An important cause of dyspnea after coronary artery bypass grafting: phrenic nerve paralysis

Koroner arter baypas cerrahisi sonrası nefes darlığının önemli bir nedeni: Frenik sinir felci

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Diaphragmatic paralysis (DP) due to phrenic nerve paralysis is a rare complication after cardiac surgery. A 48-year-old male patient developed respiratory insufficiency, tachypnea, sinus tachycardia, chest pain, pneumonia, and fever immediately after coronary artery bypass grafting. Paradoxical movement of the epigastrium was noted during spontaneous ventilation and the chest X-ray showed elevation of the left hemidiaphragm. The diagnosis of DP was confirmed by ultrasonographic assessment. Antibiotherapy and intermittent positive airway pressure ventilation by a nasal mask resulted in significant improvement in the general condition of the patient. Respiratory problems were observed only on exertion. Spontaneous recovery of DP was considered and the patient was discharged 10 days after surgery with grade 1 dyspnea. However, after six months of follow-up, increased elevation of the left hemidiaphragm was noted on the chest X-ray with worsening respiratory discomfort even at rest. Thoracoscopic diaphragmatic plication was performed. After the operation, dyspnea disappeared, the chest X-ray showed the left hemidiaphragm in its normal position, and there was marked improvement in spirometric values.

Key words: Coronary artery bypass/adverse effects; diaphragm/surgery; dyspnea; phrenic nerve/injuries; postoperative complications; respiratory paralysis/etiology.

Frenik sinir felci sonucu oluşan diyafram felci kalp cerrahisi sonrası görülen nadir bir komplikasyondur. Kırk sekiz yaşında bir erkek hastada, koroner arter baypas cerrahisinin hemen sonrasında solunum sıkıntısı, hızlı solunum, sinüs taşikardisi, göğüs ağrısı, pnömoni ve ateş gelişti. Spontan ventilasyon sırasında epigastriyumun paradoksal hareketi ile birlikte göğüs grafisinde sol hemidiyaframın yükseldiği görüldü. Diyafram felci tanısı ultrasonografiyle doğrulandı. Antibiyoterapi ve burun maskesiyle aralıklı pozitif havayolu basınçlı ventilasyon uygulaması sonrasında hastanın durumunda önemli derecede düzelme görüldü. Solunum sorunları sadece hareketle ortaya çıkmaktaydı. Diyafram felcinin kendiliğinden düzeleceği düşünülerek, hasta cerrahiden 10 gün sonra derece 1 nefes darlığı ile taburcu edildi. Ancak, altı aylık izlem sonunda, göğüs grafisinde sol hemidiyaframda daha fazla yükselme ve istirahatte bile solunum sorunları gözlenmesi üzerine hastaya torakoskopik diyafram plikasyonu yapıldı. Ameliyat sonrasında hastanın solunum sıkıntısı kayboldu, pulmoner fonksiyon testleri düzeldi ve göğüs grafisinde sol hemidiyafram normal yerleşimde görüldü.

Anahtar sözcükler: Koroner arter baypas/yan etki; diyafram/cerrahi; nefes darlığı; frenik sinir/yaralanma; ameliyat sonrası komplikasyon; solunum felci/etyoloji.

Diaphragmatic paralysis (DP) following phrenic nerve paralysis (PNP) is a well-recognized complication of cardiac surgery.[1] Paradoxical motion of the diaphragm may cause severe respiratory difficulty resulting in tachypnea, atelectasis, pneumonia, continued respiratory distress, and CO_2 retention after extubation.[2] 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.[1,2]

CASE REPORT

A 48-year-old male patient developed respiratory insufficiency, tachypnea, sinus tachycardia, chest pain, pneumonia, and fever immediately after coro-

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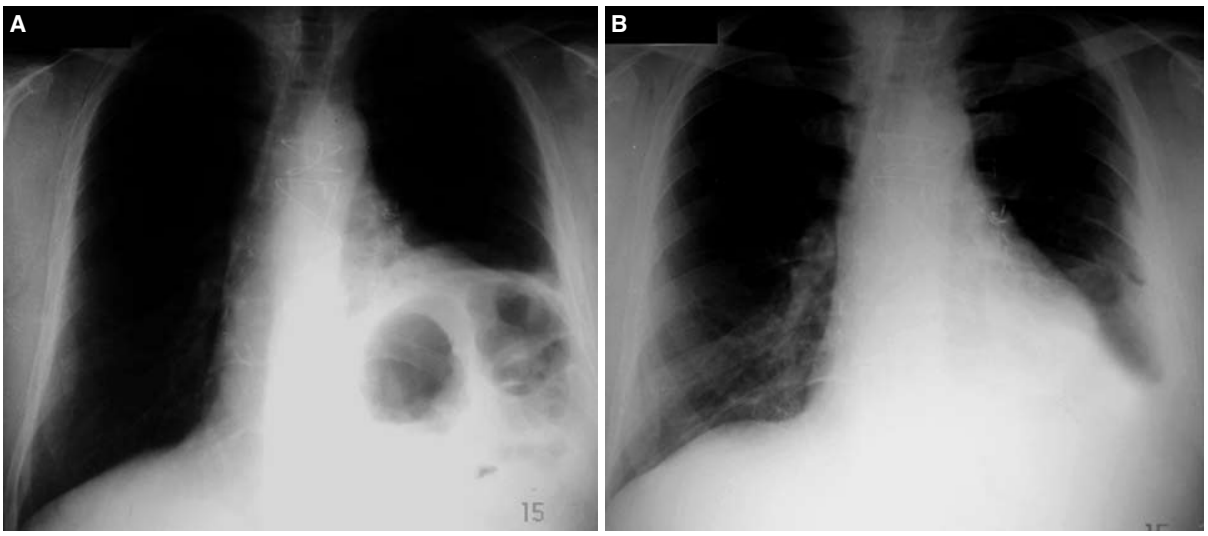

Figure 1. (A) Chest X-ray showing elevation of the left hemidiaphragm three days after coronary artery bypass grafting. **(B)** One month after plication, the diaphragm appears in its normal position.

nary artery bypass grafting (CABG) for significant coronary artery disease (total occlusion of the proximal left anterior descending artery and right coronary artery). He was a nonsmoker and nondrinker, had diabetes mellitus and hypertension, but no history of chronic pulmonary disease. Preoperatively, left ventricular systolic and diastolic functions were normal on echocardiographic examination (ejection fraction 55%).

Surgery was performed with a median sternotomy and under total cardiopulmonary bypass (CPB) through the ascending aorta and right atrium cannulation. Myocardial protection was obtained by moderate total body hypothermia (28-32 °C) and cold crystalloid cardioplegic solution (4°C). The mammary artery was prepared using a skeletonized harvesting technique with papaverine application. The radial artery and the left internal mammary artery (LIMA) were used for the right coronary artery and left anterior descending coronary artery, respectively. At the end of the procedure, the patient was weaned off CPB.

Postoperatively, there were no electrocardiographic and echocardiographic changes. Upon development of 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 (Fig. 1a). 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 (FEV_1), 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 left 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. No other cardiac and pulmonary causes of dyspnea were present.

Thoracoscopic diaphragmatic plication was performed with standard thoracotomy. A 10x5-cm longitudinal plication was accomplished with a series of nine 2-0 nylon monofilament U-stitches placed between the medial and lateral parts of the diaphragm, taking care not to damage the phrenic nerve. 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 FEV_1/FVC, and a notable increase in TLC (Table 1). One

Table 1. Changes in pulmonary function

	4 days after CABG	1 month after plication	6 months after plication
Vital capacity (l)	2.18	2.20	2.23
Forced vital capacity (FVC) (l)	2.12	2.19	2.19
Forced expiratory volume in 1 second (FEV_1) (l)	1.70	1.82	1.83
FEV_1/ FVC (%)	80.1	83.1	83.5
Total lung capacity (l)	2.82	3.34	3.46

CABG: Coronary artery bypass grafting.

month after plication, the position of the diaphragm was normal on the chest X-ray (Fig.1b) and remained normal within the next six months. Ultrasonographic and fluoroscopic examinations during spontaneous breathing showed complete normalization of the diaphragmatic motion.

DISCUSSION

Thoracic surgery, especially cardiac surgery, is still associated with noncardiac complications related to injury to intrathoracic structures. The incidence of unilateral DP varies between 2.1% to 10% and retrospective studies report lower rates when compared to those of prospective ones.[3,4] The causes of DP are cardiothoracic surgery, mediastinal mass or surgery, cold paralysis, infections (Lyme disease, HIV), motor neuron disease, and thoracic radiotherapy.[1,2,5] Some studies attribute this complication to cold-induced paralysis during myocardial protection strategies and to mechanical injury during internal mammary artery harvesting.[6] In addition to LIMA use, surgical technique, diabetes, low preoperative myocardial performance, and increased age have been implicated as potential risk factors.[7,8]

Hypothermic injury to the phrenic nerve was first reported by Scannell as a causative factor.[9] In an experimental study with dogs, Marco at al.[10] demonstrated that contact of the phrenic nerve with ice chips for 30 to 60 min resulted in nerve paralysis lasting for 6 to 28 days. The pathologic examination showed a spectrum of injuries extending from mild demyelination to severe axonal fragmentation. Several hypotheses have been proposed to explain the mechanism of cold-induced PNP, including the rate of cooling, the lowest temperature obtained, alteration in intracellular solute concentrations, formation of intracellular ice crystals, and the rate of rewarming.[11]

Setina et al.[12] 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. Two mechanisms have been proposed, by which the use of internal mammary artery can interfere with phrenic nerve function: surgical injury to the nerve during LIMA harvesting and ischemia of the nerve caused by the ligation of the arterial side branches supplying the nerve.[13] In addition, stretch injury during cardiac surgery may also result in PNP.[14]

Diaphragmatic paralysis is frequently seen on the left side of the diaphragm. 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.[11-13]

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.[15] Nowadays, ultrasound evaluation of diaphragm function is a sensitive, safe, and noninvasive method without radiation exposure and has replaced the use of radioscopy and electromyography.

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. Spontaneous recovery is seen within 5 days to 12 months after PNP but cannot

be predicted.[2,7,8] In unilateral DP, spontaneous recovery usually occurs and surgical plication is occasionally necessary. Recovery of the hemidiaphragm to the normal position occurs in 80% within one year, with increasing rates in two years.[15] Indications for plication include inability to wean from the ventilator, recurrent pneumonia, respiratory distress, chest pain, poor exercise tolerance, and cardiac arrhythmias.[2,10,11] 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.[1,5,7,15]

We believe that the development of DP in our patient was due to perioperative use of topical ice-slush or LIMA harvesting, or both. The patient did not require prolonged ventilation, but elevation of the left hemidiaphragm on the chest X-ray and respiratory distress increased in the postoperative six months without spontaneous recovery. Symptoms of respiratory failure were present even at rest. Therefore, diaphragmatic plication was performed, which resulted in significant improvements in left lung expansion and all spirometric values.

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 dyspnea 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.

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