7	0.1
Urea (mg/dL)	0.1	0.0	0.4	3.2	**0.0020**
Creatinine (mg/dL)	0.0	0.0	0.2	1.2	0.2
GOT (U/L)	0.0	0.0	0.0	−0.2	0.9
GPT (U/L)	−0.1	0.0	−0.2	−2.7	**0.0080**
ALP (U/L)	0.0	0.0	0.2	3.1	**0.0020**
TSB (mg/dL)	1.0	0.3	0.2	3.1	**0.0020**
Total cholesterol (mg/dL)	0.0	0.0	1.0	1.4	0.2
Triglycerides (mg/dL)	0.0	0.0	0.5	0.6	0.5
VLDL (mg/dL)	−0.1	0.1	−0.9	−0.7	0.5
LDL (mg/dL)	0.0	0.0	−0.4	−0.9	0.4
HDL (mg/dL)	0.0	0.0	−0.1	−0.5	0.6
Uric acid (mg/dL)	−0.2	0.1	−0.2	−2.7	**0.0090**

Significant *p*-values ($p < 0.05$) are shown in bold.
B, unstandardized regression coefficient; SE, standard error; β, standardized regression coefficient.
Abbreviations: ALP, alkaline phosphatase; BMI, body mass index; FBG, fasting blood glucose; GH, growth hormone; GOT, glutamic oxaloacetic transaminase; GPT, glutamate pyruvate transaminase; HDL, high-density lipoprotein; IGF-1, insulin-like growth factor-1; LDL, low-density lipoprotein; TSB, total serum bilirubin; VLDL, very-low-density lipoprotein.

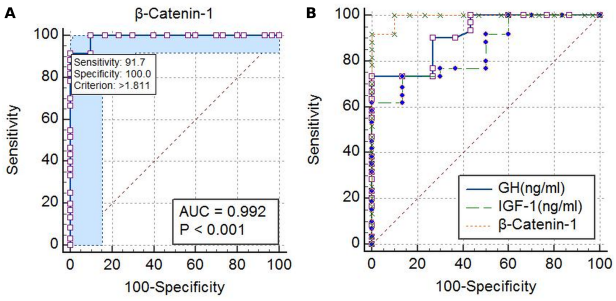

Figure 1. Receiver operating characteristic (ROC) analysis for acromegaly diagnosis. **(A)** ROC curve of serum β-catenin for discriminating patients with acromegaly from healthy subjects (HS). **(B)** Comparative ROC curves of serum β-catenin, GH, and IGF-1.

DISCUSSION

Acromegaly is a hormonal condition caused by an overabundance of GH, typically as a result of a benign pituitary tumor [9]. Because acromegaly is characterized by excessive growth hormone (GH) synthesis, which alters metabolism and body composition, the link between BMI and acromegaly is complicated. According to studies, people with acromegaly frequently have greater BMIs and different metabolic profiles than the control group. Obesity may cover up or worsen the symptoms of acromegaly, which further complicates this link [10]. Also, this study agrees with Roh E. et al. and Abdullah et al. [11, 12], who explain the increase in BMI by mineral density of the bone.

In the current study, there was a highly significant increase in GH and IGF-1 in patients with acromegaly compared with HS. The anterior pituitary gland produces growth hormone. GH mainly stimulates the liver to produce IGF-1, which mediates many of GH's effects on metabolism and growth. Increased GH causes greater IGF-1 secretion, which promotes the development of tissues and bones in acromegaly [13].

In the current study, increased liver function parameters were observed in patients with acromegaly; this result agrees with the Apaydin T. et al study. Through the development of NAFLD and related metabolic disorders, acromegaly has a significant effect on liver function. Liver dysfunction is caused by increased GH, IGF-1, and hereditary predispositions [14]. Although cardiovascular issues are the primary cause of acromegaly's high death rate, the condition has also been linked more frequently to other neoplasia, which may increase mortality in affected patients. Hyperlipidemia, mainly defined by hypertriglyceridemia and reduced HDL levels, is present in up to 50% of people with acromegaly [15]. GH affects lipid metabolism by encouraging lipolysis and perhaps raising hepatic free fatty acid levels, which might increase steatosis [16].

Acromegaly management is complex and calls for personalized treatment plans. Surgery is the first choice of treatment; medical interventions are used if surgery is not feasible or is not successful [1]. First-generation somatostatin receptor ligands (SRLs), such as lanreotide autogel (LAN) and octreotide long-acting release (OCT LAR), are standard medical options, but their effectiveness in normalizing biochemical tests is moderate. In patients resistant to SRLs, second-line treatments include pasireotide long-acting release (PAS LAR) and the GH-receptor antagonist pegvisomant (PEG)

[2]. Acromegaly is also associated with a range of comorbidities that significantly affect patient health and quality of life. The most prevalent comorbidities include metabolic and endocrine disorders and cardiovascular disease, which may make treating patients with acromegaly more complicated [3].

The multifunctional protein β-catenin is essential for Wnt signaling and cell adhesion, and it has important effects on both illness and development [17]. β-catenin has important roles in cancer progression, cellular differentiation, and cell fate determination. β-catenin functions as a transcriptional co-regulator and is involved in several biological activities, including immune response control, cell adhesion, and gene transcription [18, 19].

In the current study, β-catenin was increased in patients with acromegaly compared with the control group. These results indicate that the Wnt/β-catenin signaling pathway is a key player in differentiation and regulation of cell proliferation. Because GH-secreting adenomas occur in pituitary tumors, which are relevant in the setting of acromegaly, dysregulation of this pathway is frequently linked to the development of malignancies [20].

However, it remains difficult to explain the precise mechanism through which β-catenin affects patients with acromegaly, and some information may be missed because these biomarkers are novel and there is a lack of references and studies, making it difficult to understand their clear role in pathophysiology.

This study offers a new tool for diagnosing, developing, or monitoring acromegaly. Investigating the function of β-catenin may provide deeper insight into the mechanisms of acromegaly. However, the relatively small sample size may limit the generalizability of the findings, and because acromegaly is considered a rare disease, the findings need to be validated in independent cohorts before clinical application. Confounding factors, such as comorbidities or medications, could also influence β-catenin levels.

CONCLUSION

Serum β-catenin demonstrated high diagnostic accuracy for identifying and excluding acromegaly, indicating that it may serve as a sensitive and specific biomarker in affected patients. Monitoring β-catenin levels may complement existing diagnostic approaches and could support earlier detection and improved clinical management, with potential implications for identifying new therapeutic targets. In addition, patients with acromegaly should have liver function and related hepatic conditions regularly monitored and appropriately managed.

ETHICAL DECLARATIONS

Ethics Approval and Consent to Participate

This study was conducted in accordance with the Declaration of Helsinki. Ethical approval was obtained from the Local Ethics Committee of the National Diabetes Center, Al-Mustansiriyah University (Ref: NDSEC/20/435; June 6, 2024). Verbal informed consent was obtained from all participants before enrolment.

Consent for Publication

None.

Availability of Data and Material

The datasets are available from the corresponding author upon reasonable request.

Competing Interests

The authors declare that there is no conflict of interest.

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Use of Generative Artificial Intelligence

The authors declare that no generative AI tools were used in the preparation, writing, or editing of this manuscript.

Authors' Contributions

All authors contributed equally to the design and conception of the study. Baydaa Ahmed Abed and Hussein Hatam Omran experimented. Noor Ulhuda G. Mohammed analyzed the data. Hind S. Sedeeq and Ahmed R. Olewi wrote the manuscript, and Layla Othman Farhan helped supervise the project and final revision; all authors reviewed the manuscript and approved the final manuscript.

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