Scholars Journal of Applied Medical Sciences (SJAMS) **ISSN 2320-6691 (Online)**
Sch. J. App. Med. Sci., 2015; 3(8D):2998-3000 **ISSN 2347-954X (Print)**

(An International Publisher for Academic and Scientific Resources)
www.saspublishers.com DOI: 10.36347/sjams.2015.v03i08.050

Case Report

Recurrent Respiratory insufficiency Associated with Phrenic Nerve Paralysis after Coronary Artery Bypass Grefting: A Case Report

Duygu Kara, Ozgur Ozmen
Department of Anesthesiology and Reanimation, Erzurum Regional Training and Research Hospital, Erzurum, Turkey

***Corresponding author**
Duygu Kara
Email: drduygukara@yahoo.com

Abstract: Eventration of the diaphragm is generally regarded as a condition in which the left or the right leaf of the diaphragm has ascended abnormally high into the chest. The eventration may result from an acquired affection leading to permanent destruction of the phrenic nerve. Phrenic nerve injury is a well-recognised complication of cardiac surgery that can lead to disabling effects from diaphragmatic dysfunction, especially in children and patients with a history of chronic obstructive airway disease. Various mechanisms of injury have been recognised including hypothermia, mechanical trauma and possibly ischaemia. A clear understanding of these mechanisms is important in order to modify surgical techniques to prevent this serious complication of cardiac surgery. Diaphragmatic plication is intended to decrease lung compression, to make the thoracic base and mediastinum more stable, and to strengthen the respiratory action of intercostal, perithoracic, and abdominal muscles. In this case report, a 59-year-old male patient who developed recurrent respiratory insufficiency associated with phrenic nerve paralysis and diaphragmatic eventration after coronary artery bypass graftingin the intensive care unit was presented.
Keywords: Diaphragmatic eventration, phrenic nerve paralysis, respiratory insufficiency, coronary artery bypass grefting

INTRODUCTION

Diaphragmatic eventration, which is more common in males, is a rare anomaly defined by permanent elevation of the diaphragm without defects, representing atrophy and progressive distension of the diaphragmatic muscles. The presence of diaphragmatic eventration in adults indicates different possible etiologies, including trauma, neoplasms, infection, degenerative disease and idiopathy [1]. Diaphragmatic paralysis (DP) following phrenic nerve paralysis (PNP) is a well-recognized complication of cardiac surgery [2]. Paradoxical motion of the diaphragm may cause severe respiratory difficulty resulting in tachypnea, atelectasis, pneumonia, continued respiratory distress, and CO2 retention after extubation [3]. Early diagnosis and treatment prevent prolonged mechanical ventilation and improve outcome. It is usually a benign condition, but it may lead to severe impairment, or even to death in some cases. It may present as a wide range of manifestations ranging from an asymptomatic radiographic abnormality to severe pulmonary dysfunction requiring prolonged mechanical ventilation, causing morbidity or even mortality [2, 3].Surgical treatment is indicated only in the presence of symptoms [4].

CASE REPORT

A 59-year-old male patient who had respiratory insufficiency, tachypnea, sinus tachycardia, chest pain was admitted to our intensive care unit. He had recurrent respiratory insufficiency and admitted to the intensive care unit three times again before. He had a history of coronary artery bypass grafting (CABG) for significant coronary artery disease two years ago. He was a nonsmoker and nondrinker, had history of chronic obstructive pulmonary disease. At first, the reason of respiratory insufficiency was supposed to be the patient's chronic obstructive pulmonary disease. Upon development of recurrent respiratory problems, DP was suspected due to the paradoxical movement of the epigastrium during spontaneous ventilation and by the elevation of the left hemidiaphragm on the chest X-ray. The diagnosis was confirmed by ultrasonographic assessment during spontaneous breathing. Other causes of dyspnea were excluded by clinical, biochemical, echocardiographic, and radiologic findings. Pulmonary function tests showed reductions in vital capacity (VC), forced vital capacity (FVC), forced expiratory volume in one second (FEV1), and total lung capacity (TLC). Antibiotherapy and intermittent positive airway pressure ventilation by a nasal mask resulted in significant improvement in the general condition of the patient. Breathlessness and palpitation were observed

only on exertion. With anticipation of spontaneous recovery of DP, the patient was discharged 10 days after surgery with grade 1 dyspnea. During a follow-up period of six months, increased elevation of the right hemidiaphragm was noted on the chest X-ray with worsening respiratory distress. The patient had dyspnea at rest and experienced significant discomfort during physical activities. Thoracoscopic diaphragmatic plication was performed with standard thoracotomy. Postoperative course was uneventful and the patient was discharged on the fourth postoperative day. After the operation, dyspnea disappeared, the chest X-ray showed normalization of the position of the left hemidiaphragm, and there was marked improvement in the expansion of the left lung. Pulmonary function tests showed slight improvements in VC, FVC, and FEV1/ FVC, and a notable increase in TLC. One month after plication, the position of the diaphragm was normal on the chest X-ray and remained normal within the next six months. Ultrasonographic and fluoroscopic examinations during spontaneous breathing showed complete normalization of the diaphragmatic motion.

DISCUSSION

Most adult patients with diaphragmatic eventration remain asymptomatic, and the diagnosis is made incidentally on chest radiography. Among symptomatic patients, the most common symptom is dyspnea. Thoracic surgery, especially cardiac surgery, is still associated with noncardiac complications related to injury to intrathoracic structures. The causes of DP are cardiothoracic surgery, mediastinal mass or surgery, cold paralysis, infections (Lyme disease, HIV), motor neuron disease, and thoracic radiotherapy [1, 2]. Some studies attribute this complication to cold-induced paralysis during myocardial protection strategies and to mechanical injury during internal mammary artery harvesting [5]. Setina et al. [6] suggested that the anatomical interrelation between the phrenic nerve and the proximal segment of the mammary artery was a significant factor for PNP in cardiac surgery. In addition, stretch injury during cardiac surgery may also result in PNP [7]. Unilateral DP is much more common than bilateral involvement. Patients with unilateral DP tend to be asymptomatic. This condition is not life-threatening, but may cause post- operative complications like atelectasis or prolonged postoperative mechanical ventilation [8]. Apart from these complications, recognition of PNP is clinically very important as symptoms of respiratory impairment are frequently misinterpreted. The symptoms can mimic those associated with congestive heart failure, cardiac tamponade, and pulmonary embolism [9]. The diagnosis of DP can be missed in older patients and postoperative cases. Moreover, the diagnosis of unilateral paralysis is often delayed, unless it follows trauma or cardiothoracic surgery. Diaphragmatic paralysis may be suspected by the observation of elevated hemidiaphragm on the chest X-ray; however, it should be confirmed by the diaphragm mobility test (sniff test) with ultrasonographic and/or fluoroscopic screening during spontaneous breathing [10]. The management of DP remains controversial in adult patients. Therapeutic options are continued mechanical ventilation until recovery of phrenic nerve function, awaiting spontaneous recovery of diaphragm function during the early postoperative period, and early surgical intervention. In unilateral DP, spontaneous recovery usually occurs and surgical plication is occasionally necessary. Indications for plication include inability to wean from the ventilator, recurrent pneumonia, respiratory distress, chest pain, poor exercise tolerance, and cardiac arrhythmias [2]. Today, surgical plication is widely accepted especially in post CABG patients. However, its timing is still controversial. Some authors recommend immediate plication as soon as the confirmation of diagnosis of DP, while others recommend a waiting period in anticipation of potential spontaneous recovery [1].Our patient had recurrent respiratory insufficiency, and needed mechanical ventilation many times. Symptoms of respiratory failure were present even at rest.We believe that the development of DP in our patient was due to mechanical injury during internal mammary artery harvesting. Therefore, diaphragmatic plication was performed, which resulted in significant improvements in left lung expansion and all spirometric values.

CONCLUSION

In conclusion, DP following PNP is a severe complication and an important cause of dyspnea after cardiac surgery. Dyspnea caused by heart diseases must be distinguished from that case. Patients who suffer from respiratory insufficiency after CABG should be considered not only for cardiac diseases but also for DP. Diaphragmatic plication should be performed in cases in which spontaneous recovery is not seen. Plication is an easy and safe procedure that results in early clinical and radiological improvement.

REFERENCES

1. Deslauriers J; Eventration of diaphragm. Chest Surg. Clin. N Am, 1998;8:315-360.
2. Tripp HF, Bolton JW; Phrenic nerve injury following cardiac surgery: a review. J Card Surg 1998;13:218-223.
3. Kuniyoshi Y, Yamashiro S, Miyagi K, Uezu T, Arakaki K, Koja K; Diaphragmatic plication in adult patients with diaphragm paralysis after cardiac surgery. Ann Thorac Cardiovasc Surg 2004;10:160-166.
4. Groth SS, Andrade RS; Diaphragm plication for eventration or paralysis: a review of the literature. Ann Thorac Sur, 2010;89:2146-2150.
5. Nikas DJ, Ramadan FM, Elefteriades JA; Topical hypothermia: ineffective and deleterious as adjunct

to cardioplegia for myocardial protection. Ann Thorac Surg, 1998;65:28-31.
6. Setina M, Cerny S, Grim M, Pirk J; Anatomical interrelation between the phrenic nerve and the internal mammary artery as seen by the surgeon. J Cardiovasc Surg, 1993;34:499-502.
7. Tönz M, von Segesser LK, Mihaljevic T, Arbenz U, Stauffer UG, Turina MI; Clinical implications of phrenic nerve injury after pediatric cardiac surgery. J Pediatr Surg, 1996;31:1265-1267.
8. Benjamin JJ, Cascade PN, Rubenfire M, Wajszczuk W, Kerin NZ; Left lower lobe atelectasis and consolidation following cardiac surgery: the effect of topical cooling on the phrenic nerve. Radiology, 1982;142:11-14.
9. Esposito RA, Spencer FC; The effect of pericardial insulation on hypothermic phrenic nerve injury during open-heart surgery. Ann Thorac Surg, 1987;43: 303-308.
10. Katz MG, Katz R, Schachner A, Cohen AJ; Phrenic nerve injury after coronary artery bypass grafting: will it go away? Ann Thorac Surg, 1998;65:32-35.