Severe laryngitis following chronic anabolic steroid abuse

S Ray, A Masood, J Pickles, I Moumoulidis

Abstract
The effects of anabolic steroids on the quality of voice have been well documented; however, no study has established significant structural changes in the larynx as a direct result of anabolic steroid use. We report a unique case of a 47-year-old male smoker and professional body builder who presented with progressive stridor and hoarseness following abuse of anabolic steroids over a period of two years. Conservative management failed to resolve his symptoms and a planned tracheostomy was performed to secure his airway. Subsequently he was treated with multiple laser resections and eventually decannulated. No case of severe laryngitis in association with anabolic steroid usage has been reported previously in the literature.

Key words: Laryngitis; Steroids; Anabolic Agents; Tracheostomy

Introduction
Anabolic steroids have been used for medical conditions such as fibrocystic breast disease, endometriosis, menorrhagia and osteoporosis in post-menopausal women. They are also available without medical prescription to athletes and people weight training. The common side effects are weight gain, hirsutism, acne, receding hairline, headache, increased libido and change in voice. There have been several reports in the literature describing significant changes in vocal physiology, such as lowering of voice pitch, changed timbre and voice instability, some of which are irreversible following discontinuation of treatment. We present a unique case of stridor due to severe laryngitis in a patient who regularly used anabolic steroids as a part of his body building training.

Case report
A 47-year-old male presented to the otolaryngology department as an emergency with increasing hoarseness, dyspnoea, stridor and sore throat. He had been experiencing difficulty in breathing and had noticed a change in his voice for the four to five months prior to his admission. His breathlessness was exacerbated during sleep, which caused him to wake up five to six times a night. He was a professional body builder and admitted abusing anabolic steroids regularly for the past two to three years as a part of his routine training. He mixed and matched the following preparations, Deca Durabolin®, Testoviron® and Sustanon 250® (Table I). He also smoked 20 cigarettes a day for the past 20 years.

On examination he had an audible biphasic stridor, which was exaggerated during sleep. Flexible nasopharyngolaryngoscopy showed features of chronic laryngitis associated with generalised supraglottic and glottic swelling with restriction of the airway. He was admitted and commenced on intravenous dexamethasone 8 mg, cefuroxime 750 mg and metronidazole 500 mg, all three times daily. His symptoms failed to resolve with conservative management, so he underwent a microlaryngoscopy, which showed reddened hyperaemic laryngeal mucosa, markedly congested and swollen supraglottis along with irregularly oedematous vocal folds (Figure 1). Biopsy showed polypoid lesions with hyperkeratotic and hyperplastic epithelium. Subsequently his symptoms worsened and he had an elective tracheostomy. All repeat biopsies showed polypoid lesions covered by hyperkeratotic and hyperplastic epithelium, oedematous fibrous stroma and hyaline changes with inflammatory cell infiltrate. No evidence of malignancy or other infiltrative process was detected and special stains for fungi were also negative. A computerised tomography (CT) scan revealed diffuse swelling of the vocal folds with some obliteration of the para-epiglottic fat and localised swelling of the posterior pharyngeal wall with no significant lymphadenopathy (Figure 2). Further, hypertrophy of the intralaryngeal musculature was evident on the CT scan. Urinalysis as well as haematological tests including cytoplasmic antineutrophil cytoplasmic antibody, erythrocyte...
sedimentation rate, C-reactive protein, antinuclear antibodies test, were all negative. Gastroesophageal acid reflux was considered as a potential co-factor but pH studies were negative. The patient had three procedures to remove hyperplastic vocal fold epithelium with a CO2 laser, which produced an improvement in his voice and laryngeal airway. Microlaryngoscopy carried out after the third procedure showed a significant improvement in hypertrophic laryngitis and no remaining polypoid swelling of the vocal folds. However, the vestibular folds showed a persistent hypertrophy resulting in mild supraglottic obstruction to the airway, not sufficient to prevent decannulation.

Discussion
Chronic laryngitis has been defined as chronic non-specific inflammatory reaction of the laryngeal mucosa. The most important contributing factors are inhaled irritants and, notably, cigarette smoke. The laryngeal changes noted in the present case appear to be a severe hypertrophic laryngitis against a background of hypertrophy of the intralaryngeal musculature. The combination of these two features resulted in a significant narrowing of the glottic airway.

Anabolic steroids have been increasingly used to enhance protein metabolism. There is no safe dose of anabolic steroids with respect to vocal virilisation.2 It has been reported that there is an incidence of voice change in up to 10 per cent of patients on regular anabolic steroids.10 There have been several studies of vocal virilisation in the female larynx following anabolic steroid use for gynaecological problems but there are no reports of any gross structural changes in the larynx in male athletes/body builders on anabolic steroids. Gerritsma et al. suggested that changes are not in the dimensions of the laryngeal framework but there is an increase in muscle production instead of connective tissue. As a result, the rigidity of the fold decreases, thus preventing an increase in tension of the folds necessary for the production of the higher frequencies and thus leading to a lower vocal frequency.2 Baker observed females using anabolic steroids and found significant alterations in vocal physiology including muscle tissue changes, muscle coordination dysfunction and proprioceptive dysfunction. Interestingly, no abnormalities of the larynx were found on flexible laryngoscopy in any of these cases.1

Talaat et al. observed histological changes in the larynx of female mice following prolonged administration of anabolic steroids. Reversible connective tissue changes were in the form of vascular congestion, oedema, perivascular and periductal cellular infiltration.10

<table>
<thead>
<tr>
<th>Anabolic Steroid used</th>
<th>Generic name</th>
<th>Recommended dose</th>
<th>Dose used by patient</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deca-Durabolin</td>
<td>Nandrolone Decanoate 50–100 mg/ml</td>
<td>200–600 mg/week</td>
<td>300 mg/week</td>
</tr>
<tr>
<td>Testoviron</td>
<td>Testosterone Enanthate 50–100 mg/ml</td>
<td>200 mg/week</td>
<td>200 mg/week</td>
</tr>
<tr>
<td>Sustanon 250</td>
<td>Testoviron testarone propionate 50 mg/ml, Testorone phenylpropionate 60 mg/ml, Isocaprate decanoate 60 mg/ml</td>
<td>250–1000 mg/week</td>
<td>300 mg/week</td>
</tr>
</tbody>
</table>
Permanent irreversible changes, in the form of parakeratosis and squamous metaplasia of the epithelium and hypertrophy of the muscle fibres (affecting mainly the inner part of the thyroarytenoid muscle), were noticed along with persistence of increased alkaline phosphatase enzyme intensity in the endothelium of capillaries. Increased vascularity was also noted in the hypertrophied muscles. Vacuoles were seen between the hypertrophied muscle fibres and considered due to fluid retention. These changes were noted after a month of commencing the steroids but became very evident after two to three months. This is consistent with previous studies suggesting irreversibility of laryngeal changes following chronic anabolic steroid usage.

Severe laryngitis causing airway obstruction, as a direct result of anabolic steroid use, has not been previously reported. A case of a male body builder who presented with rapidly progressive stridor and hoarseness following abuse of anabolic steroids is presented. Laryngeal examination showed a markedly congested and swollen supraglottis along with irregular, oedematous vocal folds. Histological examination revealed polypoid lesions with hyperkeratotic and hyperplastic epithelium. Treatment included an emergency tracheostomy and multiple laser resections followed by decannulation.

In our case, there was a combination of generalised swelling of the laryngeal soft tissues and gross polypoid chronic inflammatory reaction of the laryngeal mucosa. We postulate that the chronic use of anabolic steroids results in changes in the intralaryngeal muscles and other soft tissues. The degree and nature of the chronic hypertrophic laryngitis was greater than usually seen as a result of cigarette smoking alone. We feel that it is likely the anabolic steroid use was a co-factor in the development of these severe laryngeal changes. It is noticeable that changes did not reverse spontaneously after the patient had ceased smoking and stopped taking anabolic steroids for 12 months. Surgical treatment was necessary to restore the airway.

We have been unable to find any other papers describing airway impairment due to laryngeal changes associated with chronic anabolic steroid use. The changes noted in the larynx were similar but more florid to those found in experimental animal studies. These changes in the human larynx have not been reported previously. We believe it is likely that the combination of cigarette smoking and anabolic steroid abuse has led to the laryngeal pathology described in this case.

References
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Address for correspondence:
Mr Ioannis Moumoulidis,
36 Moorhouse Way,
Kettering, Northants,
NN15 7LX, UK.

E-mail: moumoulidis@aol.com

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