CASE REPORTS

Primary amyloidosis of the larynx

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Abstract
Primary laryngeal amyloidosis is a rare benign disease of unknown aetiology. It can present with dysphonia or stridor. A woman presenting with airway compromise, who required a tracheostomy, is reported.
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Keywords: laryngeal disease; amyloidosis

Learning points
- Amyloidosis is a rare benign disease of unknown aetiology.
- Laryngeal amyloidosis can present with hoarseness or stridor which may require tracheostomy.
- Histological diagnosis from a biopsy specimen can be confirmed with characteristic staining with Congo red.
- Treatment is by surgical resection using the carbon dioxide laser. Repeated resections may be necessary.

Case report

A 47 year old woman presented with a seven year history of dysphonia which had deteriorated recently. Fibre optic laryngoscopy showed polyps on the right false vocal cord with a suggestion of subglottic stenosis (fig 1). The true vocal cords were mobile and appeared normal.

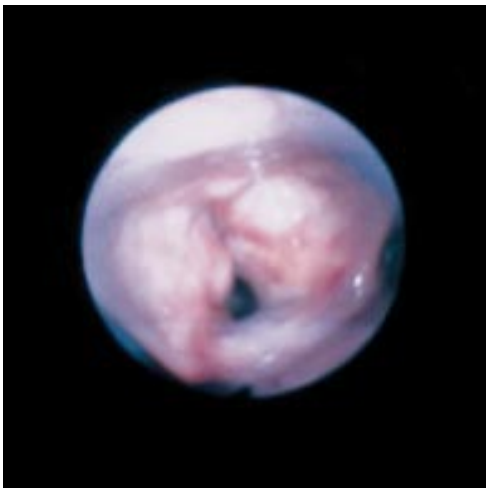

Figure 1 Fibre optic laryngoscopy showing polyps on the right false vocal cord.

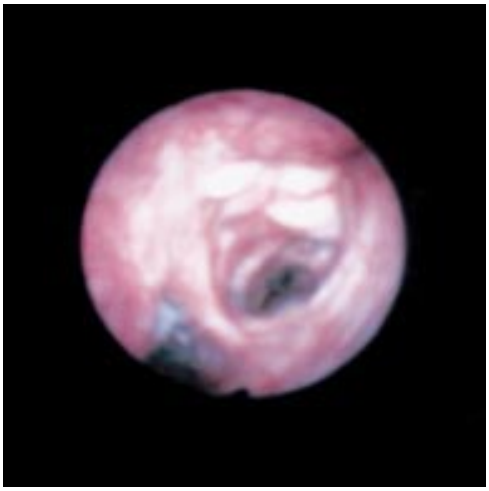

Figure 2 Flexible bronchoscopy showing isolated nodules within the trachea.

While awaiting further investigation, she presented with airway compromise requiring urgent tracheostomy. Direct laryngoscopy revealed subglottic oedema. The polypoid lesion was biopsied and showed features suggestive of amyloidosis and this was confirmed after staining with Congo red. A flexible bronchoscopy through the tracheostomy tube showed isolated nodules within the trachea (fig 2). Full blood count and erythrocyte sedimentation rates were normal.

At follow up a year later she was noted to have a change in voice. Fibre optic laryngoscopy showed new lesions on the right and left true vocal cords. These were resected with a carbon dioxide laser. The result was a subjective improvement in the quality of her voice and airway. She was decannulated two months later. She remains under review and requires no further intervention.

Discussion

Amyloidosis is a benign, slowly progressive condition that is characterised by extracellular fibular proteins. Diagnosis is confirmed by histopathological specimens that stain with Congo red.[1] Amyloidosis can be classified as either primary, developing spontaneously, or secondary to some other longstanding inflammatory disease such as rheumatoid arthritis.[2 3] The primary form can be further subdivided into a localised form, where deposits are confined to a single organ or location, or generalised, where deposits are found to some extent in all tissues.

The most common presenting symptoms of primary laryngeal amyloidosis are dysphonia and stridor. Rarely, airway compromise occurs and an alternative airway may be necessary. The presence of tender bones, lymphadenopathy, or splenomegaly should alert the clinician to the possibility of generalised amyloidosis.[2] Microlaryngoscopy reveals pinkish grey masses lying under the intact epithelium, either as nodular masses or subepithelial deposits.

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Localisation of lesions in the larynx is to the ventricle, false vocal cords, true vocal cords, aryepiglottic folds, and subglottis in that order of frequency.[2 4] Histology of the biopsied specimen using routine haematoxylin and eosin stain shows amyloid as eosinophilic extracellular infiltrate. Further staining with Congo red reveals characteristic apple green birefringence with a polarising microscope confirming the diagnosis.

Treatment of localised laryngeal amyloid deposits is by surgical removal.[5] Several authors have reported good results using the carbon dioxide laser.[6–9] This disease is slowly progressive and repeated removal may be required.[10] The carbon dioxide laser has been shown to be more effective than conventional surgery because it minimises trauma.[9] Regular follow up is important as its recurrent nature may require repeated resections using the carbon dioxide laser.

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Extensive psoriasis induced by interferon alfa treatment for chronic hepatitis C

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Abstract

A 47 year old man with chronic hepatitis C was treated with interferon alfa, 3 million units three times a week, and developed widespread plaque psoriasis within weeks of starting interferon therapy. There was no previous history of psoriasis. The psoriasis was characterised by extensive nail involvement and plaques at the interferon injection sites. The patient relapsed after a total of 12 months of interferon and was subsequently treated with interferon and tribavirin (ribavirin) with recurrence of the psoriasis.

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Keywords: hepatitis C; interferon alfa; psoriasis

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Interferon alfa is a naturally occurring glycoprotein and an immune modulating agent that is used in the treatment of several medical conditions including hepatitis B and C. There have been reports of exacerbations of autoimmune conditions after the therapeutic use of interferon.[1–3] There have also been case reports of exacerbations of psoriasis during interferon therapy.[4–6] These flare-ups of psoriasis have usually led to a cessation of interferon treatment. We present a patient who had no previous history of psoriasis, but presented with extensive psoriasis shortly after starting interferon treatment for chronic hepatitis C. Interferon was continued despite persisting active psoriasis.

Case report

A 47 year old white man was found to have hepatitis C infection after presenting with abnormal liver function tests. There was a history of intravenous drug use 15 years previously. Serum was positive for hepatitis C virus RNA, genotype 1a using the polymerase chain reaction (PCR), and a liver biopsy specimen showed severe inflammatory changes with probable cirrhosis (Knodell score = 11). There was no past history of skin disease or family history of psoriasis.

He was treated with interferon alfa-2a (Schering-Plough) at a dose of 3 million units (MU) three times a week, and received a total of 12 months of treatment. His transaminase levels reverted to normal and serum hepatitis C virus RNA was not detected while on treatment.

Interferon was initially well tolerated, but three months after starting treatment he presented with severe psoriasis affecting the entire body and scalp. Nail changes were particularly striking and there were plaques of psoriasis over his injection sites (figs 1 and 2).

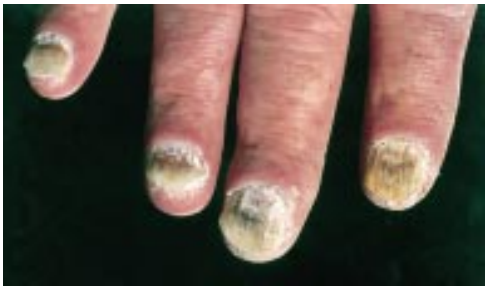

Figure 1 Severe nail dystrophy associated with psoriasis induced by interferon.