Acute coronary syndrome presenting after pseudoephedrine use and regression with beta-blocker therapy

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Pseudoephedrine, a common ingredient in cold relief drugs, dietary supplements and Chinese herbal tea, has potent sympathomimetic effects, impacting the cardiovascular system. The chemical properties and clinical effects of pseudoephedrine are similar to those of ephedrine, and its main effect is caused by the release of endogenous norepinephrine. A 45-year-old man who presented with chest pain following ingestion of pseudoephedrine-containing prescription medication is described. The patient was initially diagnosed with inferior myocardial infarction based on an electrocardiogram, and intravenous metoprolol was started pending coronary artery angiography. Metoprolol reversed the ST segment elevation and relieved the symptoms, and coronary angiography showed normal coronary arteries. The present case highlights beta-blocker therapy as part of an initial intervention of pseudoephedrine-related cardiac symptoms.

Key Words: Beta-blocker; Metoprolol; Myocardial infarction; Pseudoephedrine

A major cause of ST segment-elevation myocardial infarction (STEMI) is the rupture of atherosclerotic plaque and thrombus formation (in 95% of cases), resulting in impairment of the oxygen supply-demand balance of the cardiac muscle. In the remaining non-sclerotic STEMI cases, the etiologies are varied and include coronary artery dissection, embolism, unbalanced oxygen supply-demand to the cardiac muscle, coronary artery spasm and, especially in young patients, addiction to drugs such as cocaine, amphetamines and derivatives (1). Clinical manifestations, and laboratory and electrocardiogram (ECG) changes of nonsclerotic STEMI are indistinguishable from STEMI caused by atherosclerotic disease. The prognosis of STEMI patients presenting without significant coronary atherosclerosis is better than patients with significant atherosclerotic lesions (2).

We present a patient admitted to the emergency room (ER) with STEMI 30 min after ingestion of a pseudoephedrine-containing prescription medication. Interestingly, the administration of the intravenous beta-blocker, metoprolol, caused the resolution of chest pain and ST segment elevation. The coronary angiography showed normal coronary arteries, suggesting that, at least in patients with no atherosclerotic disease history, beta-blocker therapy as part of an initial intervention might resolve chest pain and ST segment elevation caused by ingestion of pseudoephedrine-containing medication.

CASE PRESENTATION

A 45-year-old man was admitted to the regional research and training hospital ER with the chief complaint of chest pain. He stated that he

Syndrme coronarien aigu aprs l'emploi de pseudoéphdrine, traité avec succès par bta-blocage

Le pseudoéphdrine, un ingrédient que l'on trouve couramment dans les médicaments contre le rhume, les suppléments alimentaires et le thé chinois, exerce de puissants effets sympathomimétiques qui se répercutent sur l'appareil cardiovasculaire. Les propriétés chimiques et les effets cliniques de la pseudoéphdrine sont semblables à ceux de l'éphdrine et son principal effet est imputable à la libération de noradrénaline endogène. On décrit ici le cas d'un homme de 45 ans qui a consulté pour douleurs rétro-sternales aprs avoir pris un médicament d'ordonnance renfermant de la pseudoéphdrine. Le patient a tout d'abord reçu un diagnostic d'infarctus du myocarde inférieur sur la base de l'électrocardiogramme et du métoprolol a été débuté par voie intraveineuse en attendant la coronarographie. Le métoprolol a corrigé l'élévation du segment ST et soulagé les symptômes, et la coronarographie a révélé des artères normales. Ce cas illustre l'utilité du bta-blocage dans le cadre d'une intervention initiale pour corriger les symptômes cardiaques liés à la pseudoéphdrine.

was in good health until one week before when he developed nonproductive cough without dyspnea. The night before admission, he started to experience generalized muscle and joint pain, and 30 min before admission, he ingested a prescription cold medication containing 500 mg paracetamol, 4 mg chlorpheniramine and 60 mg pseudoephedrine. He then started to feel a crushing chest pain, radiating to the back with left arm hypoesthesia. He denied any chest trauma.

His medical history was consistent with 30 years of smoking. The patient denied the use of narcotics and stated that he never had any chest pain or dyspnea (with or without exertion) in the past. Of note, the patient had a physically demanding job at a car wash centre.

On arrival to the ER, the patient was mildly hypotensive with 89 mmHg systolic and 59 mmHg diastolic arterial pressure. His heart rate was 105 beats/min with a respiratory rate of 12 breaths/min. The head, eyes, throat and neck, as well as cardiovascular and pulmonary examinations appeared normal.

While obtaining the intravenous line and initiating oxygen, the ECG showed a 2 mm ST segment elevation in leads II, III and aVF, and a 2 mm ST segment depression between leads V2 and V6 (Figure 1). Posterior and right-sided leads appeared normal.

Therapy was started with an initial dose of 300 mg of oral acetylsalicylic acid and the patient was given 500 mL of saline. Cardiac catheterization for coronary artery angiography was scheduled. The patient became normotensive after saline administration and was given 5 mg of metoprolol intravenously pending coronary artery angiography. The chest pain ceased soon after metoprolol treatment

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Figure 1) The first electrocardiogram after admission of the patient to the emergency department

and the ECG showed resolution of ST segment elevation. While on the way to the cardiac catheterization unit, first laboratory results came with normal levels of total creatine kinase, MB fraction and troponin I levels. Cardiac catheterization showed normal coronary arteries with normal filling rate and an ejection fraction of 55%.

The patient's follow-up troponin I value peaked at 0.328 ng/mL (cutoff value for troponin I was 1.00 ng/mL) and creatine kinase and creatine kinase-MB values were within the normal range. After 24 h of observation, the patient was discharged with oral acetylsalicylic acid therapy and was advised to quit smoking. The echocardiography and Holter ECG, as part of an outpatient follow-up plan, were within normal limits. The patient remained free of chest pain at his one-month follow-up examination.

DISCUSSION

We have described a patient with STEMI, which started after ingestion of pseudoephedrine-containing cold medication. After the administration of a beta-blocker (metoprolol), the patient's chest pain was resolved with normalization of ECG changes. Coronary angiography showed normal coronary arteries. The patient's peak troponin I level of 0.328 ng/mL suggested minimal cardiac myocyte necrosis.

Pseudoephedrine is a stereoisomer of ephedrine and is commonly found in over-the-counter cold medication, causing relief of upper respiratory infection symptoms by local vasoconstriction and reduction in edema. Pseudoephedrine and derivatives have weak agonist alpha- and beta-adrenergic receptor activity but their main effects are

caused by the release of endogenous noradrenaline from storage (2); thus, these chemicals act as sympathomimetic agents, leading to increases in heart rate, blood pressure and cardiac output. These effects can be critical for patients with risk factors for atherosclerotic heart disease and for geriatric patients. Although norepinephrine's alpha-adrenergic effects are more pronounced, beta-adrenergic effects also exist.

Following reported cases of heart palpitations, tremors and insomnia in patients ingesting ephedrine-containing dietary supplements and medications, the US Food and Drug Administration implemented a ban on ephedrine-containing supplements because of increased risk for myocardial infarction and cerebrovascular events (3). Because of the adverse effects of pseudoephedrine and its derivatives, the number of cases presenting to the ER with cardiac symptoms after ingestion of medications containing these chemicals is increasing every year.

In the literature, patients with STEMI have been described after the ingestion of pseudoephedrine-containing medication, mainly for upper respiratory system-related symptoms (4,5). However, none of the reported cases so far used an intravenous beta-blocker as part of an initial therapy which, in our case, improved symptoms and regressed ST segment elevation.

We believe that in young patients with few or no risk factors who present with STEMI, it is imperative to question them regarding ingestion of pseudoephedrine-containing medications or other stimulants. The absence of atherosclerotic coronary arteries on the coronary angiogram of the present patient suggests that the adrenergic effects of

pseudoephedrine-containing medication are the main pathology triggering this clinical picture. Administration of an intravenous beta-blocker reversed the vasospasm, relieved pain and likely limited the area affected. The present case highlights the potential danger of the use of pseudoephedrine in young, healthy people but more importantly,

beta-blockers may play a crucial role in reversing the effects of ephedrine derivatives on coronary arteries. Clinicians must be aware that beta-blockers should be considered as part of the initial therapy to relieve the symptoms and potentially reverse or limit the effect of pseudoephedrine on cardiac muscle.

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