Development of left ventricular apical akinesis and thrombus during pericardiocentesis for pericardial tamponade

Perikard tamponadı için perikardiyosentez sırasında sol ventrikül apikal akinezisi ve trombüs gelişimi

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Although pericardiocentesis is a more practical and comfortable alternative to surgical drainage in patients with pericardial tamponade, it may sometimes be associated with transient ventricular dysfunction due to rapid drainage of the fluid. We presented a 42-year-old female patient who developed left ventricular systolic dysfunction and thrombus concomitant with segmental wall motion disorder in the left ventricle during pericardiocentesis for the treatment of massive pericardial effusion and cardiac tamponade. The patient developed acute dyspnea and tachycardia on the second day of pericardiocentesis with a drainage of 500 ml per day. Left ventricular ejection fraction decreased to 20%, and there was akinesis in the left ventricular apex, and severe hypokinesis in the septum. The amount of daily drainage was decreased to 250 ml. Echocardiography performed on the fifth day showed an image, 1x1 cm in size, compatible with an apically located thrombus and unfractionated heparin infusion was initiated. Coronary angiography showed normal coronary arteries. At the end of the first week, the drainage decreased below 50 ml/day. Ejection fraction returned to normal at the end of 10 days and the thrombus diminished and disappeared. Analysis of the pericardial fluid showed tuberculous pericarditis and antituberculous treatment was instituted.

Key words: Cardiac tamponade/therapy; drainage; pericardial effusion; pericardiocentesis/adverse effects; ventricular dysfunction, left.

Perikard tamponadı olan hastalarda perikardiyosentez cerrahi drenaja göre daha pratik ve konforlu bir seçenek olmasına rağmen, sıvının hızlı boşaltılmasına bağlı olarak geçici ventrikül disfonksiyonu gelişebilmektedir. Bu yazıda, yaygın perikard efüzyonu ve kardiyak tamponad tedavisi için uygulanan perikardiyosentez sırasında sol ventrikülde segmenter duvar hareket bozukluğu ile birlikte sol ventrikül sistolik disfonksiyonu ve trombüs gelişen 42 yaşında bir kadın hasta sunuldu. Günde 500 ml olarak belirlenen sıvı çekilmesinin ikinci gününde hastada akut dispne ve taşikardi gelişti. Hastanın sol ventrikül ejeksiyon fraksiyonu %20'ye düştü; sol ventrikül apeksinde akinezi, septumda hipokinezi saptandı. Günlük sıvı çekimi miktarı 250 ml'ye düşürüldü. Beşinci günde yapılan ekokardiyografik kontrolde, sol ventrikül apeksinde trombüsle uyumlu, 1x1 cm büyüklüğünde bir görüntü saptandı. Hastada fraksiyone olmayan heparin infüzyonuna başlandı. Koroner anjiyografide koroner arterler normal bulundu. Bir haftanın sonunda, çekilen sıvı miktarı 50 ml'nin altına düştü. Onuncu günde hastanın ejeksiyon fraksiyonu normal değerde idi, trombüsün tümüyle çözülüp kaybolduğu görüldü. Perikard sıvısının incelemesinde tüberküloz perikarditi saptanması üzerine hastada antitüberküloz tedaviye başlandı.

Anahtar sözcükler: Kardiyak tamponad/tedavi; drenaj; perikard efüzyonu; perikardiyosentez/yan etki; ventrikül disfonksiyonu, sol.

Pericardiocentesis is a method frequently used in the treatment of pericardial tamponade.[1] Although it is quite a safe procedure when performed by experienced hands, certain complications may develop including damage to cardiac structures.[2] Impaired ventricular function after pericardiocentesis is a rare complication.[3,4]

In this report, we presented a case in which pericardiocentesis was complicated by left ventricular systolic dysfunction and thrombus in the left ventricular apex.

CASE REPORT

A 42-year-old female patient was admitted to our clinic with complaints of chest pain and dyspnea of

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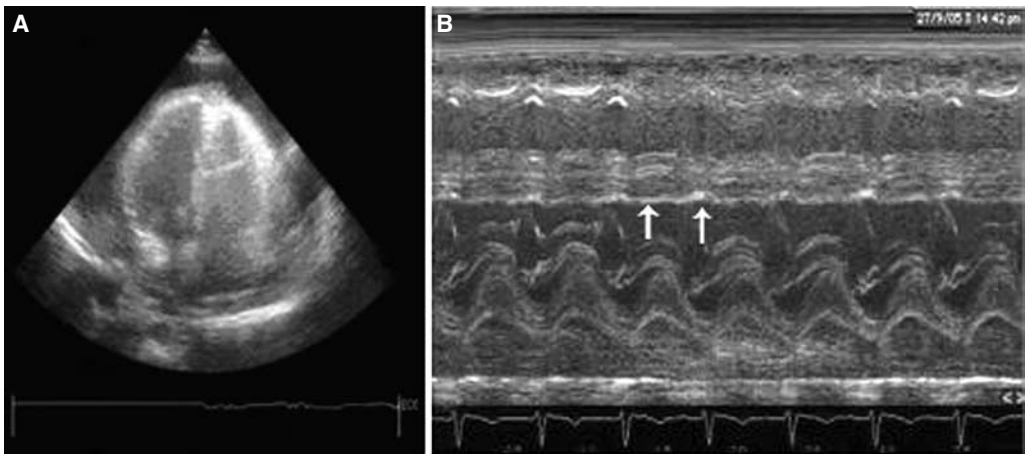

Figure 1. (A) Pericardial fluid completely surrounding the heart in the apical four-chamber view. **(B)** Parasternal long-axis M-mode image showing severe septal hypokinesia (arrows) due to left ventricular systolic dysfunction after pericardiocentesis. Posterior wall contractility is normal.

three-month history. On physical examination, her blood pressure was 90/60 mmHg, and pulse rate was 110 bpm and rhythmic. Cardiac auscultation showed deep heart sounds and there was a paradoxical pulse. Low voltage activity was noted on the electrocardiogram. Teleradiography showed an increased cardiothoracic index. Hematological parameters were within normal ranges. Kidney and liver functions were evaluated as normal. Transthoracic echocardiography showed massive pericardial effusion with signs of tamponade (Fig. 1a). Left ventricular function was normal. All valves showed normal flow and structure. Pericardiocentesis was performed with a 16-gauge puncture needle using a subxiphoidal approach. A 6-F pigtail catheter was then inserted into the pericardial cavity. To avoid heart failure, drainage was limited to 500 ml per day. Acute dyspnea and tachycardia developed after the drainage of 500 ml fluid on the second day of pericardiocentesis. Her blood pressure was 100/70 mmHg, and pulse rate was 120 bpm and rhythmic. A negative T wave, which was not present on the initial electrocardiogram, was observed in all precordial leads. A control echocardiographic examination showed left ventricular ejection fraction (EF) as 20%, akinesis in the left ventricular apex, and severe hypokinesis in the septum (Fig. 1b). Treatment with an angiotensin-converting enzyme (ACE) inhibitor, diuretic, and digoxin was initiated. The amount of daily drainage was decreased to 250 ml. Echocardiography performed on the fifth day showed an image, 1x1 cm in size, compatible with an apically located thrombus (Fig. 2a) and unfrac-

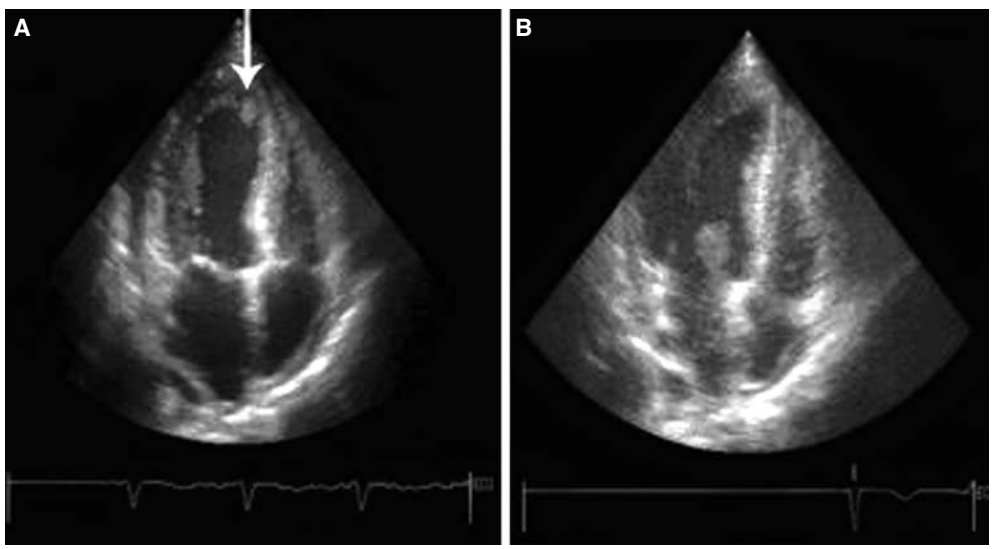

Figure 2. (A) Apical four-chamber view showing a mass, 1x1 cm in size, compatible with thrombus (arrow) in the left ventricular apex. **(B)** Echocardiographic image obtained five days after initiation of heparin therapy shows resolution of the thrombus.

tionated heparin infusion was initiated, which would make aPTT twice the normal value. Coronary angiography was performed to assess coronary anatomy and impaired wall motion. Coronary arteries were found to be normal. A total of 2000 ml hemorrhagic fluid was drained from the patient. At the end of the first week, the drainage decreased below 50 ml/day and the pigtail catheter was removed. No increases were observed in the pericardial fluid. In serial echocardiographic follow-up, EF returned to normal at the end of 10 days and the thrombus diminished and disappeared (Fig. 2b). Analysis of the pericardial fluid showed tuberculous pericarditis and antituberculous treatment was instituted.

DISCUSSION

Transient ventricular dysfunction is a very unusual complication after removal of pericardial effusion for cardiac tamponade. The left ventricle, right ventricle, or both ventricles may be affected from this condition.[2,3,5] Various hypotheses have been proposed to explain the pathophysiology of this phenomenon. Some authors addressed fluctuations in the hemodynamic features of the heart for the development of ventricular dysfunction.[5-7] It was suggested that pulmonary edema was precipitated by a mismatch between preload and afterload. A rapid drainage of a large amount of pericardial effusion would release the compression of the right heart and produce a sudden increase in venous return, resulting in left ventricular overload, while systemic vascular resistance is still high due to adrenergic stimulation occurring in cardiac tamponade.[2,6] In another study, Konstam and Levine[7] suggested that, after acute pericardial decompression, right ventricular output would exhibit a greater increase than left ventricular output, leading to ventricular dysfunction.

It has also been suggested that ventricular dysfunction might develop as a result of ischemic causes. Decreased coronary blood flow was demonstrated with increased pericardial pressure.[8] Braverman and Sundaresan[9] suggested that diminished coronary blood flow due to pericardial fluid compression of epicardial coronary arteries might lead to myocardial stunning and hibernation, thus contributing to transient systolic dysfunction. This was based on a report of decreased left ventricular contractility observed in experimental cardiac tamponade with changes in coronary perfusion pressure.[8] Ligero et al.[5] speculated that pericardial fluid pressure over the coronary arteries might produce myocardial ischemia and stunning, which would probably be masked by sympathetic overdrive in the acute phase of cardiac tamponade. In this setting, drainage of pericardial effusion might lead to left ventricular overload and overt heart failure. They suggested that, since ventricular dysfunction associated with pericardiocentesis was not a common finding in clinical practice, transient myocardial dysfunction following pericardial drainage would be more likely to develop from the removal of a great volume of pericardial fluid in a short time, requiring rapid adjustment of coronary resistance and autonomic nervous system modulation. Anguera et al.[2] proposed a similar theory. We also feel that this theory is more acceptable.

As an another alternative mechanism, the interplay between the sympathetic-parasympathetic system may be associated with the development of ventricular dysfunction following pericardial drainage. Wolfe and Edelman[10] reported that the removal of the stimulus for sympathetic outflow (drainage of pericardial effusion) might have an unmasking effect on left ventricular dysfunction, which may have been obscured by transient tachycardia and inotropic effect associated with high catecholamine levels.

In addition, Takotsubo syndrome, first described in 1991 by Dote et al.,[11] may be a mechanism of transient left ventricular dysfunction. In this syndrome, transient left ventricular apical ballooning occurs without evidence for relevant coronary artery stenosis and clinical signs of acute myocardial infarction.[12] Several mechanisms have been proposed, including epicardial coronary spasm, microvascular coronary spasm, or catecholamine-mediated toxicity.[13,14]

In our case, the localization of the contraction defect in the left ventricular apical region and septum and the development of the apical thrombus suggest that left ventricular systolic dysfunction might be due to the causes proposed in the ischemic theory or Takotsubo syndrome.

In conclusion, transient left ventricular systolic dysfunction is a rare complication after the treatment of pericardial tamponade with pericardiocentesis and is more frequently observed in subjects in whom rapid drainage of the pericardial fluid is performed. Mostly, it improves spontaneously without requiring any treatment. In our case, the condition progressed to wall motion impairment and development of apically located thrombus. Patients undergoing pericardiocentesis for pericardial tamponade should be closely monitored following the procedure for the development of left ventricular dysfunction.

REFERENCES

1. Buchanan CL, Sullivan VV, Lampman R, Kulkarni MG. Pericardiocentesis with extended catheter drainage: an effective therapy. Ann Thorac Surg 2003;76:817-20.
2. Anguera I, Pare C, Perez-Villa F. Severe right ventricular dysfunction following pericardiocentesis for cardiac tamponade. Int J Cardiol 1997;59:212-4.
3. Geffroy A, Beloeil H, Bouvier E, Chaumeil A, Albaladejo P, Marty J. Prolonged right ventricular failure after relief of cardiac tamponade. Can J Anaesth 2004;51:482-5.
4. Uemura S, Kagoshima T, Hashimoto T, Sakaguchi Y, Doi N, Nakajima T, et al. Acute left ventricular failure with pulmonary edema following pericardiocentesis for cardiac tamponade-a case report. Jpn Circ J 1995;59: 55-9.
5. Ligero C, Leta R, Bayes-Genis A. Transient biventricular dysfunction following pericardiocentesis. Eur J Heart Fail 2006;8:102-4.
6. Vandyke WH Jr, Cure J, Chakko CS, Gheorghiade M. Pulmonary edema after pericardiocentesis for cardiac tamponade. N Engl J Med 1983;309:595-6.
7. Konstam MA, Levine HJ. Pulmonary edema after pericardiocentesis. N Engl J Med 1984;310:391.
8. Wechsler AS, Auerbach BJ, Graham TC, Sabiston DC Jr. Distribution of intramyocardial blood flow during pericardial tamponade. Correlation with microscopic anatomy and intrinsic myocardial contractility. J Thorac Cardiovasc Surg 1974;68:847-56.
9. Braverman AC, Sundaresan S. Cardiac tamponade and severe ventricular dysfunction. Ann Intern Med 1994; 120:442.
10. Wolfe MW, Edelman ER. Transient systolic dysfunction after relief of cardiac tamponade. Ann Intern Med 1993;119:42-4.
11. Dote K, Sato H, Tateishi H, Uchida T, Ishihara M. Myocardial stunning due to simultaneous multivessel coronary spasms: a review of 5 cases. J Cardiol 1991;21: 203-14. [Abstract]
12. Bahlmann E, Schneider C, Krause K, Pankuweit S, Harle T, Kuck KH. Tako-Tsubo cardiomyopathy (apical ballooning) with parvovirus B19 genome in endomyocardial biopsy. Int J Cardiol 2007;116:e18-21.
13. Kurisu S, Sato H, Kawagoe T, Ishihara M, Shimatani Y, Nishioka K, et al. Tako-tsubo-like left ventricular dysfunction with ST-segment elevation: a novel cardiac syndrome mimicking acute myocardial infarction. Am Heart J 2002;143:448-55.
14. Wittstein IS, Thiemann DR, Lima JA, Baughman KL, Schulman SP, Gerstenblith G, et al. Neurohumoral features of myocardial stunning due to sudden emotional stress. N Engl J Med 2005;352:539-48.