Intrathoracic Impedance Monitor Alarm in a Patient with Cardiac Resynchronisation Therapy and Advanced Lung Carcinoma

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The intrathoracic impedance monitor system measures impedance between the device case and the right ventricular coil and reflects intrathoracic fluid status. It is used to detect early volume overload in patients with chronic heart failure. We report a case of inappropriate activation of the intrathoracic impedance monitor alarm in a patient with epidermoid lung cancer and pleural carcinosis.

A cardiac resynchronisation device equipped with intrathoracic impedance monitoring is a useful complementary tool for the ambulatory management of chronic heart failure. However, other causes of pulmonary fluid retention/accumulation should be considered if clinical evidence of exacerbation of heart failure is lacking. Here we describe a case of inappropriate activation of the intrathoracic impedance monitor alarm in a patient with epidermoid lung cancer and pleural carcinosis.

Case presentation

A 77-year-old man presented to our pacemaker outpatient clinic with a triggered audible alarm of the OptiVol® intrathoracic impedance monitor system of his cardiac resynchronisation therapy-defibrillator (CRT-D) device (Medtronic, Concerto II) in November 2010. He had no complaints of dyspnoea, oedema, weight gain, or poor exercise performance. The cardiac resynchronisation device had been implanted 2 years previously because of advanced ischaemic cardiomyopathy. After CRT-D implantation, the patient’s physical condition improved and recent echocardiography revealed improved left ventricular (LV) systolic function and reduced LV dimensions. Device interrogation showed that daily intrathoracic impedance started to decrease in October 2010, until the OptiVol fluid index crossed the nominal threshold of 60 Ω-days and caused the alert on November 3, 2010 (Figure 1A, B). As physical examination, chest X-ray and laboratory values demonstrated no deterioration in heart failure, the OptiVol fluid index threshold was empirically reprogrammed from 60 to 100 Ω-days. A few days before the alarm alert, chest computer tomography (CT) was performed in response to the patient’s complaints of a chronic dry cough. The CT scan revealed a tumour in the lower right pulmonary lobe with enlarged mediastinal lymph nodes, a small lesion in the upper left pulmonary lobe, and pleural carcinosis (Figure 2). After bronchoscopy and a histological examination of biopptic material, the diagnosis of advanced epidermoid lung cancer was made and chemotherapy with gemcitabine and carboplatin was initiated. Two months later the patient had no symptoms of heart failure and a follow-up CT scan showed no progression of the lung carcinoma. However, a further decrease in intrathoracic impedance was observed. Despite the higher fluid index threshold, the OptiVol alarm was again triggered and the
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Optivol fluid index reached >200 Ω-days in late November 2010 (Figure 1A). At that time we decided to turn off the OptiVol alarm system.

Discussion

The intrathoracic impedance monitor system measures impedance between the device case and the right ventricular coil and can detect early changes in intrathoracic fluid volume in patients with chronic heart failure. With pulmonary congestion the intrathoracic impedance decreases, and when the OptiVol fluid index (calculated by comparing the daily average impedance values with a reference impedance line) exceeds a programmable threshold the alarm is triggered.¹

According to the literature, 40% of reported alerts are not associated with heart failure deterioration.² There are several reports of inappropriate OptiVol alarm activation linked with pneumonia, pleural effusion, pacemaker pocket seroma, pericardial effusion or false calibration of the system because of baseline dehydration at the time of implantation.²⁻⁴ Therefore, anything that changes the thoracic fluid status may be reflected by a reduction in the intrathoracic impedance measurement. To the best of our knowledge this is the first case in the literature of inappropriate activation associated with advanced lung carcinoma. We speculate that the detection of tumour mass with local oedema, lymph flow disturbances, or disturbances of nodal architecture by metastases and pleural thickening caused the decrease in the intrathoracic impedance.

Our case suggests that intrathoracic impedance measurements should always be interpreted in the context of clinical findings, especially if heart failure deterioration cannot be clinically confirmed.

References

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