

Acute myocardial infarction type 2 without ST elevation following administration of butylbromide hyoscine

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Abstract

We report the case of a woman in her eighth decade who developed atrial fibrillation and a type 2 non–ST-elevation myocardial infarction (type 2 NSTEMI) after intravenous administration of 20 mg hyoscine butylbromide as premedication for colonoscopy. Coronary angiography showed no obstructive lesions.

Main findings: *Onset was sudden, 2 minutes after administration of hyoscine butylbromide, with oppressive retrosternal chest pain radiating to the jaw, dyspnea, diaphoresis and palpitations. Physical examination revealed marked hypotension and tachycardia. Electrocardiogram demonstrated new-onset atrial fibrillation without ischemic ST–T changes. High-sensitivity troponin T was significantly elevated and transthoracic echocardiography showed segmental wall-motion abnormalities of the basal anterolateral wall.*

Diagnosis, interventions and outcome: *A diagnosis of type 2 NSTEMI secondary to unstable atrial fibrillation with rapid ventricular response—likely triggered by an adverse reaction to hyoscine butylbromide—was made. The patient underwent coronary catheterization without evidence of coronary stenosis. Management included hemodynamic support with intravenous fluids, beta-blocker therapy, statin therapy, initial antiplatelet therapy and anticoagulation. She had a favorable clinical course and was discharged on a beta-blocker and oral anticoagulation.*

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Conclusions:

- *Acute myocardial infarction may result from mechanisms other than atherosclerotic coronary obstruction, notably supply–demand mismatch.*
- *Hyoscine butylbromide, although commonly associated with xerostomia, constipation and nausea, can be associated with serious cardiovascular adverse events that require prompt recognition and management.*
- *Retrosternal chest pain requires a broad differential diagnosis (including pulmonary embolism and acute coronary syndromes); an accurate and timely diagnostic approach is essential.*

Infarto Agudo de Miocardio Tipo 2 sin Elevación del ST tras administración de butilbromuro de hioscina: reporte de caso

Resumen

Se presenta el caso de una mujer en la octava década de la vida que desarrollo episodio de fibrilación auricular e infarto agudo de miocardio sin elevación del ST (IAMSEST) tipo 2 tras la administración intravenosa de 20 mg de butilbromuro de hioscina como premedicación para colonoscopia sin hallazgos en la arteriografía coronaria.

Hallazgos principales: *Fue de inicio subitito 2 minutos posteriores a la aplicación del butil bromuro hioscina iniciado como un dolor torácico retroesternal opresivo irradiado a mandíbula, disnea, diaforesis y palpitaciones. Al examen físico con hipotensión marcada y taquicardia. A los paraclínicos con evidencia de un electrocardiograma con fibrilación auricular de novo, no signos de isquemia. Troponina T ultrasensible con aumento significativo y ecocardiograma con trastornos segmentarios de la contractilidad basal anterolateral.*

Diagnósticos, intervenciones y resultados: *Se considero un IAMSEST tipo 2 secundario a una fibrilación auricular con respuesta ventricular rápida inestable, secundario a una reacción adversa al butilbromuro de hioscina. Fue llevada a cateterismo coronario sin evidencia de estenosis coronaria por lo que se consideró manejo con líquidos endovenosos, betabloqueo, estatina, antiagregación inicial y anticoagulación. Adicionalmente por los hallazgos y la evolución favorable egreso con betabloqueador y anticoagulación oral.*

conclusión:

- *El infarto agudo de miocardio es una condición clínica que puede ser ocasionada por causas diferentes a la enfermedad coronaria, como cambios en la oferta y demanda de oxígeno miocárdico.*
- *El butilbromuro de hioscina tiene efectos adversos que son más frecuentes como lo pueden ser xerostomía, constipación y nauseas, sin embargo, los efectos adversos cardiovasculares pueden ser presentados y tienen que ser manejados de manera adecuada.*

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- *El dolor torácico retroesternal es un síntoma que puede ser atribuido a diferentes causas como lo es el tromboembolismo pulmonar o el infarto agudo de miocardio, a razón de esto es esencial el manejo y enfoque diagnóstico acertado.*

Palabras clave: *Infarto, hioscina, electrocardiograma, angiografía, adversos, troponina.*

Infarto agudo do miocárdio tipo 2 sem elevação do ST após administração de brometo de butil-hioscina: relato de caso

Resumo

Relatamos o caso de uma mulher na oitava década de vida que desenvolveu fibrilação atrial e infarto agudo do miocárdio sem supradesnivelamento do segmento ST (IAM-SSST) tipo 2, após administração intravenosa de 20 mg de butilbrometo de hioscina como premedicação para colonoscopia. A angiografia coronária não revelou lesões obstrutivas.

Achados principais: *O início foi súbito, 2 minutos após a administração do butilbrometo de hioscina, manifestando-se por dor torácica retroesternal opressiva irradiada à mandíbula, dispneia, diaforese e palpitações. Ao exame físico havia hipotensão marcada e taquicardia. O eletrocardiograma evidenciou fibrilação atrial de novo sem alterações isquêmicas de ST–T. A troponina T de alta sensibilidade apresentou elevação significativa e o ecocardiograma transtorácico mostrou alterações segmentares do movimento parietal na região basal anterolateral.*

Diagnóstico, intervenções e evolução: *Foi estabelecido diagnóstico de IAMSSST tipo 2 secundário a fibrilação atrial com resposta ventricular rápida instável, provavelmente desencadeada por reação adversa ao butilbrometo de hioscina. A paciente foi submetida a cateterismo coronário sem evidência de estenose coronária. A conduta incluiu suporte hemodinâmico com reposição volêmica endovenosa, betabloqueador, estatina, terapia antiplaquetária inicial e anticoagulação. Evoluiu favoravelmente e recebeu alta com betabloqueador e anticoagulação oral.*

Conclusões:

- *Infarto agudo do miocárdio pode decorrer de mecanismos distintos da doença aterosclerótica coronariana, como desequilíbrio entre oferta e demanda de oxigênio miocárdico.*
- *Butilbrometo de hioscina, embora frequentemente associado a efeitos adversos como xerostomia, constipação e náuseas, pode provocar eventos cardiovasculares graves que exigem reconhecimento e manejo imediato.*
- *A dor torácica retroesternal possui um amplo diagnóstico diferencial (incluindo tromboembolismo pulmonar e síndromes coronarianas agudas); portanto, uma abordagem diagnóstica rápida e precisa é essencial.*

Introduction

Hyoscine butylbromide is an antimuscarinic drug indicated for muscle spasms in the gastrointestinal tract [1]. Its injection can cause serious cardiovascular adverse effects in patients with underlying heart disease, and there are even international reports of associated deaths [2,3]. These include AMI, as it can cause type 2 AMI in a few cases. Up to 26% of type 2 AMI cases are secondary to cardiac arrhythmia, mainly tachyarrhythmias such as atrial fibrillation [4]. Hyoscine butylbromide has been prescribed as a pre-colonoscopy treatment to reduce discomfort during the procedure [5,6]. This report seeks to provide clinical evidence on a rarely described adverse cardiovascular reaction associated with hyoscine butylbromide, contrasting the case with existing literature and demonstrating possible pathophysiological mechanisms, providing recommendations for evaluation and management.

Case presentation

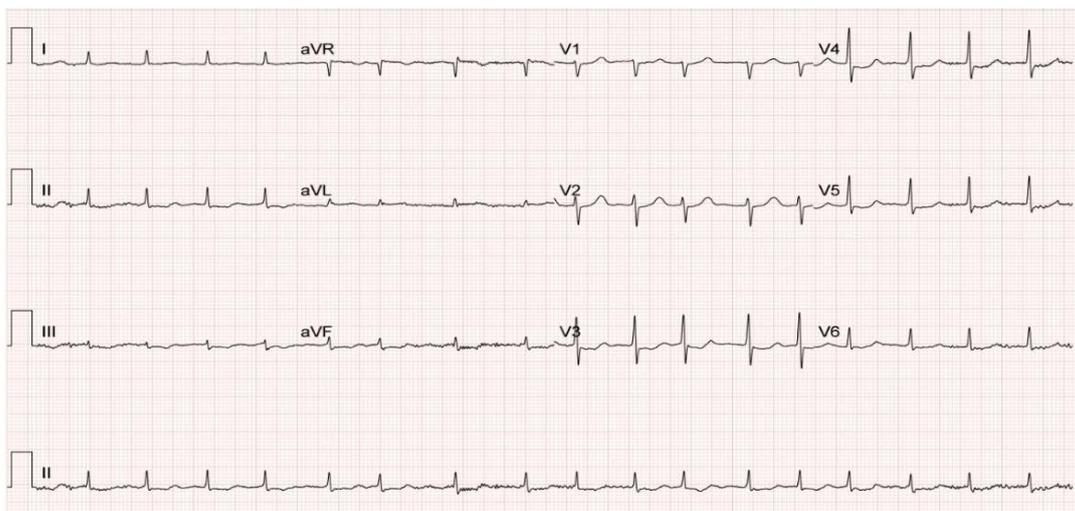
We report a case of collapsing atrial fibrillation with hypotension and type 2 acute myocardial infarction [7] following intravenous administration of hyoscine as premedication for colonoscopy. The patient was a woman in her 80s who attended for a scheduled colonic polypectomy by colonoscopy. Prior to the procedure, 20 mg

of hyoscine butylbromide was prescribed intravenously. Two minutes after administration, she presented with sudden-onset chest pain, oppressive, radiating to the jaw, associated with dizziness, dyspnoea, diaphoresis, and palpitations. On physical examination, she was alert, hypotensive, and tachycardic, so volumetric resuscitation with crystalloid intravenous fluids was initiated with a slight response, and she was transferred to the emergency department. She was admitted to the resuscitation room with the following physical examination findings: Glasgow Coma Score 15/15, blood pressure 56/36 mmHg, TAM 41 mmHg, heart rate 105 bpm, respiratory rate 20 rpm, temperature 35.5°C, oxygen saturation 93%, Fio2 24%.

Electrocardiogram shows atrial fibrillation with rapid ventricular response, with no electrocardiographic traces suggestive of acute coronary injury or myocardial necrosis. (Figure 1)

Figure 1. Electrocardiogram (ECG) — Date: [10/11/2021]. ECG recorded after intravenous administration of 20 mg of hyoscine butylbromide. Shows de novo atrial fibrillation with rapid ventricular response, without ST segment elevations or depressions or pathological Q waves. Acquisition parameters:

Standard 12-lead leads, speed 25 mm/s, calibration 10 mm/mV.



The patient is considered to have a diagnostic impression of cardiac chest pain, type 2 acute myocardial infarction without ST segment elevation (GRACE Score: 165 points Killip and Kimbal II TIMI Score: 1 point), de novo atrial fibrillation with rapid ventricular response secondary to adverse effect of hyoscine butylbromide (CHA2DS2 VASC: 4 points HASBLED: 2 points)

Management with intravenous Ringer's lactate is indicated, and metoprolol 50 mg, atorvastatin 80 mg, acetylsalicylic acid 100 mg, clopidogrel 75 mg, and enoxaparin 60 mg subcutaneously are administered orally.

Table 1. Paraclinical report

| Paraclinical tests | Interpretation |
|----------------------------|---|
| Blood glucose measurement | 70 mg/dl |
| Complete blood count | Normal |
| Serum creatinine | 0.9 mg/dl |
| Ultra-sensitive troponin T | 0.076 positive (reference value less than 0.014) |
| D-dimer | 11.221 positive (reference value 500) |
| Arterial blood gases | Metabolic acidaemia, without oxygenation disorder or hyperlactataemia |
| Chest X-ray | Bilateral parahilar peribronchovascular interstitial opacities. Left basal atelectasis. Cardio-mediastinal silhouette magnified by projection. Rest normal. |

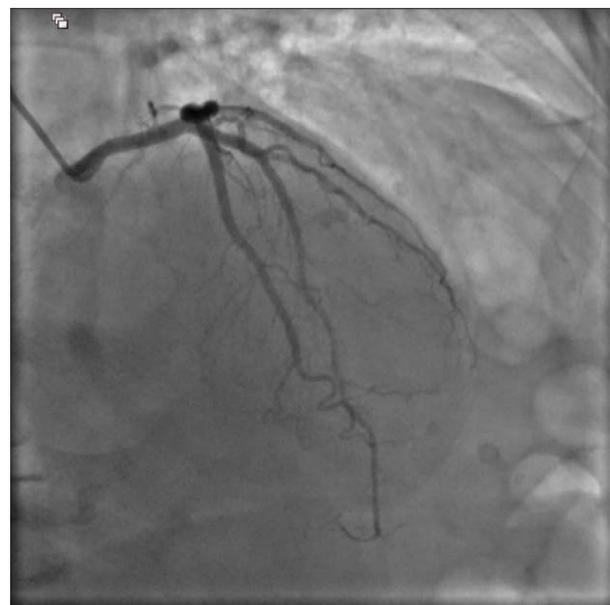
Due to elevated D-dimer levels and the sudden onset of dyspnoea and chest pain, pulmonary thromboembolism was suspected. A pulmonary artery angiography, troponin test, cardiology assessment, and transthoracic echocardiogram were ordered, yielding the following results:

Table 2. Paraclinical report

| Paraclinical tests | Interpretation |
|-------------------------------|--|
| Ultra-sensitive troponin T | 0.606 delta of 690% |
| Pulmonary artery angiography | Negative for TEP |
| Trans-thoracic echocardiogram | Left ventricle of normal size with segmental disorders of ANTEROLATERAL BASAL contractility and preserved ventricular systolic function, LVEF 55%. |

She was transferred to the Coronary Care Unit, where emergency percutaneous coronary intervention (PCI) was performed using the IAMSEST technique, with coronary arteriography showing no evidence of stenosis or obstruction (Figure 2), without the need for balloon angioplasty or stenting. The patient's outcome was favourable, and she was discharged on beta-blocker therapy and oral anticoagulation.

Figure 2. Coronary arteriography — Date: [10/11/2021]. Coronary arteriography performed after the diagnosis of AMI. Coronary projections showing absence of angiographic obstructive stenosis in the main epicardial arteries. No angioplasty or stenting was performed.



Discussion

Hyoscine butylbromide contains a nitrogen molecule with four different bonds to various chemical groups, making it a quaternary ammonium derivative of hyoscine with a rapid-onset anticholinergic effect [8]. The mechanism of action of anticholinergics is non-selective competitive antagonism of muscarinic acetylcholine receptors [9]. Five subtypes of muscarinic receptors have been identified: M1 receptors are found in the autonomic ganglia, salivary and gastric glands; M2 receptors are found predominantly in the heart; M3 receptors are involved in smooth muscle contraction, acetylcholine-mediated vasodilation, control of secretory glands and ocular accommodation; their activation induces emesis and their antagonism has antiemetic properties [10]. The M4 and M5 subtypes, predominantly expressed in the CNS, have actions at various levels [11].

In 2017, the Medicines and Healthcare products Regulatory Agency in the United Kingdom reported nine cases of patients who died after receiving an injection of BB Hyoscine. In most cases, the adverse reaction was reported as cardiac arrest. It also reported that hyoscine butylbromide injection can cause adverse effects including tachycardia, hypotension, and anaphylaxis. These effects may be more severe in patients with underlying heart disease. Several reports have indicated that anaphylaxis is more likely to be fatal in patients with underlying coronary artery disease compared to those without [11].

In 2018, a case report was published in Neiva, Colombia, on the occurrence of Kounis syndrome, which is characterised by cardiac involvement secondary to an allergic response following the administration of hyoscine and dipyrone BB. In this case, the patient presented with chest pain accompanied by clinical manifestations of anaphylaxis. An electrocardiogram was performed, showing ST segment depression. [12] It is important to clarify that our patient had no signs or symptoms of anaphylaxis and also had a cardiac catheterisation report with no evidence of coronary lesions, which rules out type 1 acute myocardial infarction. No drug interactions or causes related to prescription, dispensing, suitability or administration that could have led to the reaction were identified. A thorough review of the literature was conducted, revealing that most existing reports are associated with colonoscopy under sedation, which would introduce a bias in determining the causality of the adverse effect given the drug interaction with the sedative. However, in this case, only hyoscine butylbromide was administered. To our knowledge, the above supports the hypothesis that this is the first reported case of a serious cardiovascular adverse effect caused by a single administration of hyoscine butylbromide, triggering collapsing atrial fibrillation with type 2 non-ST-segment elevation acute myocardial infarction in a patient with no underlying heart disease, and ruling out other causes.

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