Anterior STEMI: Induced By Puffing On An Atypical Offender

Justin Harrell, DO; Heather Hesselson PharmD BCCP; Adrian Messerli, MD
University of Kentucky

Introduction

Selective beta-2 agonists such as albuterol are used primarily for bronchodilation and are first line therapies for symptomatic relief in chronic obstructive lung disease (COPD).

Given their local administration to the airways via metered-dosed inhalers or nebulizers systemic side effects are rare but the most common of which are anxiety, tachycardia and tremor.

A rare side effect of systemic absorption of a beta-2 agonist is myocardial ischemia, and aberrant infarction.

The mechanism of beta-2 agonist myocardial ischemia may be multifactorial.

Case Report

A 70-year-old man with a past medical history of COPD and tobacco use presented to the cardiac catherization laboratory with an anterior ST-elevation myocardial infarction.

The patient reported a significant history of upper respiratory symptoms with worsening in the last week. He woke from sleep with severe congestion for which he took a nebulizer treatment with albuterol 0.083%. Shortly after the nebulizer treatment he became diaphoretic and developed substernal chest pain with nausea and vomiting.

Angiography noted severe lesions in both the left anterior descending (LAD) coronary artery and the circumflex (LCx) coronary artery (Figure 1). A drug eluting stent was placed in the LAD and the patient was given intracoronary nitroglycerin. Importantly, the lesion in the LCx completely resolved, which raised suspicion for coronary vasospasm. (Figure 3)

Review of the patient's medication history revealed only albuterol as a potential culprit for vasospasm which coincided with the patient's timing of symptoms. Upon further questioning the patient reported having similar substernal chest pressure with previous albuterol nebulizer use.

The patient was discharged on hospitalization day two and was noted to have a left ventricular ejection fraction of 50%.

The patient was counseled on proper use of inhalers and his albuterol was discontinued with continued therapy of inhaled corticosteroid and long-acting muscarinic antagonist. He was also discharged with nitroglycerin and instructed to use if pain returns.

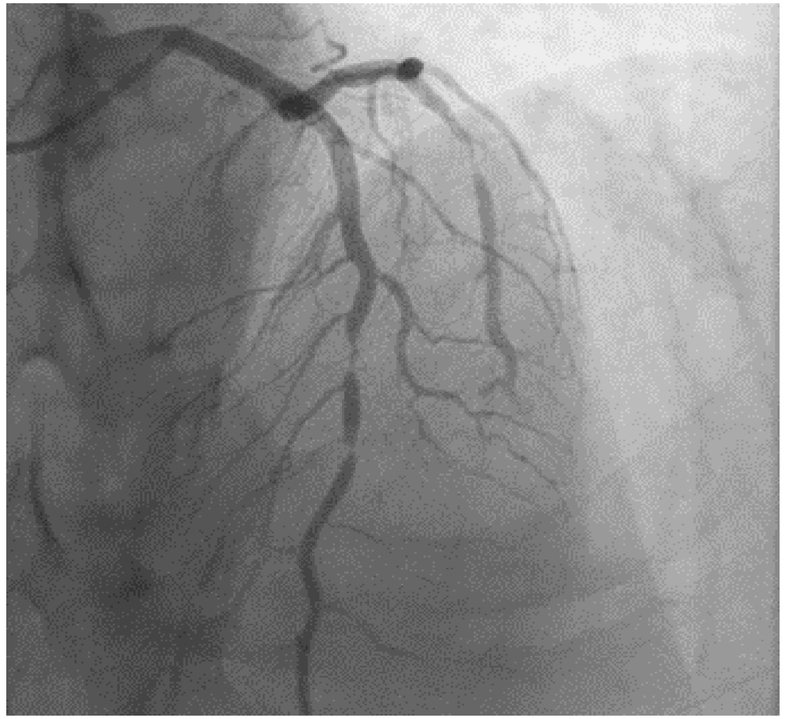

Image 1 revealing significant stenosis of the LAD and LCx

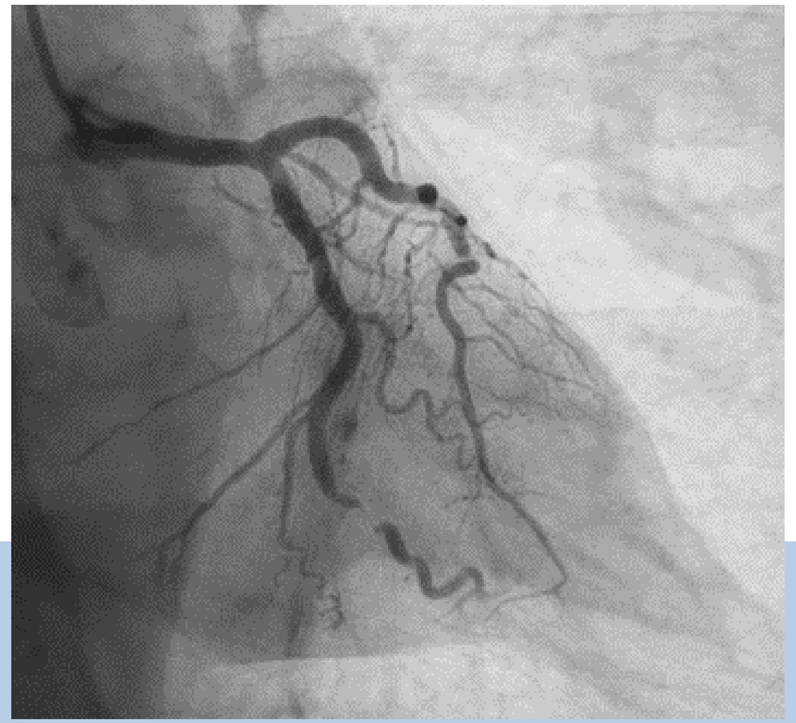

Image 2 Lesion of the LCx following PCI to the LAD

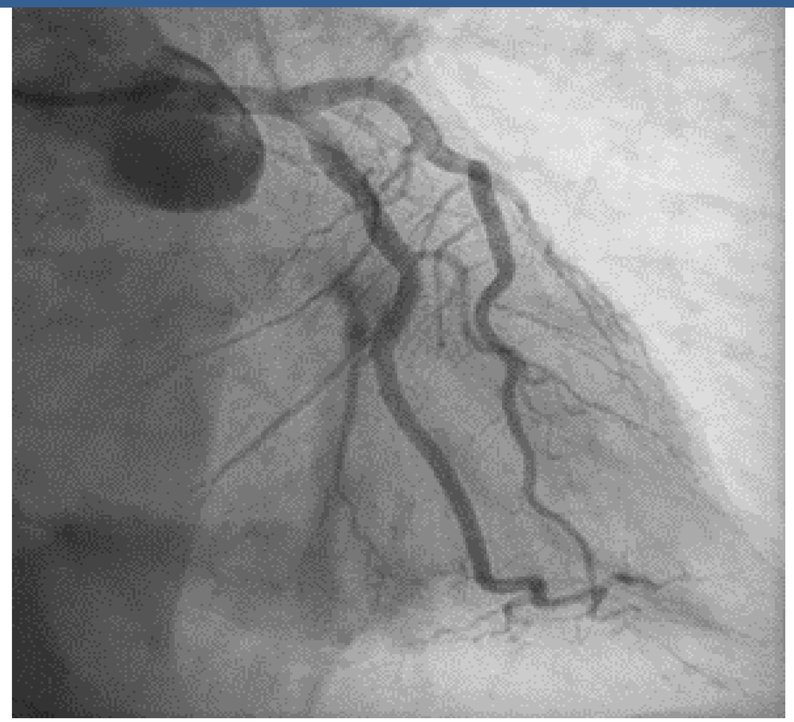

Image 3 Post nitroglycerin administration with resolution of the LCx lesion

Discussion

Coronary vasospasm has also been observed as a cause for ischemia or infarction due to beta-2 agonist use. This specific mechanism is not clearly elucidated. Large coronary arteries have alpha-1 receptors which could induce vasospasm but cross-reactivity to alpha receptors from albuterol have not been previously described.

Another possible mechanism is local inflammation that may be present during acute COPD exacerbations which increases the release of interleukin-1B, histamine and serotonin which could induce local hyper reactivity.

As in the case we describe and in other instances in the literature, coronary vasospasm may be diffusely seen in multiple coronary arteries and might suggest a heightened role of the autonomic nervous system exacerbated by systemic beta-2 stimulation.

Given the described rare side effect of myocardial injury due to coronary vasospasm, it is important for patients with COPD to be educated on appropriate rescue beta-2 agonist intake, and the risk of overuse of albuterol. Clinicians should also consider vasospasm as a mechanism for angiographically identified coronary stenosis with resultant ST-elevation myocardial infarction. In this instance the infarct was not potentiated by long-standing inhalation of tobacco, but rather short-term inhalation of albuterol!