An unusual cause of OptiVol alarm: increased intra-abdominal pressure associated with irritable bowel syndrome

OptiVol alarmına yol açan alışılmamış bir durum: İrritabl bağırsak sendromundan kaynaklanan karıniçi basınç artışı

Cengizhan Türkoğlu, M.D., Farid Aliyev, M.D., Cengiz Çeliker, M.D., Gökhan Çetin, M.D.

Department of Cardiology, Division of Pacing and Electrophysiology, İstanbul University, Institute of Cardiology, İstanbul

Monitoring intrathoracic impedance has become an integral part of follow-up of patients with implantable cardioverter defibrillator (ICD) or cardiac resynchronization therapy/defibrillator due to heart failure. However, several noncardiac factors may influence intrathoracic impedance. We report on an unusual cause of decrease in intrathoracic impedance in a 54-year-old male patient following successful implantation of biventricular ICD for heart failure symptoms due to nonischemic dilated cardiomyopathy and severely impaired left ventricular systolic function. During the follow-up period, the patient presented several times with the OptiVol alarm due to an increase in the OptiVol fluid index, in the absence of symptoms or signs of heart failure. Further inquiry into the possible causes of decreased intrathoracic impedance revealed that the patient had frequent episodes of irritable bowel syndrome, which increased intra-abdominal pressure, leading to elevation of diaphragm and subsequent compression of intrathoracic organs, and thus to a decrease in intrathoracic impedance.

Key words: Abdomen; body fluids/physiology; cardiography, impedance; defibrillators, implantable; heart failure; hemodynamics; pressure.

Kalp yetersizliği nedeniyle kardiyoverter defibrilatör (ICD) yerleştirilen veya kardiyak resenkronizasyon tedavisi/defibrilatör uygulanan hastalarda göğüsiçi impedans ölçümlerine dayalı izlem tedavinin önemli bir parçası haline gelmiştir. Bununla birlikte, göğüsiçi impedans ölçümleri kalp yetersizliği dışındaki nedenlerden de etkilenebilmektedir. Bu yazıda, 54 yaşında bir erkek hastada, iskemik olmayan dilate kardiyomiyopati ve ileri derecede bozulmuş sol ventrikül sistolik fonksiyonları nedeniyle başarılı biventriküler ICD yerleştirilmesinden sonra göğüsiçi impedans düşüşüne yol açan alışılmamış bir neden bildirildi. İzlemi sırasında hasta, kalp yetersizliğine dair semptom ve bulgular olmaksızın, birkaç kez OptiVol sıvı indeksindeki artışa bağlı olarak oluşan OptiVol alarmı sonucu başvurdu. Göğüsiçi impedans düşüşüne yol açan nedeni ortaya çıkarmak için yapılan incelemelerde, hastanın sık olarak irritabl bağırsak sendromu atakları geçirdiği görüldü. Bu sorunun karıniçi basıncı artırarak diyafram yükselmesine ve göğüsiçi organ basısına yol açtığı, göğüsiçi impedansın da bu nedenle düştüğü sonucuna varıldı.

Anahtar sözcükler: Karın; vücut sıvısı/fizyoloji; kardiyografi, impedans; defibrilatör, takılabilir; kalp yetersizliği; hemodinami; basınç.

Today, follow-up and adjustments of treatment based on measured values of intrathoracic impedance are receiving more attention and importance in patients with heart failure who have implantable cardioverter defibrillator (ICD) or cardiac resynchronization therapy/defibrillator (CRT-D). However, values of intrathoracic impedance can be affected by other noncardiac factors. This report illustrates an unusual cause of decrease in thoracic impedance due to increased intra-abdominal pressure resulting from irritable bowel syndrome.

CASE REPORT

A 54-year-old male patient with NYHA class III heart failure symptoms due to nonischemic dilated cardiomyopathy and severely impaired left ventricular

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Correspondence: Dr. Farid Aliyev. İstanbul Üniversitesi Kardiyoloji Enstitüsü, Kardiyoloji Anabilim Dalı, 34034 Haseki, İstanbul, Turkey. Tel: +90 212 - 459 20 00 / 29522 e-mail: drfaridaliyev@yahoo.com.tr

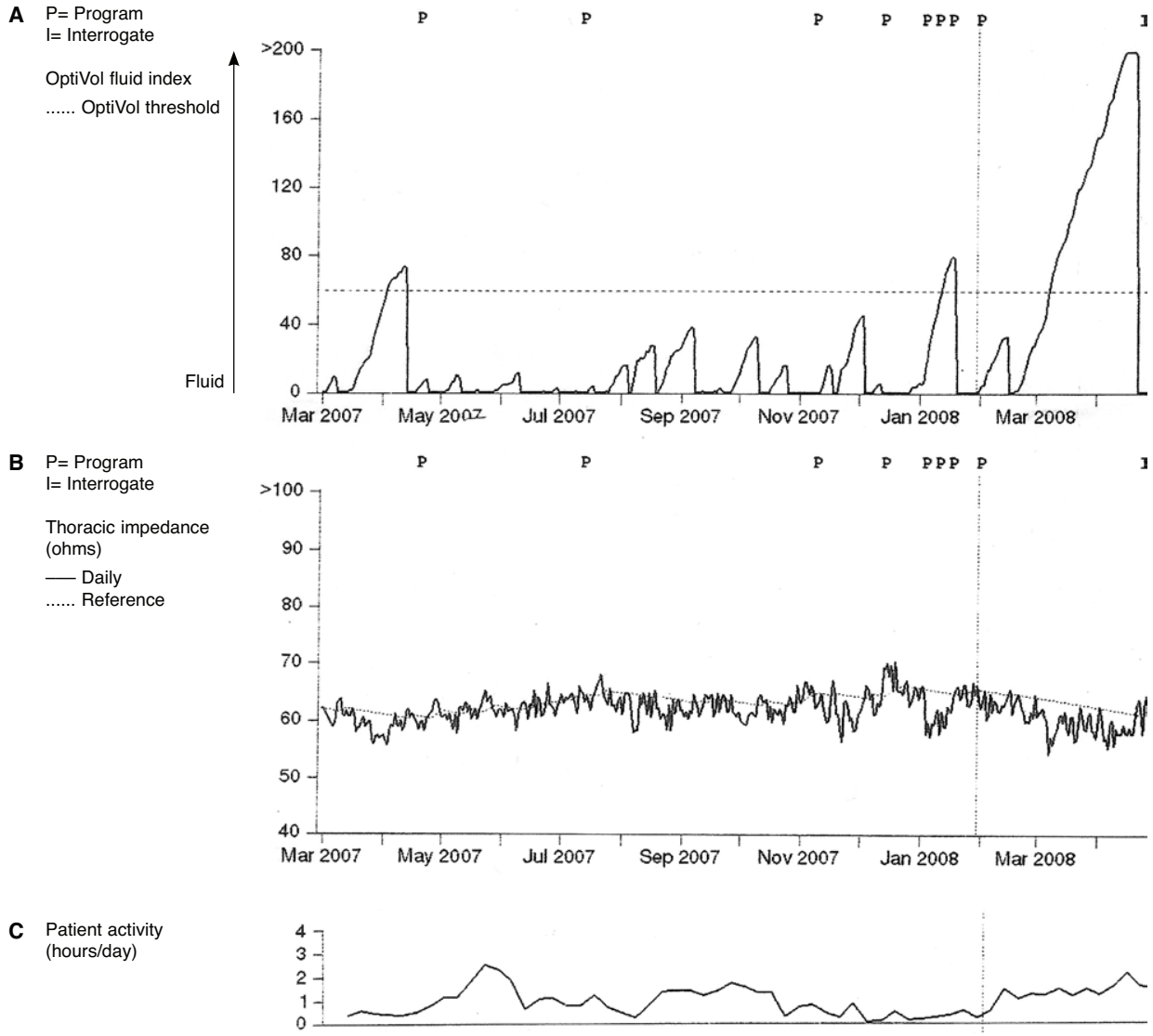

Figure 1. (A) Three episodes of increase in the fluid index exceeding the threshold level and **(B)** corresponding thoracic impedance values. **(C)** Patient activity graph showing relatively normal activity during the periods of decrease in thoracic impedance.

systolic function (ejection fraction 20%) underwent successful implantation of biventricular ICD (InSync Sentry, Medtronic Inc., Minneapolis, MN, USA). His symptoms significantly resolved following device implantation. During the follow-up period, the patient was admitted because of the OptiVol alarm due to an increase in the OptiVol fluid index. This increase was slight and was not accompanied by symptoms or signs of heart failure and we did not make any change in his therapy. The patient had been asymptomatic during the following nine months until he again came into physician's attention because of the OptiVol system alarm. At this stage, we again did not make any change in his medications. Two months later, the fluid index considerably increased again, with values exceeding 200 Ω, without symptoms of heart failure, and in the presence of normal brain natriuretic peptide level (Fig. 1). In an attempt to find out possible causes of this increase, spirometric evaluation was performed and its findings were suggestive of mild-to-moderate restrictive pulmonary physiology. FEV_1 (forced expiratory volume in 1 second), FVC (forced expiratory vital capacity), and TLC (total lung capacity) were found to be decreased, while DLCO (diffusion capacity of carbon monoxide) was within normal limits. Based on these findings, the possibility of any pulmonary parenchymal process was excluded and this restrictive physiology was attributed to extrapulmonary causes. A more detailed evaluation revealed that the patient had frequent episodes of severe

constipation, flatulence, and meteorism, suggesting that increases in the fluid index might be related to constipation-induced increases in intra-abdominal pressure, leading to elevation of diaphragm and subsequent compression of intrathoracic organs. This compression effect decreased intrathoracic impedance, which resulted in increases in the OptiVol fluid index. Further gastroenterological evaluation led to the diagnosis of irritable bowel syndrome.

DISCUSSION

Management of patients with a biventricular device is challenging, even for those experienced in dealing with conventional pacemakers. Monitoring thoracic fluid status via intrathoracic impedance is the newest device-based diagnostic capability and in November 2004, the US Food and Drug Administration (FDA) approved a novel heart failure monitoring tool, OptiVol fluid status monitoring (Medtronic Inc.), for use on some biventricular ICDs. Newer ICDs introduced by the same manufacturer also have this feature. This system measures impedance every 20 minutes from noon to 05 PM for a total of 64 measurements over a 5-hour period. The device records the average of all measurements as a daily impedance value. Although some threshold values for fluid indexes are generally programmed (for example 60 Ω-days), the system has the advantage of comparing the patient's current data with previous measurements. There is an inverse relationship between intrathoracic impedance and intracardiac filling pressures. It has also been shown that intrathoracic impedance decreases before symptom onset for all heart failure-related hospitalizations and decreases in intrathoracic impedance precede symptom onset by an average of 15.3±10.6 days.[1] These data emphasize the importance of intrathoracic impedance measurement when dealing with heart failure patients.

Several intrathoracic causes of changes in thoracic impedance have been reported in the literature. Decreases in impedance have been demonstrated in patients with pleural/pericardial effusion,[2] pneumonia,[2,3] and those undergoing pacemaker pocket revision,[2,4,5] while increased impedance was observed in a case of pneumothorax.[6] Here, it should be noted that any pulmonary event that occurs in the lung contralateral to the device pocket is not expected to affect intrathoracic impedance value.[2] Another important cause of decreased thoracic impedance is revision of the pacemaker pocket or postimplantation period. This period is characterized by local edema and accumulation of fluid within the pocket, resulting in decreased intrathoracic impedance values, c the amount of time required for complete recovery and stabilization may vary. For this reason, measurement of impedance values is frequently postponed in the early recovery period and is initiated approximately in the fifth week following implantation.[2,4,5]

Our case suggests that intrathoracic impedance can be affected by other noncardiac and extrathoracic factors. It is well-known that the thorax and abdomen are two adjacent closed spaces separated by the diaphragm, and a rise in the intra-abdominal compartment pressure may be associated with an increase in the thoracic pressure. In our case, increased intra-abdominal pressure possibly led to the elevation of the diaphragm and compression of intrathoracic organs, giving rise to a parallel increase in intrathoracic pressure and consequently to a decrease in thoracic impedance. This mechanism is supported by the observation that increased intra-abdominal pressure is associated with elevated pleural and other intrathoracic pressures.[7,8] Here, we can speculate that other causes such as obesity can also lead to increases in abdominal and so thoracic pressures. All these factors should be considered in cases with decreased intrathoracic impedance without coexistent heart failure symptoms. Considering that fluid accumulation can be asymptomatic or can precede symptom onset by more than 1-2 weeks depending on disease severity, a detailed diagnostic evaluation should always be undertaken in all cases with decreased intrathoracic impedance.

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