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# **Anterior Cervical Hypertrichosis: A Case Report**

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#### **Abstract**

**Observation:** Anterior cervical hypertrichosis is a dermatosis of unknown cause, seen on the front of the neck, which is characterised by an increase in terminal hairs. It is generally sporadic and may be accompanied byanomalies such as peripheral and central neuropathy, mental retardation, retarded development, hallux valgus, ocular anomalies (optic atrophy, chorioretinal changes, hypermetropia), kyphoscoliosis, spina bifida, facial dysmorphism, conjoined muscles (synophyrys), lumbosacral or dorsal hypertrichosis and Turner syndrome. In this paper, the case is presented of an 11-year old male patient diagnosed with sporadic anterior cervical hypertrichosis.

## Introduction

The terms of hypertrichosis and hirsutism are often confused. However, these terms are used to describe different situations. Hypertrichosis, the cause of which is generally unknown, is an increase in hair which occurs independently of androgeny. There are no symptoms of oligomenorrhea, amenorrhea or virilisation (male-type hair on the temples, shrinkage of breast tissue, muscle tissue definition, clitoris growth and voice deepening). Hirsutism is hairiness which occurs with excessive androgeny. There is an increase in thick, dark hairs in body areas associated with androgen hormone, such as the upper lip, chin, around the breast, the lower abdomen, the groin and thighs [1].

Anterior cervical hypertrichosis (ACH) is a rarely seen congenital localised hypertrichosis,

which was first described in 1991 by *Trattner* et al. It is characterised by terminal hair on the anterior side of the neck [1, 2]. While it is often seen in the form of hypertrichosis alone, various anomalies may accompany ACH. In this paper, the case is presented of an 11-year old male patient diagnosed with sporadic ACH.



**Figure 1.** It is characterised by terminal hair on the anterior side of the neck

# **Case Report**

An 11-year old male presented at our polyclinic with the complaint of hair on the anterior side of the neck which had been ongoing for 1 year. There was no anamnesis of any use of topical or systemic medication, trauma or inflammation before the onset of the hairiness. No family member had any similar complaint.

In the dermatological examination, there were seen to be long, brown terminal hairs in an area of approximately 5 x 6 cm diameter (**Figure 1**). No other dermatological findings were determined. The systemic examination and laboratory test results were normal. No pathology findings were determined in the evaluations made by the neurology and ophthalmolofy specialists. Treatment of laser epilation was planned for the future because of the patient's age.

## **Discussion**

Hypertrichosis is classified in different ways [3]. According to the etiology it may be idiopathic or acquired; according to the age at onset, it may be congenital or acquired; according to the area involved, it may be claslocalised generalised as or hypertrichosis [3, 4]. Localised congenital hypertrichosis is characterised by well-defined excessive hair growth in any area of the body. It often has autosomal recessive transfer. There are 4 sub-types as lumbosacral hypertrichosis, hypertrichosis cubiti, posterior cervical hypertrichosis and ACH **[5**].

Lumbosacral hypertrichosis (faun tail) is the most frequently seen form of localised congenital hypertrichosis. Increased hair growth in the sacral mid region is determined at birth. It is often seen together with anomalies such as hyperpigmentation, sacral dimple, spinal dysraphism, lipoma, hamartoma, port-wine stain, dermatoid cyst, sinus tract and aplasia cutis [5].

Hypertrichosis cubiti (hairy elbow syndrome), which occurs at birth or in infancy, is hypertrichosis seen on the extensor surfaces of the wrists. This sub-type of localised congenital hypertrichosis is bilateral and seen together with anomalies such as short height or facial asymmetry. Posterior cervical hypertrichosis is associated with autosomal dominant or X recessive transfer.

It is characterised by excessive hair on the posterior cervical vertebrae. Occasionally, it may be determined together with kyphoscoliosis [5].

ACH is hair seen in the area of the laryngeal prominence in particular. Often, no underlying reason can be determined. Despite autosomal dominant transference, autosomal recessive [6] and X dominant [7] forms have also been described. The vast majority of reported patients are familial [1]. Just as ACH may be congenital, it is generally determined as sporadic, as in the current case.

Although not previously reported in Turkey, there have been 41 ACH patients reported in literature. The reported cases are generally female and determined at birth or in early childhood (n=31). In the majority of cases there was no other finding (n=32,78%). In 9 patients (22%), there were various other anomalies. Peripheral and central neuropathy was determined in 5 patients, mental retardation in 2, and retarded development in 2 [8]. Other anomalies determined in ACH are hallux valgus, ocular anomalies (optic atrophy, chorioretinal changes, hypermetropia), kyphoscoliosis, spina bifida, facial dysmorphism, conjoined muscles (synophyrys), lumbosacral or dorsal hypertrichosis and Turner syndrome [1,

The pathogenesis of ACH has not been proven and no genetic cause has been determined. In recent studies, the hair follicles of ACH patients have been determined to show significant plasticity. Focus has been directed on an extended anagen phase or excessive growth of the hair follicles leading to an abnormal signal pathway in ACH disease [9]. Treatments may include trimming, waxing, bleaching, physical and chemical depilation, electrolysis, intense pulsed light (IPL) and laser [1, 10].

ACH causes both social and psychological problems for the patient. Although a benign disease, ACH may sometimes accompany neurological, ocular and orthopaedic anomalies. Therefore, in all patients diagnosed with ACH a thorough clinical examination (skin, neurology and ophthalmology examinations) and radiological evaluations must be made.

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