

Case Report

Anesthetic management of a life-threatening stenosis of large airways in a patient with Wegener's granulomatosis: A case report

Hamzeh Hoseinzade, Ata Mahmoodpoor, Sarvin Sanaie

From Department of Anesthesiology and Intensive Care, Tabriz University of Medical science, Imam Khomeini Hospital, Tabriz, Iran.

Correspondance: Dr Ata Mahmoodpoor, No. 423. Arman residential tower, 22nd Bahman Ave.

Ashrafi Esfahani Highway, Tehran. Iran. E-mail: Dr_am5757@yahoo.com

Received: May 3, 2007 Accepted: June 10, 2007

ABSTRACT

Large airway stenosis is common in patients with Wegener's granulomatosis. We used high frequency jet ventilation for management of anesthesia during rigid bronchoscopy for dilation of left main bronchus in a patient with Wegener's granulomatosis. (Rawal Med J 2007;32:201-203).

Keywords: Wegener's granulomatosis, anesthetic management, high frequency jet ventilation.

INTRODUCTION

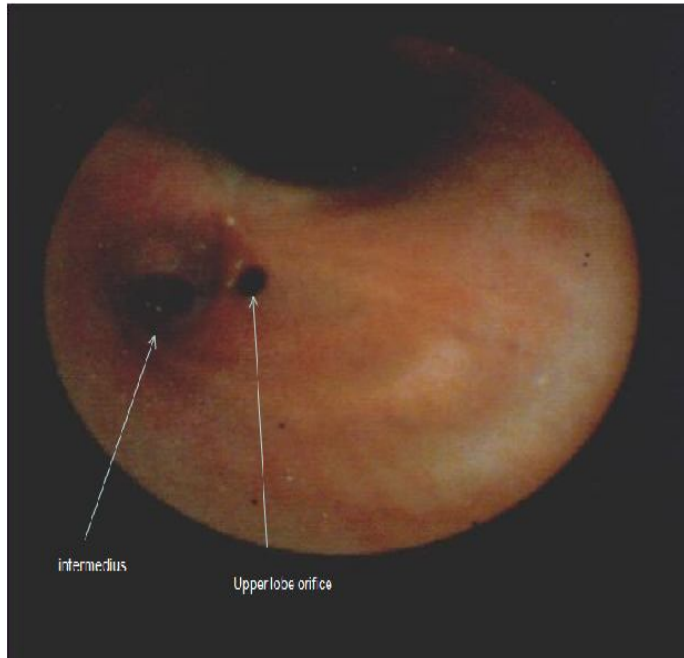
There are many causes of stenosis of large airways which may generate a life-threatening situation. Wegener's granulomatosis is a disease of unknown characterized by granulomatous vasculitis of the upper and lower respiratory tract and other organs.^{1,2} Patients most frequently require intensive care treatment for severe pneumonitis, glomerulonephritis, stroke, myocardial infarction, multiorgan system dysfunction and infection due to immunosuppression and anatomic abnormalities secondary to the granulomatous inflammation.³⁻⁵ Most patients (85-90%) present with symptoms referable to the upper respiratory tract including sinusitis and nasal septal deformity.⁶ Laryngeal involvement may result in severe narrowing of the upper respiratory tract.⁷ Lower respiratory tract disease is found in almost all patients after evaluation.⁸

We present a case with bronchial stenosis where we used high frequency jet ventilation (HFJV) for anesthesia management.

CASE REPORT

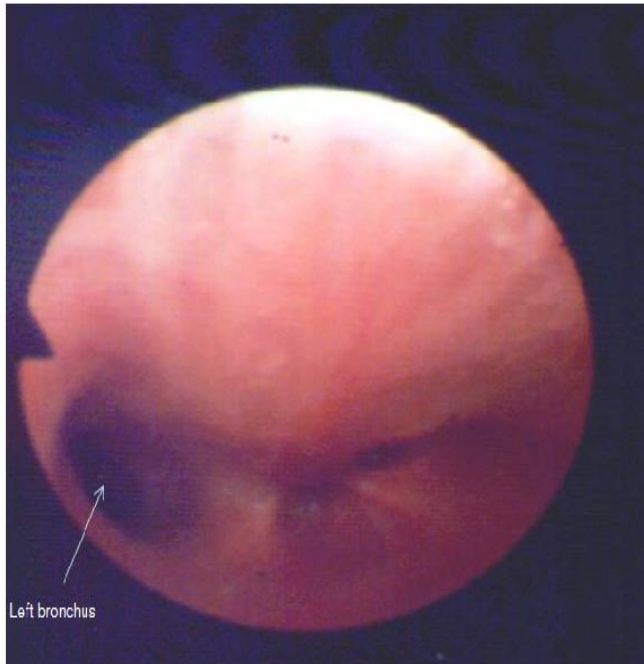
The patient was a 57-year-old woman, a known case of Wegener's granulomatosis for 8 years being treated with cyclophosphamide. She was admitted with dyspnea and cough. She had saddle nose deformity, tachycardia, biphasic stridor, crackles, and SpO₂: 75-80% (without O₂ supplementation). Chest CT-Scan showed right upper lobe collapse. There was severe obstructive pattern in spirometry. Fiberoptic bronchoscopy showed swelling of arytenoid and aryepiglottic folds, normal trachea, stenosis of right bronchus distal to upper lobe branch (figure 1), 95% stenosis of left bronchus (figure 2), and fibrotic tissue in carina. Superior laryngeal nerve block was performed with lidocaine 2%, and glottis and oropharynx were anesthetized with lidocaine 10% spray. She fiber optic bronchoscopy and a black-colored suction catheter was inserted into proximal right main bronchus.

Fig 1. Stenosis of right bronchus.



The catheter was connected to manual jet ventilator and the patient was ventilated (SpO_2 :90%, Fio_2 :1). After certainty of adequacy of ventilation, rigid bronchoscope was inserted into left bronchus (SpO_2 :85%, Fio_2 :1, just with ventilation of right upper lobe). Severe stenosis of left bronchus was seen and dilation was performed. Then, the catheter was withdrawn up to carina and the bronchoscope was inserted into the right bronchus where obstruction of right main bronchus was seen. After withdrawing the bronchoscope, both lungs were ventilated sufficiently (SpO_2 :96%, Fio_2 :1).

Fig 2. Stenosis of left bronchus



She developed signs of subcutaneous emphysema and pneumomediastinum and, on fiberoptic bronchoscopy, rupture of trachea at the site of stenosis was found. The patient expired before any further intervention was possible.

DISCUSSION

In cases of clinically significant stenosis of large airway if the patient is not a suitable candidate for resection, interventional bronchoscopy dilation of airway is an appropriate alternative for the management of these problems. Since our patient was a candidate for bronchoscopy, we decided to perform it under local anesthesia but oxygenation of the patient was still a problem. Only ventilation of right upper lobe was possible, so we performed ventilation by an intra-bronchial catheter which was connected to manual jet ventilator.

High frequency ventilation, has been variously defined but in general, represents mechanical ventilation at high rates of usually 60-100 breaths/min (bpm). It has three types and we used HFJV in this study. HFJV ventilates between 100-1200 bpm and the tidal volume is delivered by a jet port that injects a bolus of gas down the tube with exhalation occurring on the outside of the tube lumen. Adult patients can be intubated with a triple lumen endotracheal tube, one port for the jet, one for the distal pressure manometer and one for the cuff.⁹ FDA has approved the use of HFJV for hemodynamically unstable patients with restrictive defect in which controlled mechanical ventilation failed, presence of bronchopleural fistula in which significant amounts of airway gas is leaving via the chest tubes and during bronchoscopy so that ventilation is not interrupted. As we could not intubate this patient, we used modified HFJV.

Monitoring of patients receiving high-frequency ventilation requires the ability to monitor O₂ and CO₂ exchange, airway disconnection and obstruction and airway pressure (extremely important) in particular. Expiratory port occlusion can result in extremely high airway pressures. On the other hand, increasing peak airway pressure can result in lower Paco₂.¹⁰ In this case, tracheal rupture was due to dilatation, and HFJV is not considered as the cause since it usually causes parenchymal injury and not large airways injury. In conclusion, HFJV with adequate monitoring can be used as an appropriate alternative for ventilation in ill patients with bronchial stenosis requiring rigid bronchoscopy.

REFERENCES

1. Passey J, Walker R. Wegener's granulomatosis: an unusual cause of upper airway obstruction. *Anesthesia* 2000;55:682-684.

2. Cupps TR, Fauci AS. Wegener's granulomatosis, in Smith LH Jr (ed): The vasculitides. Philadelphia, WB Saunders, 1981;72.
3. Ewart BH, Jennette JC, Falk RS. The pathogenic role of antineutrophil cytoplasmic autoantibodies. *Am J Kidney Dis* 1992;18:1888.
4. Savage CO, Pottinger BE, Gaskin G. Autoantibodies developing to myeloperoxidase and proteinase 3 in systemic vasculitis stimulate neutrophil cytotoxicity toward cultured endothelial cells. *Am J Pathol* 1992;141:335.
5. Harper L, Cockwell P, Dwoma A. Neutrophil priming and apoptosis in anti-neutrophil cytoplasmic autoantibody-associated vasculitis. *Kidney Int* 2001;59:1729.
6. Fauci AS, Haynes BF, Katz P. Wegener's granulomatosis: prospective clinical and therapeutic experience with 85 patients for 21 years. *Ann Intern Med* 1983;98:76.
7. Harrington JT, McCluskey RT. Case 24-1979. Case records of the Massachusetts General Hospital: weekly clinicopathological exercises. *N Engl J Med* 1979;300:1378.
8. McDonald TJ, DeRemee RA. Wegener's granulomatosis. *Laryngoscope* 1983;93:220.
9. William CW, Jonathan LB. Anesthesia for Thoracic Surgery. In: Ronald D. Miller's Anesthesia. Sixth edition. 2005;vol 2:1847-1939.
10. Richard EM, Enrico MC. Respiratory Monitoring. In: Ronald D. Miller's Anesthesia. Sixth edition, 2005;vol 1:1437-1481.

