Surgical correction of acquired unilateral diaphragmatic paralysis by plication technique

INTRODUCTION

Acquired diaphragmatic paralysis can result from direct involvement of the diaphragm or injury to the phrenic nerve from thoracic or cardiac surgery, trauma, infections (mainly viral), tumours or autoimmune diseases or it may be idiopathic. In terms of pathophysiology, loss of normal diaphragmatic contractility leads to muscular atrophy, dilatation of the dome, reduction in the effectiveness of contraction during inspiration and atelectasis of the adjacent pulmonary segments. Chronic dyspnoea and occasionally respiratory insufficiency may gradually develop. If the dyspnoea significantly impairs simple daily activities, surgical intervention using the diaphragmatic plication technique and reposition of the hemidiaphragm are definitely indicated. This intervention allows the lung to re-expand, resulting in improvement of the pulmonary function tests and leading to alleviation of the symptoms.

CASE REPORT

A 76 year-old female patient with a long-term history of bronchial asthma, hyperlipidaemia, hypertension and chronic atrial fibrillation presented with dyspnoea of several years’ duration. An elevated left hemidiaphragm had been noted on chest X-ray 6 years earlier. The dyspnoea had become pro-

SUMMARY. Acquired diaphragmatic paralysis may compromise lung mechanics and cause dyspnoea and/or lead to respiratory failure in the long term. A 76 year-old female patient presented with progressive worsening of dyspnoea and spirometric indices, and imaging studies revealed elevation of the left hemidiaphragm. Surgical correction was carried out by diaphragmatic plication technique, through a mini-thoracotomy approach. Immediate alleviation (within days) of her symptoms was observed, while improvement of radiological and pulmonary function tests occurred some weeks later. Pneumon 2013, 26(2):6-10.
gressively worse over the last two years (MRC:3) and pulmonary function testing was significantly impaired [FVC: 1100 mL (43% pred.), FEV₁: 660 mL (35% pred.), FEV₁/FVC: 0.60], despite treatment with maximum doses of inhaled bronchodilators and steroids and modification of cardiac medication. Mild calcification and moderate regurgitation of the aortic valve were detected on cardiac ultrasound. The left ventricle showed normal ejection fraction (>60%) and marginal myocardial wall thickening. The right heart chambers and pulmonary arterial pressure were within normal limits. Measurement of the arterial blood gases (FiO₂:0.21) showed: PO₂=69 mmHg, PCO₂=36 mmHg, pH=7.407. Smooth elevation of the left hemidiaphragm was observed on chest X-ray (Figure 1A) and a CT scan revealed an enlarged calcified lymph node (maximum diameter 2.5cm) near the aortopulmonary window (Figures 2A,2B). Fluoroscopy confirmed unilateral paralysis and paradoxical motion of the left hemidiaphragm.

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**FIGURE 1.** Chest X-ray in a 76 year-old woman with dyspnoea: **1A** pre-operative, showing elevation of the left hemidiaphragm (by 2-3 intercostal spaces); **1B** 3 months after left diaphragmatic plication, showing obvious radiological improvement.

**FIGURE 2.** Chest CT scan in 76 year-old woman with dyspnoea, showing: **A** Elevation of abdominal organs into the left hemithorax; **B** A sizeable, uniformly calcified lymph node near the aortopulmonary window.
PPD skin test was negative. Surgical correction of the diaphragmatic position by the technique of plication was decided upon. Left mini-thoracotomy was performed through the 7th intercostal space and the dilated redundant hemidiaphragm was plicated with the use of grasping forceps, along the transverse axis and at a level 10-12 cm higher than the pleurodiaphragmatic angle. After excluding the presence of abdominal viscera in the diaphragmatic fold by careful palpation, the basal segment of the pleated diaphragm was sutured with two 90 mm type TA staples (Figures 3A to 3D). Subsequently, the apex of the fold was anchored to the anterior dome of the left hemidiaphragm and sutured in place (Prolene No 2) in parallel with the staples of the base (Figures 3E, 3F). The postoperative period was uneventful and the dyspnoea progressively improved (MRC:1), while improvement of the radiological appearance and pulmonary function [FVC:1700 mL (67% pred.), FEV1: 760 mL (40% pred.)] was observed three months later (Figure 1B).

**DISCUSSION**

Acquired paresis or paralysis of the hemidiaphragm can result from a series of abnormalities that affect the neuromuscular axis between the cervical spinal cord and the diaphragm\(^2\). The idiopathic form is the most common in adults, followed by tumour invasion, traffic accidents and surgical injury of the phrenic nerve. In the present case, the calcified lymph node could indicate a possible cause of unilateral phrenic nerve paralysis, since there was no history of major thoracic surgery or severe injury. The chest X-ray performed 6 years earlier could not exclude the presence of diaphragmatic paralysis at that time.

Medical treatment or surgical excision of enlarged lymph nodes may remove the pressure from the phrenic nerve, but frequently fail to restore diaphragmatic function in patients with benign mediastinal lymphadenopathy from causes such as tuberculosis. In this situation, diaphragmatic plication may be necessary\(^3,4\). Indications for surgical correction and its appropriate timing have not
been fully defined, since most studies reported have been retrospective and uncontrolled. Clinically significant dyspnoea, cough, chest pain and the need for mechanical ventilation are included among the possible indications for surgery. In the case of trauma, a 1-2 year post-injury period of observation can be rationally recommended, provided that adequate respiratory reserve permits this. The patient presented here was operated on because of severe dyspnoea and lack of response to medical treatment. The worsening of her dyspnoea did not appear to be related to deterioration of bronchial asthma or cardiac function, as evidenced by clinical and imaging studies.

The traditional surgical approach is open thoracotomy through the 6th, 7th or 8th intercostal space, with or without reinforcing mesh. The thoracoscopic approach is less invasive (two to four ports are used), but single-lung ventilation is required for this method and workspace is limited. A number of plication techniques with continuous sutures, interrupted sutures or metallic staples have been described. In order to avoid single-lung ventilation and injury to the intercostal nerves, and to ensure greater workspace and excellent view of the diaphragm, surgical correction is occasionally performed by laparotomy or laparoscopic plication. In rare cases of neuromuscular disease, it is possible to correct both hemidiaphragms surgically by thoracotomy in a single operation or two separate procedures. The low FEV1 and respiratory insufficiency in the present case were the reasons for choosing mini-thoracotomy through the 7th intercostal space, as this ensures optimal ventilation during operation and prevents extended injury to the accessory respiratory muscles.

It has been demonstrated that diaphragmatic plication increases tidal volume (Vt), transdiaphragmatic pressure (∆Pdi), gastric to oesophageal pressure ratio (∆Pga/∆Pao) and dynamic pulmonary compliance (∆dyn), and decreases respiratory work in experimental animals with unilateral or bilateral diaphragmatic paralysis. The positive effects of the plication technique on pulmonary function tests (FEV1, FVC), dyspnoea (MRC) and quality of life scales (SGRQ) have been reported for every type of surgical approach, thoracotomy, thoracoscopy and laparoscopy, although there has been no comparative study. The mean observation period in the small series of patients documented ranges from 1 month to 10 years. Improvement may occur during the first post-operative weeks and be sustained for several years after surgery.

The major complications of diaphragm plication include pneumonia, pleural effusion with prolonged retention (>7 days) of the chest tube, respiratory insufficiency and need for mechanical ventilation, abdominal compartment syndrome, trauma to abdominal viscera, conversion of an initially closed approach to an open procedure (4% to 26.8%), deep venous thrombosis, pulmonary embolism, acute myocardial infarction and cardiac arrhythmia. Although thoracoscopy requires a smaller incision than thoracotomy, the frequency of occurrence and the intensity of chronic postoperative thoracic pain show no difference between the two techniques.

Apart from spirometry in the seated position and arterial blood gas testing, a complete diagnostic approach for diaphragmatic dysfunction includes spirometry in the supine position, static lung volumes, maximal static inspiratory pressure and sniff nasal inspiratory pressure. Patients with clinically significant diaphragmatic paralysis should be tested by nocturnal oxymetry and polysomnography, especially when there are constitutional symptoms (fatigue, daytime somnolence, depression, morning headache, frequent nocturnal wakening). Most of these diagnostic tests were not included in the diagnostic work-up of this patient, which is recognized as a major shortcoming of the case report.

In conclusion, surgical correction of acquired unilateral diaphragmatic paralysis by plication technique offers the important benefits of improving dyspnoea, pulmonary function tests and quality of life. Beneficial effects have been reported for every type of surgical approach (transthoracic or transabdominal, open or videoscopic), although there have been no comparative studies. The choice of the technique is based on the training experience and the preference of the thoracic surgeon, taking into account the postoperative morbidity dependent on the type of intervention.

REFERENCES