Cutis verticis gyrata in a patient with acromegaly: an unusual case and review of literature

Ossama Achach[1] MD, Imane Assarrar[1] MD, Mohamed Aymane Loukili[1] MD, Siham Rouf[1,2] MD, Hanane Latrech[1,2] MD

Affiliations: [1]Department of Endocrinology-Diabetology and Nutrition, Mohammed VI University Hospital Center, Faculty of Medicine and Pharmacy, University of Mohammed First, Oujda, Morocco, [2]Laboratory of Epidemiology, Clinical Research and Public Health, Mohammed VI University Hospital Center, Faculty of Medicine and Pharmacy, University of Mohammed First, Oujda, Morocco

Corresponding Author: Hanane Latrech, Mohammed VI University Hospital Center, 4806, 60049 Oujda Morocco, Tel: 212-670-073553, Email: hlatrech@hotmail.fr

Abstract

Acromegaly is a rare systemic syndrome induced by the overproduction of growth hormone (GH) and insulin-like growth factor type one (IGF1). It is responsible for changes in the skeletal and soft tissue systems and it almost always occurs because of a pituitary adenoma. Amongst the skin complications related to acromegaly, cutis verticis gyrate (CVG) is occasionally found. It is a skin condition characterized by excessive growth of the skin of the scalp, resulting in furrows and folds. Only a few cases of this uncommon association have been reported in the literature. The present clinical case illustrates typical CVG associated with acromegaly. Imaging revealed a pituitary macroadenoma lesion and hormonal evaluation revealed elevated IGF1 and hypopituitarism. The patient underwent a transsphenoidal resection of the pituitary adenoma and the histopathological examination confirmed the diagnosis. The diagnosis of CVG is clinical, so radiologic assessments are generally not necessary. The management of acromegaly associated with CVG depends on controlling the serum levels of GH and IGF1. In some cases, specific injections or surgery can be used to minimize CVG.

Keywords: acromegaly, adenoma, cutis, growth hormone, pituitary, rare, verticis gyrate

Introduction

Acromegaly is a rare endocrine disorder caused by hypersecretion of growth hormone (GH), leading to an excessive level of IGF1. Often, this condition is caused by a pituitary adenoma. The real prevalence of acromegaly is hard to define as it ranges from 2.8 to 13.7 cases per 100,000 population, whereas the annual incidence goes from 0.2 to 1.1 cases per 100,000 person-years [1-3]. Acromegaly is classically associated with acral enlargement, prominent facial features, and overgrowth of other soft tissues. Cutis verticis gyrata is a medical condition of rare occurrence featuring scalp and forehead skin tissue thickening, producing furrows and folds similar to the gyri of the cerebral cortex. It was first described by Alibert in 1837 under the term *cutaneous furrow* [4]. Robert provided the first clinical description of this condition in 1843, and in 1907 Unna gave it the name CVG, which has been generally accepted ever since [5,6]. It is more frequent in men, with an estimated prevalence of one per 100,000 in males and 0.026 per 100,000 in females [7]. Cutis verticis gyrata can be either primary or secondary. Cutis verticis gyrata is considered primary when the etiology is unknown or has a neurological cause. Secondary CVG is the most common presentation form and has been associated with conditions such as pachydermoperiostosis, eczema, psoriasis, amyloidosis, myxedema, insulin-resistance syndrome, and acromegaly [8,9]. Its association with acromegaly is exceptional as only 13 cases have been ever reported in the literature, to the best of our knowledge.

In this paper, we report a 34-year-old patient who presented with the unusual association of

acromegaly and cutis verticis gyrata. This case is reported in line with the SCARE criteria [10].

Case Synopsis

A 34-year-old man was referred to our endocrinology-diabetology and nutrition department for suspected acromegaly because of clinical acro-facial dysmorphic syndrome. He exhibited enlargement of the feet and hands and had a height of 186cm and a body mass index of 26kg/m^2. The patient showed an elongated head, protrusion of the superciliary arches, thickened lips, enlarged nose, tooth separation, slight gingival hypertrophy, prognathism, macroglossia, and marked wrinkles of the face (**Figure 1**). Besides, the patient noticed an increasing shoe size from 44 to 46 by the age of 31 years. He also reported that his hands grew bigger, his voice hoarser, and his scalp thickened progressively during the prior three years. Scalp examination showed increased skin folds, furrows, and convolutions in the parietal and occipital regions (**Figure 2**). The patient reported having difficulty cutting his hair owing to the irregularities on the scalp during the last few years. The skinfolds were about 1cm deep and were not easily flattened by traction or pressure. The majority of the folds were rough, symmetrical, and sponge-like. Moreover, the patient was asthenic and admitted to erectile dysfunction and decreased libido. The patient reported having a history of uncontrolled diabetes mellitus that was diagnosed at the age of 32 years and treated with metformin initially, then multiple insulin injection therapies. The patient suffered from nocturnal snoring, hepatosplenomegaly, and diffuse mechanical arthralgias.

The diagnosis of acromegaly was confirmed by an elevated IGF1 at 550ng/ml (normal 82-241ng/ml, by chemiluminescent microparticle immunoassay). Other pituitary-related hormonal assessments revealed central hypothyroidism with a level of thyroid stimulating hormone of 0.48mIU/l (normal 0.27-4.2mIU/l, by chemiluminescent microparticle immunoassay), and free T4 at 11.58pmol/l (normal 12-22pmol/l), treated with levothyroxine therapy at a dose of 75µg per day. Moreover, the patient had biological hypogonadotropic hypogonadism with low testosterone level at 0.55ng/ml (normal 0.47-9.80ng/ml, by chemiluminescent microparticle immunoassay) and a level of luteinizing hormone at 1.56mIU/ml (normal 1.2-10mIU/ml, by chemiluminescent microparticle immunoassay) and follicle

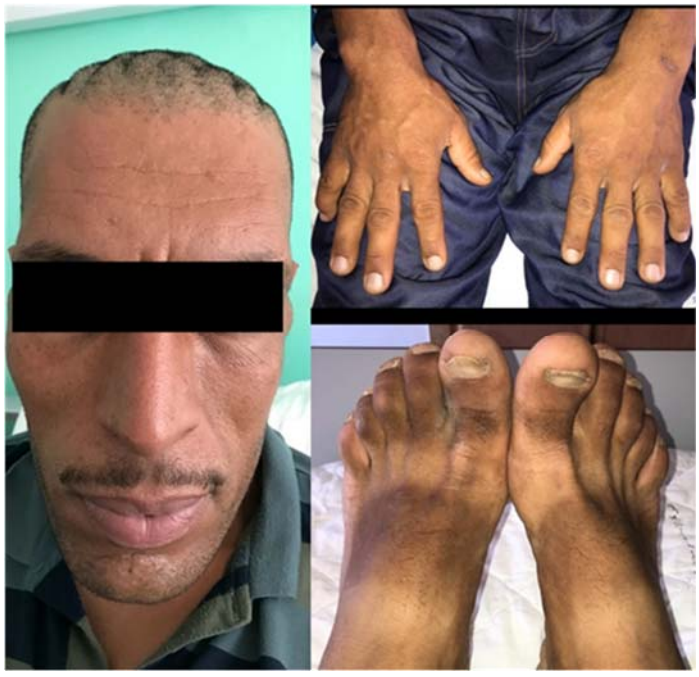

Figure 1. *Clinical features of acromegaly. Facial manifestation of acromegaly. Enlargement of hands in a patient with acromegaly and cutis verticis gyrate, and enlargement of feet.*

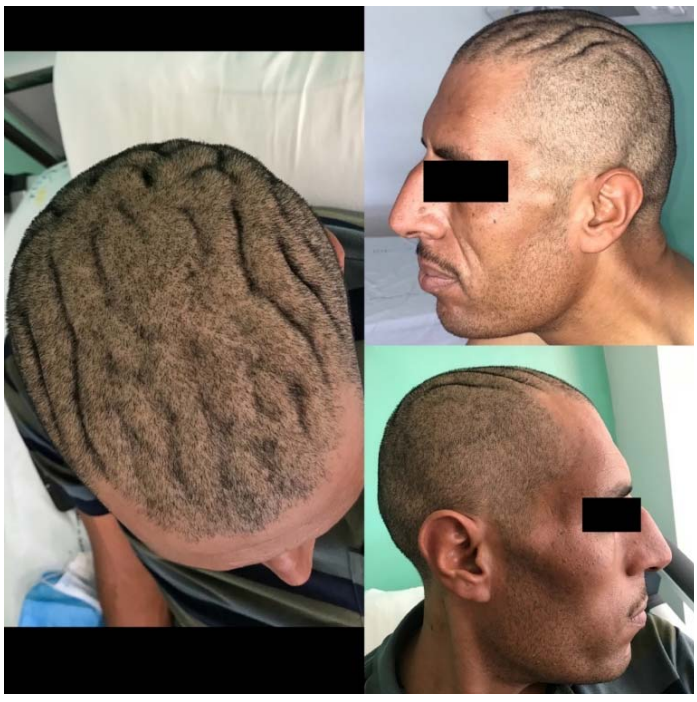

Figure 2. *Cutis verticis gyrata appearance in our patient.*

stimulating hormone at 3.99mIU/ml (normal 1.5-14mIU/ml, by chemiluminescent microparticle immunoassay).

Magnetic resonance imaging antenna for the sellar region was unfortunately incompatible with his head morphology. Therefore, we proceeded with a cerebral computed tomography scan (CT scan). It showed a pituitary macroadenoma lesion measuring 30×27×30mm (height×width×depth). The tumor invaded the homolateral cavernous sinus with an absence of mass effect on the optic chiasma. This lesion was responsible for an enlargement of the pituitary fossa and had lateral extensions on both sides. The mass encircled the intra-cavernous portion of the internal carotid artery) at less than 180 degrees circumference and seemed to reach the optic chiasm. Visual acuity and visual field were not impaired.

Preoperatively, the medical case was discussed over a multidisciplinary meeting and the decision was to perform transsphenoidal surgery. Thus, the patient underwent gross total removal of the tumor. Macroscopically, the tumor was whitish and easily aspirable with suction. The pathological examination of the specimen confirmed the pituitary adenoma. Immunohistochemistry was positive for GH. The Ki67 labeling index was inferior to 1%.

At the 3-month postoperative evaluation, CVG remained unchanged. Unfortunately, the patient didn't show up for his next appointments and was lost to follow-up.

Case Discussion

Acromegaly is a rare disease characterized by excessive GH secretion that stimulates the synthesis of IGFI. In about 75% of cases, excessive GH production is caused by pituitary macroadenomas [11]. The main clinical features are extremities enlargement and soft tissue thickening. The sides of the face are distinctive, with a wide and thick nose, protruding cheekbones, a raised forehead, full lips, and pronounced facial lines. Forehead bumps may develop because the skin on and over the forehead thickens. The patients are also prone to mandibular overgrowth with maxillary enlargement, tooth separation, and jaw misalignment. The disease also has rheumatologic, cardiovascular, respiratory, and metabolic effects that determine prognosis.

The skin expresses IGF1 exclusively in epidermal keratinocytes, sebaceous units, and fibroblasts, whereas GH receptors are shared by all types of cells [7]. The features of acromegaly on the skin and soft tissue are caused by the trophic effects of GH and IGF1, leading to an increased accumulation of glycosaminoglycans, overproduction of connective tissue, and tissue edema [12,15].

Cutis verticis gyrata is an uncommon skin complication of acromegaly. It is an expression of skin and scalp hypertrophy directly caused by elevated levels of GH and IGF1 [15]. In our case, skinfolds were 1cm deep and were not easily flattened by traction or pressure; they followed an anteroposterior direction and involved the vertex and frontoparietal region. Cutis verticis gyrata is an uncommon and benign dermatological lesion that shows thickened, wrinkled skin on the scalp and/or forehead and face, with folds and furrows ranging over a depth of 1cm and a width of 0.5-2cm, similar to gyri and sulci [16,18].

Cutis verticis gyrata is a rare condition with a prevalence of 0.026 to 0.1 per 100,000 individuals and reports of its association with acromegaly are even rarer [19]. In the available literature, there are no well-documented data concerning the prevalence of CVG in acromegaly. However, CVG appears to be an unusual finding in acromegaly and may be one of the rarest cutaneous features reported in such patients with only a few cases reported to date.

Cutis verticis gyrata can be generally classified into two categories: primary (essential and non-essential) or secondary [17-20]. Secondary CVG is more common than primary CVG and is associated with various disorders. Other than acromegaly, CVG is associated with many etiologies including hypothyroidism and insulin resistance, Turner syndrome, Klinefelter syndrome, pachy-periosteal intradermal nevus, chronic inflammation, eczema, atopic dermatitis, paraneoplastic syndromes, leukemia, lymphoma, radiotherapy, and amyloidosis

[17,20-26]. In our patient, based on physical examination and biochemical workup, we eliminated other causes for CVG. The presence of CVG alone does not necessarily imply acromegaly; it can occur as a standalone disorder or be linked to additional underlying etiologies.

For our systematic review of acromegaly associated with cutis verticis gyrata, we searched for the terms "acromegaly" and "cutis verticis" in the PubMed database. A total of 13 cases were published between June 1966 and 2022. We included full texts and abstracts in our review. We extracted and summarized demographic, clinical, imaging, and management data in **Table 1**.

The prevalence of acromegaly-related CVG is not well known. Nevertheless, some series reported different prevalence rates of CVG in acromegaly ranging from 0% up to 29.4% [27-28]. Resende et al. didn't register any cases of CVG in their series [27], whereas Kolawole et al. reported an estimated prevalence of 29.4% in their study [28]. According to our systematic review, CVG is far more common in male patients with acromegaly with a male-female sex ratio of 12 (12 males to one female), (**Table 1**).

As for clinical presentation, unlike primary CVG in which symmetrical furrows are typically observed along the sagittal axis, in acromegaly-related CVG variable patterns affect virtually all regions of the scalp, as well as the skin of the forehead and face [29-32]. Cutis verticis gyrata may be associated with other cutaneous manifestations in patients with acromegaly, including skin spots, acanthosis nigricans, and hidradenitis suppurativa [30-33]. As the diagnosis of CVG is clinical, it is generally not required to undergo radiological examinations [29]. Our patient reported having difficulties cutting his hair for a few years due to furrows, which points to the discovery of cutis verticis gyrata simultaneously with the development of dysmorphic syndrome. The diagnosis of acromegaly was confirmed clinically and biologically with an elevated IGF1 for age and sex and the confirmation of GH-secreting adenoma on immunochemistry. Nevertheless, sometimes CVG may be diagnosed on CT or MRI or even appear as an incidental finding when the clinical diagnosis is not obvious [29,36,37].

The diagnostic delay between the discovery of CVG and the diagnosis of acromegaly can vary depending on several factors. It's crucial to remember that CVG is a pretty uncommon syndrome in and of itself and that it rarely occurs in conjunction with other conditions like acromegaly. However, when CVG is a manifestation of acromegaly, it often develops later in life, usually after the excessive production of GH has already begun. Acromegaly itself typically has a slow and insidious onset with symptoms gradually progressing over time. As a result, the development of CVG-associated acromegaly tends to occur in adulthood.

According to our systematic review, noticeable high IGF1 levels were observed with an average IGF1 of 1799ng/ml. Our patient had the lowest level of IGF1 compared to other reported cases (**Table 1**). In the literature, there is limited specific data available on the levels of IGF1 in patients with both conditions.

It should be noted that in our literature review, all patients for whom information was available had a pituitary macroadenoma (**Table 1**). Also, the presence of a macroadenoma in patients with acromegaly and CVG is highly likely to relate to the severity and duration of the acromegalic symptoms. Macroadenomas are generally associated with a longer duration of symptoms and bring about more significant hormonal disturbances stemming from their larger size and potential compression of surrounding structures.

The management of CVG in patients with acromegaly, like other secondary CVG, depends on controlling acromegaly, maintaining good hygiene, and managing infectious complications as a first-line treatment [7,29,30,34]. Even if the affected region is asymptomatic, odor and itching caused by the accumulation of secretions can compromise further the quality of life for patients. For that reason, good scalp hygiene is essential for symptom relief. Surgery or other treatments can be considered when the results are not satisfactory and depending on the patient's choice [7,30,34]. According to the literature, noticeable improvement or complete regression of acromegaly-associated CVG was observed when GH and IGF1 levels decreased after pituitary surgery or medical therapy with somatostatin analogs,

dopamine agonists, or pegvisomant [31,35-39]. Pinguela et al. reported a significant correlation between lower serum IGF1 levels and lower CVG [40]. Vice versa, high levels of IGF1 may be related to the apparition of CVGs in certain patients with acromegaly as suggested above (**Table 1**). After ensuring the best possible control of acromegaly, preserving hygiene and managing infectious problems, additional management options include surgery, injections of hyaluronidase, and injections of polylactic acid [29].

Some reports in the literature consider the treatment of CVG as being essentially surgical, using expanders or excisions such as a butterfly-shaped scalp excision [7, 44].It remains the best option for patients who experience complications such as maceration or infections, and for those who express major psychological or cosmetic concerns. The surgical approach consists of a scalp reduction surgery. Some authors support the use of tissue expansion before the scalp reduction surgery, whereas others prefer excision and direct closure [48]. Our patient underwent transsphenoidal resection of the pituitary adenoma. At three months follow-up, the CVG remained unchanged but he was lost to follow-up afterward.

Our case describes the rare association of CVG and acromegaly and draws attention to the possibility of this complication. Therefore, clinicians and especially dermatologists should always suspect and rule out the potential diagnosis of acromegaly when patients present with CVG.

Conclusion

Cutis verticis gyrata can be the first clinical manifestation of acromegaly. This skin manifestation is uncommon but its prompt identification may allow an early diagnosis of acromegaly. Healthcare providers must maintain a high index of suspicion for acromegaly in individuals with CVG or other relevant clinical features and conduct appropriate evaluations to ensure timely diagnosis and management.

Potential conflicts of interest

The authors declare no conflicts of interest.

References

1. Lavrentaki A, Paluzzi A, Wass JAH, Karavitaki N. Epidemiology of acromegaly: review of population studies. *J Pituitary*. 2017;20:4–9. [PMID: 27743174].
2. Caputo M, Ucciero A, Mele C, et al. Use of administrative health databases to estimate incidence and prevalence of acromegaly in Piedmont Region, Italy. *J Endocrinol Invest*. 2019;42:397–402. [PMID: 30069856].
3. Crisafulli S, Luxi N, Sultana J, et al. Global epidemiology of acromegaly: a systematic review and meta-analysis. *Eur J Endocrinol*. 2021;185:251–63. [PMID: 34061771].
4. Jean Louis Alibert. Monographie der Dermatosen:Baron Alibert's Vorlesungen über die Krankheiten der Haut. vol. 1. kollmann. 1837.
5. Robert A. Journal de Chirurgie par Malgaigne, Paris, I:125, 1843. J Chir Paris. 1843;1.
6. Unna P. Cutis verticis gyrata. Monatsh Prakt Derm ;1907. p. 227-233.
7. Schenato LK, Gil T, Carvalho LA, et al. [Essential primary cutis verticis gyrata]. *J Pediatr (Rio J)*. 2002;78:75–80. [PMID: 14647817].
8. Alderson HE. CUTIS VERTICIS GYRATA FOLLOWING TRAUMA AND INFECTION. *Arch Dermatol*. 1932;26:1020. [DOI: 10.1001/archderm.1932.01450031022007].
9. Diven DG, Tanus T, Raimer SS. Cutis verticis gyrata. *Int J Dermatol*. 1991;30:710–2. [PMID: 1955225].
10. Agha RA, Franchi T, Sohrabi C, et al. The SCARE 2020 Guideline: Updating Consensus Surgical CAse REport (SCARE) Guidelines. *Int J Surg Lond Engl*. 2020;84:226–30. [PMID: 33181358].
11. Maione L, Chanson P. National acromegaly registries. *Best Pract Res Clin Endocrinol Metab*. 2019;33:101264. [PMID: 30894298].
12. Vilar L, Vilar CF, Lyra R, Lyra R, Naves LA. Acromegaly: clinical features at diagnosis. *J Pituitary*. 2017;20:22–32. [PMID: 27812777].
13. Katznelson L, Laws ER, Melmed S, et al. Acromegaly: an endocrine society clinical practice guideline. *J Clin Endocrinol Metab*. 2014;99:3933–51. [PMID: 25356808].
14. Ben-Shlomo A, Melmed S. Skin manifestations in acromegaly. *Clin Dermatol*. 2006;24:256–9. [PMID: 16828406].
15. Kanaka-Gantenbein C, Kogia C, Abdel-Naser MB, Chrousos GP. Skin manifestations of growth hormone-induced diseases. *Rev Endocr Metab Disord*. 2016;17:259–67. [PMID: 27571787].
16. Harish V, Clarke F. Isolated cutis verticis gyrata of the glabella and nasal bridge: a case report and review of the literature. *J Plast Reconstr Aesthetic Surg JPRAS*. 2013;66:1421–3. [PMID: 23434500].
17. Chentli F, Terki B, Azzoug S. Cutis verticis gyrata and acromegaly. *Presse Medicale Paris Fr 1983*. 2015;44:958–61. [PMID: 26049904].
18. Escobar K, Díaz JM. Primary Variant of Cutis Verticis Gyrata: Report of Two Cases and Review of the Literature. *J Dermatol Cos Méd Quir*. 2019;17.
19. Akesson HO. CUTIS VERTICIS GYRATA AND MENTAL DEFICIENCY IN SWEDEN. II. GENETIC ASPECTS. *Acta Med Scand*. 1965;177:459–

64. [PMID: 14296606].
20. Larsen F, Birchall N. Cutis verticis gyrata: three cases with different aetiologies that demonstrate the classification system. *Australas J Dermatol*. 2007;48:91–4. [PMID: 17535195].
21. Tucci A, Pezzani L, Scuvera G, et al. Is cutis verticis Gyrata-Intellectual Disability syndrome an underdiagnosed condition? A case report and review of 62 cases. *Am J Med Genet A*. 2017;173:638–46. [PMID: 28019079].
22. Orkin M, Frichot BC, Zelickson AS. Cerebriform intradermal nevus. A cause of cutis verticis gyrata. *Arch Dermatol*. 1974;110:575–82. [PMID: 4412371].
23. Wyatt EH. Cutis verticis gyrata. *Clin Exp Dermatol*. 1987;12:293. [PMID: 3427816].
24. Fustà-Novell X, Riquelme-Mc Loughlin C, Morgado-Carrasco D, García-Herrera A, Mascaró JM. Cutis verticis gyrata secondary to acute myelogenous leukaemia. *Clin Exp Dermatol*. 2020;45:342–4. [PMID: 31437867].
25. Walshauser MA, Birch NW, Michaelis LC. Cutis verticis gyrata. *Am J Med Sci*. 2014;348:176. [PMID: 23328837].
26. Sahu P, Dayal S, Gera G, Amrani A. Cutis verticis gyrata - A rare presentation of Primary systemic Amyloidosis. *Int J Trichology*. 2018;10:141–3. [PMID: 30034198].
27. Resende M, Bolfi F, Nunes V dos S, Miot HA. Prevalence of dermatologic disorders in 15 patients with acromegaly. *An Bras Dermatol*. 2012;87:166–8. [PMID: 22481676].
28. Kolawole TM, Al Orainy IA, Patel PJ, Fathuddin S. Cutis verticis gyrata: its computed tomographic demonstration in acromegaly. *Eur J Radiol*. 1998;27:145–8. [PMID: 9639140].
29. Chentli F, Terki B, Azzoug S. Cutis verticis gyrata and acromegaly. *Presse Medicale Paris Fr 1983*. 2015;44:958–61. [PMID: 26049904].
30. Walia R, Bhansali A. Cutis verticis gyrata. *BMJ Case Rep*. 2011;2011. [PMID: 22693312].
31. Llamas-Velasco M, Pérez-Gónzalez YC, Moya-Chimenti E. Spontaneous improvement of cutis verticis gyrata secondary to acromegaly following surgical treatment of pituitary adenoma. *J Dtsch Dermatol Ges J Ger Soc Dermatol JDDG*. 2018;16:895–6. [PMID: 29927514].
32. Abu-Jamra F, Dimick DF. Cutis verticis gyrata. A report of a case secondary to acromegaly treated with plastic surgery and x-ray therapy. *Am J Surg*. 1966;111:274–7. [PMID: 5903705].
33. Degirmentepe EN, Gungor S, Kocaturk E, et al. Dermatologic manifestations of acromegaly: A case in point and a focused review. *Dermatol Online J*. 2017;23. [PMID: 29469738].
34. Garden JM, Robinson JK. Essential primary cutis verticis gyrata. Treatment with the scalp reduction procedure. *Arch Dermatol*. 1984;120:1480–3. [PMID: 6497415].
35. Lundberg PO, Wålinder J. Cutis verticis gyrata and neuroendocrine diseases. *Acta Neurol Scand*. 1971;47:369–80. [PMID: 4106417].
36. Schunter JA, Metze D, Böhm M. Is IGF-1 a key player in the pathogenesis of acromegaly-associated cutis verticis gyrata? *Exp Dermatol*. 2017;26:85–6. [PMID: 27488099].
37. Giustina A, Arnaldi G, Bogazzi F, et al. Pegvisomant in acromegaly: an update. *J Endocrinol Invest*. 2017;40:577–89. [PMID: 28176221].
38. Aziz W, Al Felasi M, Najm P, Aldahmani KM. Significant Cutaneous Improvement within Two Months of Surgery. *Dubai Diabetes Endocrinol J*. 2020;26:180–2. [DOI: 10.1159/000511693].
39. Nabarro JD. Acromegaly. *Clin Endocrinol (Oxf)*. 1987;26:481–512. [PMID: 3308190].
40. Pinggera D, Marsoner D, Seiz-Rosenhagen M, Thomé C, Franz Freyschlag C. Reduction in Cutis Verticis Gyrata after Transsphenoidal Pituitary Resection of Growth Hormone–Secreting Adenoma. *Indian J Neurosurg*. 2016;05:084–8. [DOI: 10.1055/s-0036-1584591].
41. Abu-Jamra F, Dimick DF. Cutis verticis gyrata. A report of a case secondary to acromegaly treated with plastic surgery and x-ray therapy. *Am J Surg*. 1966;111:274–7. [PMID: 5903705].
42. O'Reilly FM, Sliney I, O'Loughlin S. Acromegaly and cutis verticis gyrata. *J R Soc Med*. 1997;90:79. [PMID: 9068435].
43. Al-Bedaia M, Al-Khenaizan AS. Acromegaly presenting as cutis verticis gyrata. *Int J Dermatol*. 2008;47:164. [PMID: 18211488].
44. Pura M, Vanuga P. A peculiar affection of the scalp. *J Clin Endocrinol Metab*. 2010;95:3091. [PMID: 20610603].
45. Araujo KM, da Silva FT, Bestane GC, Soares MM, Denadai R. Delayed Diagnosis of Acromegaly in an Elderly Adult that Initially Presented as Cutis Verticis Gyrata. *J Am Geriatr Soc*. 2016;64:685–7. [PMID: 27000365].
46. Yerawar C, Bandgar T, Lila A, Shah NS. Acromegaly presenting as cutis verticis gyrata. *QJM Mon J Assoc Physicians*. 2016;109:423. [PMID: 26873450].
47. Degirmentepe EN, Gungor S, Kocaturk E, et al. Dermatologic manifestations of acromegaly: A case in point and a focused review. *Dermatol Online J*. 2017;23. [PMID: 29469738].
48. Parolin M, Dassie F. Cutis Verticis Gyrata Associated with Acromegaly. *N Engl J Med*. 2019;380:e31. [PMID: 31042829].
49. Roque J, Marques P. Clinico-radiological features of acromegaly-related cutis verticis gyrata. *Horm Athens Greece*. 2022;21:519–20. [PMID: 35435624].
50. Landenberger GMC, Ongaratti BR, Pereira-Lima JFS, Oliveira M da C. Cutis verticis gyrata: a cutaneous finding in acromegaly. *An Bras Dermatol*. 2022;97:369–71. [PMID: 35288001].

Table 1. *Clinical and biochemical features of patients with acromegaly associated with cutis verticis gyrata available in the literature.*

Case No	Author (year)	City/ country	Gender	Age at diagnosis of acromegaly (year)	Fasting morning serum GH (NV)	IGF1 (NV)	Pituitary adenoma type	CVG description	Other skin features	Treatment
1	Abu-Jamra & Dimick (1966), [41]	Chicago, USA	M	49	0.72µg percent (NV:0.1-0.5µg percent)	NA	NA	Sagittal folds and furrows across the forehead	Redundant skin of the face, marked varicosities of the abdomen	Plastic reconstruction of the scalp; X-ray therapy to the pituitary
2	O'Reilly et al. (1997), [42]	Dublin, Ireland	M	60	NA	NA	NA	Horizontal folds on the forehead	Basal cell carcinoma	Pituitary surgery
3	Al-Bedaia & Al Khenaizan (2008), [43]	Riyadh, Saudi Arabia	M	17	37.3mIU/l (NV:0.2-13.0mIU/ml)	NA	NA	Multiple gyriform oblong skin folds on the fronto-parietal scalp	Acanthosis nigricans	NA
4	Pura & Vanuga (2010), [44]	Ľubochňa, Slovakia	M	25	NA	NA	Macroadenoma	Multiple folds and furrows in the parietal region, with anterior to posterior direction None	None	NA
5	Walia & Bhansali (2011), [30]	Chandigarh, India	M	30	NA	1126ng/ml (NV=117–329ng/ml)	Macroadenoma	NA	Acanthosis nigricans, skin tags	NA
6	Chentli et al. (2015), [29]	Ivry-sur-Seine, France	M	47	40mIU/ml (NV <3mIU/ml)	1279ng/ml (NV=175–375ng/ml)	Macroadenoma	Thick horizontal and vertical folds on the forehead and anterior scalp	None	Dopamine analogs; Somatostatin analogs; pituitary surgery
7	Araujo et al. (2016), [45]	Sao Paulo, Brazil	F	78	43ng/ml	805ng/ml	Macroadenoma	Multiple folds and furrows on	None	Dopamine analogs and

								the forehead and frontal scalp		somatostatin analogs
8	Yerawar et al. (2016), [46]	Mumbai, India	M	41	48.1ng/ml (NV:0–30ng/ml)	958ng/ml (NV:101–267ng/ml)	Macroadenoma	Multiple gyriform oblong skin folds on the parieto-occipital scalp	increasing ridges	Pituitary surgery
9	Degirmentepe et al. (2017), [47]	Istanbul, Turkey	M	37	2.65ng/ml (NV:0-5ng/ml)	757ng/ml (NV:109-284ng/ml)	Macroadenoma	Multiple gyriform oblong skin folds in the occipital region	Acanthosis nigricans, hidradenitis suppurativa	Pituitary surgery and somatostatin analogs
10	Llamas-Velasco et al. (2018) [31]	Madrid, Spain	M	36	4.09ng/ml (NV:2ng/ml)	1431ng/ml (NV:360ng/ml)	Macroadenoma	Folds and furrows in the parietal and occipital area	None	Pituitary surgery
11	Parolin & Dassie (2019), [48]	Padua, Italy	M	37	73ng/ml (NV: 0-0.8ng/ml)	9070ng/ml (NV:820-2370ng/ml)	Macroadenoma	Multidirectional ridges and furrows on the forehead, parietal and occipital scalp	None	Pituitary surgery; somatostatin analogs; GH receptor antagonist
12	Roque & Marques (2022), [49]	Athens, Greece	M	41	NA	1280ng/ml	NA	CVG of occipital scalp. Frontal bossing with skin wrinkles.	None	None
13	Landenberger et al. (2022), [50]	Porto Alegre, Brazil	M	60	21.5ng/ml (NV:<3ng/ml)	734ng/ml (NV:81-225ng/ml)	Macroadenoma	Convolutions of the scalp in the parietal and occipital regions	increased skin folds and furrows, increased skin sweating	Pituitary surgery; Somatostatin analogs
14	Our case	Oujda, Morocco	M	34	203ng/ml (NV: < 2.47ng/ml)	550ng/ml (NV:82-241ng/ml)	Macroadenoma	Convolutions of the scalp in the parietal and occipital regions	None	Pituitary surgery

CVG, cutis verticis gyrata; F, female; GH, growth hormone; IGF1, insulin-like growth factor 1; M, male; NA, not available; NV, normal value.