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CASE REPORT

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A case of Takotsubo cardiomyopathy after bronchoscopy

Maria Ferslev Friis[1*], Simon Teilmann Husted[1,2] and Ulla Møller Weinreich[1,3]

[1]Department of Respiratory Diseases, Aalborg University Hospital, Aalborg, Denmark
[2]Department of Psychiatric Diseases, Aalborg University Hospital, Aalborg, Denmark
[3]The Clinical Institute, Aalborg University, Aalborg, Denmark

Abstract

Takotsubo Cardiomyopathy (TCM), also known as Broken Heart Syndrome, is an acute stress-induced cardiomyopathy with transient cardiac failure. It is triggered by preceding extreme physical or psychological stress. The etiology remains unclear, however, pathophysiological activation of the adrenergic system causing catecholamine-induced myocardial dysfunction is proposed. Use/overuse of short acting beta-2-agonist (SABA) is a possible predisposing factor.

We report a case of TCM in a 64-year-old female after bronchoscopy with endobronchial ultrasound (EBUS) and transbronchial needle aspiration biopsy (TNAB). The case shows a possible delay from symptom onset to echocardiographic visibility of TCM on and underlines the importance of reviewing a patient's use of SABA prior to invasive procedures.

Keywords: *broken heart syndrome, pathophysiological activation, transbronchial needle aspiration biopsy*

Introduction

Bronchoscopy and other pulmonary invasive procedures are frequent procedures in Respiratory Medicine. Those examined are often patients with chronic lung diseases who frequently have multiple comorbidities. Bronchoscopy has a low incidence of complications and lethality is <0,1% [1]. Complications are hemoptysis, fever, infections, larynx spasm, anoxia, pneumothorax and arrhythmia [1,2]. A possible unrecognized complication to bronchoscopy is Broken Heart Syndrome/Takotsubo cardiomyopathy (TCM).

TCM is an acute stress-induced cardiomyopathy with transient cardiac failure characterized by left ventricular dysfunction, reduced ejection fraction, abnormal ECG, elevated coronary markers and absence of obstructed coronary arteries [3]. Patients are predominantly older women, presenting with symptoms and paraclinical findings resembling acute myocardial syndrome (ACS), triggered by preceding extreme physical or psychological stress [4]. The precise etiology and pathogenesis remains unclear, however, an abnormal activation of the adrenergic system and catecholamine-induced myocardial dysfunction are acknowledged as possible determinants [5].

Case report

A 64-year-old Caucasian woman underwent bronchoscopy with endobronchial ultrasound guided transbronchial needle aspiration biopsy (EBUS with TNAB) due to pathologically enlarged lymph nodes and a suspicious subpleural consolidation. The patient suffered from multiple chronic conditions; chronic obstructive pulmonary disease (COPD) with severe emphysema, type 1 diabetes mellitus, osteoporosis, hypertension and were formerly treated for rectal cancer. For COPD, the patient was treated with maintenance triple combination therapy (inhaled corticosteroids, long-acting muscarinic antagonists and long-acting beta-2-agonists) and short acting beta-2-agonist (SABA) as a reliever. Retrospectively, a review of the patient's prescriptions revealed redemption of seven prescriptions (a total of 1400 doses) of SABA during the preceding nine months, which accumulates to a daily use of >5 doses.

Bronchoscopy with EBUS was performed in local anesthesia with midazolame. After the first TNAB the patient desaturated to 79% despite provided with an oxygen flow of 14 liters. The procedure was immediately terminated, and the patient received intravenous flumenazile on suspicion of benzodiazepine overdosing and short acting β-2-agonist (SABA) inhalations due to auscultatory bronchospastic breathing. An electrocardiogram revealed supraventricular tachycardia with ST-depressions in the precordial leads, but acute echocardiography (Echo) performed bedside by a cardiologist showed a normal left ventricular ejection fraction (LVEF). It was concluded that the incidence was hypoxia-induced, and the patient was admitted to the Respiratory Ward for observation and oxygen therapy.

At the ward, the patient was normoxic, but heart rate was continuously around 100 beats per minute (BPM). Seven hours later the patient developed tachycardia (>140 BPM) and tachypnea. An arterial blood gas examination showed acute combined hypoxic and hypercapnic failure, and the patient was transferred to the intensive care unit (ICU). Echo was repeated and showed apical akinesia, ballooning and LVEF of 45%. Coronary markers were significantly elevated. An acute coronary arteriography (CAG) demonstrated normal coronary arteries. Based on these findings TCM was diagnosed. Shortly after the CAG, the patient's condition deteriorated, and she was intubated. After six days with anti-congestive treatment LVEF was >60%, judged by Echo. Ventilation was still needed due to respiratory failure and the patient remained in the ICU for 30 days. After further stabilization in the Respiratory ward the patient was discharged to rehabilitation almost 50 days after developing TCM.

Discussion

Takotsubo cardiomyopathy and β-2-agonist

The pathophysiology of TCM is not fully understood. High levels of serum-catecholamines triggered by physical/psychological stress may be a predisposing factor [5]. In addition, the use of SABA inhalations has been suspected of inducing TCM during a stressful event [5]. In rat models, the interaction between β-2-adrenoceptor-stimulation and the development of TCM demonstrated that low serum concentrations of catecholamines induced a positive inotropic response via stimulatory G-protein (Gs) of the β-2-adrenoceptor.5 Paradoxically, high serum concentrations induced a shift from stimulatory G-protein (Gs) to inhibitory G-protein (Gi) exerting a negative inotropic effect. Gi-signaling caused cardio depression with apical akinesia and ballooning (characteristic of TCM) possibly due to a higher density of β-2-adrenoceptors in apical than basal myocytes. The shift to Gi-signaling may be antiapoptotic and a cardioprotective response to reduce the cardiotoxic effects of high serum catecholamine levels. Therefore, use/overuse of β2A in a stressful event might exacerbate the inhibitory pathway, potentially inducing TCM [6].

The patient had a long-lasting considerable intake of SABA prior to the bronchoscopy, and the stress associated with undergoing bronchoscopy may have had elevated levels of catecholamines. This could be one determining factor for the TCM seen in this case.

Takotsubo cardiomyopathy and bronchoscopy

Flexible bronchoscopy is considered a relatively safe procedure with very few post-procedure pulmonary complications such as respiratory failure, pneumothorax and bronchial hemorrhage seen in less than one % of all procedures [1,2].

Cardiovascular complications, such as arrhythmia, acute ST-elevations infarction and myocardial ischemia also occur, however, the risk for cardiovascular complications to be low [7-9].

Bronchoscopy induces cardiac stress as elevated blood pressure, cardiac output and heart rate during the procedure [7]. Increased cardiac stress and myocardial oxygen demands may induce arrhythmia and ischemia in at-risk patients [9]. Although cardiac and pulmonary complications to bronchoscopy are well documented, TCM is only reported casuistically [10-15] and primarily in cardiac high-risk patients. The patients in this case had, despite multiple comorbidities, no known cardiac condition, however COPD, hypertension or malignant disease are also seen in these patients14,15. As of this date, no systematic studies have carried out on the prevalence of post-bronchoscopy TCM, however, as patients undergoing invasive procedures of the respiratory system are often patients at risk and TCM may well be a severely under-diagnosed condition.

Takotsubo cardiomyopathy and delayed diagnostic

In this case there was a 10-hour delay of TCM characteristics on echocardiography after symptom onset and ST-depressions on ECG. This is consistent with previous studies which have shown a delay from symptom onset to diagnosis varying from hours [16,17], to days and weeks [18]. However, the studies are all reports and, as in this case, it may illustrate a delay in diagnosis. In the present case we do, however, have echocardiographic reports from onset, after 7 hours, and during the days to recovery. This case report may therefore well reflect the natural cause of the disease.

Conclusion

In this case a patient with a high consumption of SABA developed TCM after bronchoscopy. SABA-use, combined with stress may be a risk for TCM. Echo signs of the disease developed after clinical signs of TCM. TCM should therefore not be ruled out in at-risk patients with acute cardiac symptoms in relation to bronchoscopy.

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*Correspondence: Maria Ferslev Friis, Department: Department of Respiratory Diseases, Aalborg University Hospital, Aalborg, Denmark, Tel: (+45) 28708855; E-mail: mffriis@hotmail.com

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