

## Resolution of pericardial effusion after minimally invasive surgical repair of pectus excavatum

*Pektus ekskavatum minimal invaziv cerrahi onarımı ile düzelen kronik perikardiyal efüzyon*

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Pectus excavatum may cause the compression of the heart, thereby, disturbing the pericardial fluid turnover which results in pericardial effusion. In this article, we report a 32-year-old female case with pectus excavatum and chronic recurrent pericardial effusion. After the minimally invasive repair of pectus excavatum and pericardiocentesis, no recurrence was observed during one-year follow-up. By elevating the sternum and relieving the cardiac compression, minimally invasive repair of pectus excavatum may be helpful in restoring the pericardial fluid production and re-absorption mechanism.

**Keywords:** Minimally invasive surgery; pectus excavatum, carinatum; pericardium.

Pectus excavatum (PE) is the most common chest wall deformity, occurring as frequently as 1 in 300-400 live births. The depressed sternum in this deformity may compress the thoracic viscera, especially the heart and lungs.<sup>[1]</sup> To repair PE, minimally invasive repair of pectus excavatum (MIRPE) via a retrosternal metal bar has increasingly become the treatment of choice.<sup>[2]</sup>

Pericardial effusion (PCE) is a relatively common finding in everyday clinical practice which is mostly related to a wide variety of conditions such as pericarditis, neoplasia, renal failure, and trauma.<sup>[3]</sup> It can be treated medically, but in some cases, pericardiocentesis or surgery is necessary.<sup>[4]</sup>

Pektus ekskavatum, kalpte sıkışmaya ve buna bağlı olarak perikardiyal sıvı döngüsünü bozarak perikardiyal efüzyona neden olabilir. Bu yazıda pektus ekskavatum ve kronik tekrarlayan perikardiyal efüzyonlu 32 yaşında bir kadın olgu sunuldu. Minimal invaziv pektus ekskavatum tamiri ve perikardiyosentez sonrasında, bir yıllık takipte nüks gözlenmedi. Sternumu kaldırarak ve kalbin sıkışmasını rahatlatarak, minimal invaziv pektus ekskavatum tamiri perikardiyal sıvı üretimi ve yeniden emilim mekanizmasının tekrar inşa edilmesine yardımcı olabilir.

**Anahtar sözcükler:** Minimal invaziv cerrahi; pektus ekskavatum, karinatum, perikardiyum.

This report describes a case with both PE and recurrent PCE. The deformity was corrected with MIRPE, and perioperative pericardiocentesis was also performed. No PCE recurrence developed during the follow-up period.

### CASE REPORT

A 32-year-old woman was hospitalized because of PE. Not only did the patient complain about the deformity, but she was also suffering from palpitations and mild dyspnea that had begun more than 10 years earlier. Furthermore, she had been followed up by a cardiologist for the previous four years with multiple



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echocardiographies, and these had revealed mild-to-moderate PCE ranging from 8 to 20 mm in thickness. However, there was no sign of pericardial tamponade. The patient had also undergone multiple diagnostic and therapeutic pericardiocenteses that revealed transudative PCE. She was then diagnosed as having chronic PCE. The tests that had been performed to rule out connective tissue disease were negative, and she had not been taking any medications. A prior magnetic resonance imaging (MRI) of the heart had demonstrated that the depressed sternum was compressing both the left ventricle and atrium, and PCE measuring 20 mm in thickness (Figure 1) had been identified. The patient's past medical history was otherwise unremarkable.

At our facility, a physical examination revealed a deep symmetric PE deformity. Her blood pressure was 125/80 mmHg, and electrocardiography revealed a normal sinus rhythm of 80 beats per minute. However, the jugular venous pressure was not elevated nor was there any hepatomegaly or pulsus paradoxus present. The routine blood chemistries, coagulation studies, and blood gas analysis were all within normal limits, and the results of her respiratory function tests were also normal [forced vital capacity (FVC): 3.99L (104%), forced expiratory volume in one second (FEV1): 3.83 (115%)]. A preoperative computed tomography (CT) of the chest revealed the PE deformity as well as mild-to-moderate PCE.



**Figure 1.** Sagittal magnetic resonance imaging section of the thorax showing the sternal depression (thick arrow). The heart chamber (thin arrow) was compressed, and there was a moderate amount of pericardial effusion (star).

The patient underwent MIRPE with the technique defined by Yüksel et al.<sup>[5]</sup> and a 230 mm bar was used in the procedure. We drained 100 ml of serous-like fluid perioperatively via pericardiocentesis, and the results of the fluid analysis showed the transudative nature of the PCE [glucose= 99 mg/dL, total protein= 2.6 g/dL, albumin= 1.6 g/dL, and lactate dehydrogenase (LDH)= 147 U/L]. In addition, the white blood cell (WBC) count was 600/U with a neutrophilic predominance (62.2%). Furthermore, microbiological and cytological examinations revealed no signs of fungal, bacterial, or mycobacterial infection or malignancy. The patient had an uncomplicated postoperative course and was discharged four days after the surgery.

We followed up the patient for a year. She no longer complained about the palpitations or dyspnea and was satisfied with her physical appearance. A chest CT at the postoperative sixth month (Figure 2) showed the pectus bar that was elevating the sternum, and echocardiography at the postoperative 12<sup>th</sup> month showed no recurrence of the PCE.

## DISCUSSION

Although PE is well tolerated in infancy and childhood, older patients may complain of chest pain and dyspnea after sustained exercise. In addition, a few patients may have mitral valve prolapse and associated atrial arrhythmia which may cause palpitations.<sup>[1]</sup> Our patient suffered from mild dyspnea and palpitations, but there was no arrhythmia. Her symptoms had developed more than 10 years earlier and had progressed slowly.

Echocardiography is the most available and reliable technique to verify the presence and amount of PCE.



**Figure 2.** Postoperative thoracic computed tomography showing the elevated sternum by the pectus bar. No signs of a pericardial effusion were present.

When the sum of the echo-free spaces in the anterior and posterior pericardial sacs is between 10 and 20 mm, the effusion is at a moderate level.<sup>[3]</sup> This technique was used to diagnosis the PCE in our patient. The effusion was mild at the beginning but became moderate over time.

Pericardial effusion is chronic when it is present for an extended period of time that can range from several months to several years. Usually chronic PCE progresses slowly, as in our case, and causes no clinically important symptoms.

In PE, the depression of the sternum can produce a deformity, in particular an anterior indentation of the right ventricle. When this occurs, the heart is displaced to the left, often with a sternal imprint on the anterior wall of the right ventricle.<sup>[6]</sup> A study by Coln et al.<sup>[7]</sup> found cardiac chamber compression in 95% of the patients with PE, but this resolved in all of the patients postoperatively. Furthermore, the cardiac symptoms of 86% of their patients also resolved after surgical repair.

The thoracic echocardiography and MRI of our patient verified the posterior displacement of the sternum and revealed the pericardial and cardiac compression. These changes may also have led to the development of PCE in our case since one of the proposed mechanisms suspected of causing PCE is the obstruction of lymphatic drainage from the pericardial space.<sup>[8]</sup> We do not know how long our patient had PCE since she had been suffering from symptoms for more than a decade but had only been diagnosed four years previously. However, since all of the other mechanisms and diseases that can cause PCE had been ruled out, it is possible that the deep PE deformity might have induced the development of PCE. The recurrence of the PCE despite multiple pericardiosyntheses also supports this thesis since the PE deformity was still causing pericardial and cardiac compression. The MIRPE procedure relieved the compression that had been identified in the postoperative echocardiography and chest CT, and this may explain why the PCE has not recurred postoperatively.

Chronic PCE may be related to cardiac compression caused by PE. Surgical repair of the PE may relieve the compression to the heart and help return the pericardial fluid production-reabsorption cycle to normal. To our knowledge, this is the first case in the literature to demonstrate the resolution of PCE following MIRPE.

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