

Acromegaly presented as a cause of laryngeal dyspnea[☆]

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Abstract

Acromegalic patients can develop obstructive sleep apnea syndrome or upper airflow obstruction. The development of dyspnea is unusual and the fixation of both vocal cords is exceptional. We report the case of a patient with bilateral vocal cord paralysis. Fiberoptic laryngoscopy and computed tomography (CT) of the neck showed a supra-glottic stenosis due to a swelling of the soft tissue. A tracheostomy was first performed. Thereafter, micro-laryngoscopy using laser vaporisation of the supra-glottic soft tissue was attempted but failed to remove the tracheostomy canula. Finally, blood tests and cerebral MRI revealed an acromegaly. The patient underwent a *trans*-sphenoidal resection of the pituitary adenoma. Fifteen months later, fiberoptic laryngoscopy showed bilateral restoration of vocal cord mobility and the tracheostomy canula was successfully removed after 18 months.

Vocal cord fixation is probably due to hypopharyngeal and laryngeal soft tissue swelling and can be reversible after successful treatment of the adenoma.

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1. Introduction

Acromegaly is a rare disease with an incidence of about 3–4 new cases/million/year [1]. This pituitary disorder, caused by excess growth hormones (GH), generates insidious soft-tissues swelling and enlargement of extremities, taking years to decades to become apparent. The treatment usually consists of surgery as the first line, sometimes followed by medical therapy for residual disease (Somatostatin, Bromocriptine). Radiation treatment is generally reserved for refractory cases. Death from respiratory causes is three times more likely in an acromegalic than in a normal subject [2]. A narrowing of the upper airways is seen in more than 50% of acromegalic patients. The obstruction of the upper airways may be due to a thickening and a hypertrophy of the soft tissues of the tongue, pharynx and larynx [3]. In exceptional cases, the dyspnea necessitates a tracheotomy. Several authors

describe cases with impaired mobility of the vocal cords. Herein, we report the case of an acromegalic patient with bilateral vocal cord fixation and review the pathogenesis and treatment of this uncommon affliction.

2. Case report

A 51-year-old man with a 2-week history of progressive shortness of breath reported to a regional hospital. On admission, he had severe respiratory distress. Fiberoptic laryngoscopy revealed fixation of both vocal cords in adduction and a tracheostomy was performed. After 10 days, the patient was referred to our center for treatment and check-up. The patient had no previous medical history. A fiberoptic laryngoscopy was performed again and confirmed the fixation of both vocal cords associated with supra-glottic soft tissue swelling. The clinical examination did not reveal any particular abnormality other than hypertrophy of the hard palate mucosa (Fig. 1). At that time, no significance was accorded to this finding, nor to the enlarged feet and fingers or to the coarse face with prominent supra-orbital ridges, thick

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Fig. 1. Hard palate mucosa hypertrophy.

lips and enlarged nose. The CT scan of the neck confirmed the supra-glottic soft tissue hypertrophy (Fig. 2) and a multinodular goiter. A micro-laryngoscopy under general anesthesia was performed. The planned arytenoidectomy could not be performed because the arytenoid could not be exposed correctly due to the soft tissue hypertrophy. A laser vaporisation of the supra-glottic mucosa was then performed but the tracheostomy canula could not be removed at that point.

Later, the oral, pharyngeal and laryngeal soft tissue hypertrophy associated with the physical features of the patient led us to suspect acromegaly. The serum GH level was 23.0 ng/ml (normal range below 8 ng/ml). The MRI of the sella turcica revealed a pituitary adenoma of 17 mm. This patient also had hyperthyroidism with TSH: 0.05 mcU/ml (0.3–4), Free T4: 21 ng/ml (8–20) and Free T3: 5.2 pg/ml (2.1–4). The thyroid scintigraphy revealed a toxic nodule on the left lobe. The patient was first treated with Thiamazol. When normothyroidy was achieved, a *trans*-nasal endoscopic

resection of the adenoma was performed. The day after the surgery, the serum GH level was within normal limits.

Fifteen months after the operation, standard fiberoptic laryngoscopy showed a moderate supra-glottic tissue hypertrophy, the vocal cords could not be seen, but a movement of the tissues was present. A fiberoptic laryngoscopy was then performed through the tracheostomy and showed the bilateral restoration of vocal cord mobility. Eighteen months after the surgery, it was possible to remove the tracheostomy canula definitively.

3. Discussion

We report a rare case of acromegaly diagnosed in a patient who presented dyspnea. The initial symptoms included bilateral vocal cord paralysis associated with pharyngeal and laryngeal soft tissue hypertrophy. When the patient came to us for treatment, we focused on finding the origin of this dyspnea and did not notice his particular physical features. As the vocal cord paralysis remained unexplained, an arytenoidectomy under micro-laryngoscopy was planned. The soft palate mucosa hypertrophy and the pharyngeal and laryngeal soft tissue swelling led us to suspect an acromegaly. We would like to emphasize that if the acromegaly had been diagnosed earlier, the micro-laryngoscopy would not have been performed because it was not the appropriate treatment.

The cause of vocal cord paralysis is a topic of interest in the literature because of the unusual character of the condition. Grotting and Pemberton [4] described four cases that arose before 1950. They proposed four possible explanations for vocal cord fixation: (1) arthritis of the crico-arytenoid joint, (2) irregular enlargement of the cartilage that stretches the vocal cords, leading to impaired mobility of the crico-arytenoid joints, (3) stretching of the recurrent nerves by progressive enlargement of the larynx and (4) injury of recurrent nerves caused by the enlargement of the thyroid gland. Since 1950, 15 cases of dyspnea in acromegalic patients have been described, most necessitating a tracheostomy. Four cases had bilateral vocal cord fixation [1,2,5,6]. The diagnosis of acromegaly had been known for several years but the established treatment had either failed or had not even been attempted. The four patients underwent a tracheostomy and in two of them, it was possible to remove the tracheostomy canula after the treatment of the acromegaly. These authors did not really explain the cause of the vocal cord fixation, but Rees et al. [7] and Bhatia et al. [8] suggest that the diminished movements of the vocal cords are probably related to the thickening of laryngeal structures. We agree with this explanation and think that pharyngeal and laryngeal soft tissue hypertrophy cause the decreased mobility of the larynx through a mass effect. Indeed, Trotman-Dickenson et al. [9] demonstrated in a large series of acromegalic patients that an upper airflow obstruction was present in 50% of them and was associated with the activity of the disease. Morewood et al. [3]

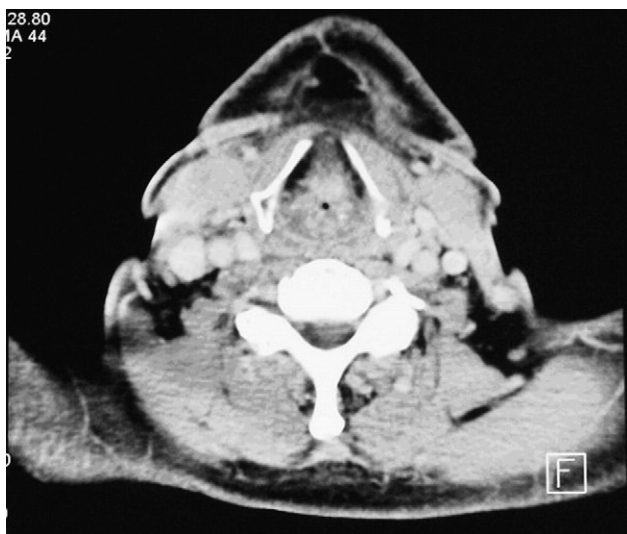


Fig. 2. CT-scanner of the neck before the arytenoidectomy showing a major pharyngeal and laryngeal soft tissue swelling that blocks the mobility of the glottis through a mass effect.

described a more significant thickness of the true and false vocal cords in acromegalic patients. These pharyngeal and laryngeal features often caused obstructive sleep apnea syndrome (OSAS) in those patients. The treatment of the acromegaly decreased the incidence of apnea [10,11] and reduced the hypopharyngeal and laryngeal soft tissue swelling [12]. Therefore, the reversibility of the vocal cord fixation makes us think that its origin is a mass effect caused by pharyngeal and laryngeal soft tissue swelling.

4. Conclusion

Vocal cord fixation in acromegalic patients remains extremely rare. Its origin is probably a major pharyngeal and laryngeal soft tissue swelling that blocks the mobility of the glottis through a mass effect.

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