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Case report

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Coronary Arteries Spasm After Administration of Atropine for Premedication

A.Yu. Shchedrina, A.Z. Khashukoeva, L.P. Prokhorova, K.A. Eruslanova, E.A. Rodnyanskaya, A.M. Shatov, A.F. Farzutdinov, N.F. Fathi, M.A. Fedin, K.O. Orunbekova, O.N. Tkacheva, S.R. Gilyarevsky✉

Cardiology Center
N.I. Pirogov Russian National Research Medical University, Russian Gerontological Research and Clinical Center
1-st Leonova Str. 16, Moscow, Russian Federation 129226

✉ **Contacts:** Sergey R. Gilyarevsky, Head of the Cardiology Center of the Russian Gerontological Research and Clinical Center, N.I. Pirogov Russian National Research Medical University. Email: sgilarevsky@rambler.ru

ABSTRACT We report a case of severe retrosternal pain in 28-year-old patient with no cardiovascular diseases in history, developed after intravenous administration of 0.5 atropine sulfate 0.1% solution for the purpose of premedication before the planned operation for endometrioid ovarian cysts and managed with nitrates spray. Taking into account the clinical picture, transient ischemic changes in the electrocardiogram and the association with the administration of atropine, it can be assumed that the patient had a spasm of the coronary arteries caused by the administration of atropine. There are only a very small number of descriptions of similar observations in the literature, which are also discussed in the article.

Keywords: atropine, coronary artery spasm, premedication

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Affiliations

Anna Yu. Shchedrina	Candidate of Medical Sciences, Head of the Department of Geriatric Cardiology, Russian Gerontological Research and Clinical Center, Federal State Autonomous Educational Institution of N.I. Pirogov Russian National Research Medical University; https://orcid.org/0000-0002-3981-4031, ashedrina@rambler.ru; 15%, data collection during intensive care unit stay
Asiyat Z. Khashukoeva	Doctor of Medical Sciences, Professor of the Department of Obstetrics and Gynecology, Federal State Autonomous Educational Institution of N.I. Pirogov Russian National Research Medical University; https://orcid.org/0000-0001-7591-6281, azk05@mail.ru; 13%, data collection
Lyubov P. Prokhorova	Obstetrician-gynecologist of the Gynecology Department of the Russian Gerontological Research and Clinical Center, Federal State Autonomous Educational Institution of N.I. Pirogov Russian National Research Medical University; prohorova_lp@rgnkc.ru; 10%, data collection
Kseniia A. Eruslanova	Candidate of Medical Sciences, Head of the Laboratory of Cardiovascular Aging, Russian Gerontological Research and Clinical Center, Federal State Autonomous Educational Institution of N.I. Pirogov Russian National Research Medical University; https://orcid.org/0000-0003-0048-268X, eruslanova_ka@rgnkc.ru; 9%, text preparation
Ekaterina A. Rodnyanskaya	Anesthesiologist and Resuscitator of the Department of Anesthesiology and Resuscitation of the Russian Gerontological Scientific and Clinical Center, Federal State Autonomous Educational Institution of N.I. Pirogov Russian National Research Medical University; rodnanskaya_ea@rgnkc.ru; 9%, data collection during intensive care unit stay

Aleksey M. Shatov	Anesthesiologist and Resuscitator of the Department of Anesthesiology and Resuscitation of the Russian Gerontological Scientific and Clinical Center, Federal State Autonomous Educational Institution of N.I. Pirogov Russian National Research Medical University; https://orcid.org/0009-0009-0969-3660, shatov_am@rgnkc.ru; 9%, data collection
Artur F. Farzutdinov	Head of the Department of Anesthesiology-Resuscitation and Intensive Care, Anesthesiologist and Resuscitator, Russian Gerontological Research and Clinical Center, Federal State Autonomous Educational Institution of N.I. Pirogov Russian National Research Medical University; https://orcid.org/0000-0002-7439-8280, farzutdinov_af@rgnkc.ru; 8%, data collection during intensive care unit stay
Nina F. Fathi	Anesthesiologist and Resuscitator of the Department of Anesthesiology and Resuscitation of the Russian Gerontological Scientific and Clinical Center, Federal State Autonomous Educational Institution of N.I. Pirogov Russian National Research Medical University; fathi_nf@rgnkc.ru; 7%, data collection during intensive care unit stay
Maksim A. Fedin	Cardiologist, Department of Geriatric Cardiology, Russian Gerontological Research and Clinical Center, Federal State Autonomous Educational Institution of N.I. Pirogov Russian National Research Medical University; fedin_ma@rgnkc.ru; 7%, data collection during the stay in the cardiology department
Kunduz O. Orunbekova	Resident, Russian Gerontological Research and Clinical Center, Federal State Autonomous Educational Institution of N.I. Pirogov Russian National Research Medical University; kunduz.orunbekova.kg@gmail.com; 6%, description of clinical observation
Olga N. Tkacheva	Doctor of Medical Sciences, Professor, Corresponding Member of the Russian Academy of Sciences, Director of the Russian Gerontological Research and Clinical Center, Federal State Autonomous Educational Institution of N.I. Pirogov Russian National Research Medical University; Chief Freelance Geriatrician of the Russian Ministry of Health; https://orcid.org/0000-0001-5451-2915, tkacheva@rambler.ru; 4%, article planning and editing
Sergey R. Gilyarevsky	Head of the Cardiology Center of the Russian Gerontological Research and Clinical Center, Federal State Autonomous Educational Institution of N.I. Pirogov Russian National Research Medical University of Ministry; https://orcid.org/0000-0002-8505-1848, sgilarevsky@rambler.ru; 3%, preparation for publishing and editing

ANS – autonomic nervous system
BP – blood pressure
ECG – electrocardiogram
EchoCG – echocardiography
EF – ejection fraction
HR – heart rate
LV – left ventricle

Clinical observation

Patient D., 28 years old, was hospitalized in the gynecology department of the N.I. Pirogov Russian Scientific Clinical Center of the Russian National Research Medical University of the Ministry of Health of the Russian Federation for a planned operation for endometrioid ovarian cysts. There was no history of heart disease.

In the operating room, 0.5 ml of 0.1% atropine sulfate solution was administered intravenously as a bolus to the patient for premedication.

Immediately after intravenous administration of atropine, intense burning and pressing retrosternal pain developed in a patient with irradiation to the neck, as well as headache. An increase in blood pressure (BP) to 140/100 mm Hg (with the patient's usual BP of

90/60 mm Hg) and the development of bradycardia were noted.

The electrocardiogram (ECG) showed sinus bradycardia with a heart rate (HR) of 38 beats/min, ventricular extrasystole of the bigeminy type, and downward depression of the *ST segment* up to 1.5–2.0 mm in leads II, III, *aVF* , and *V*4–*V*6 (Figure A).

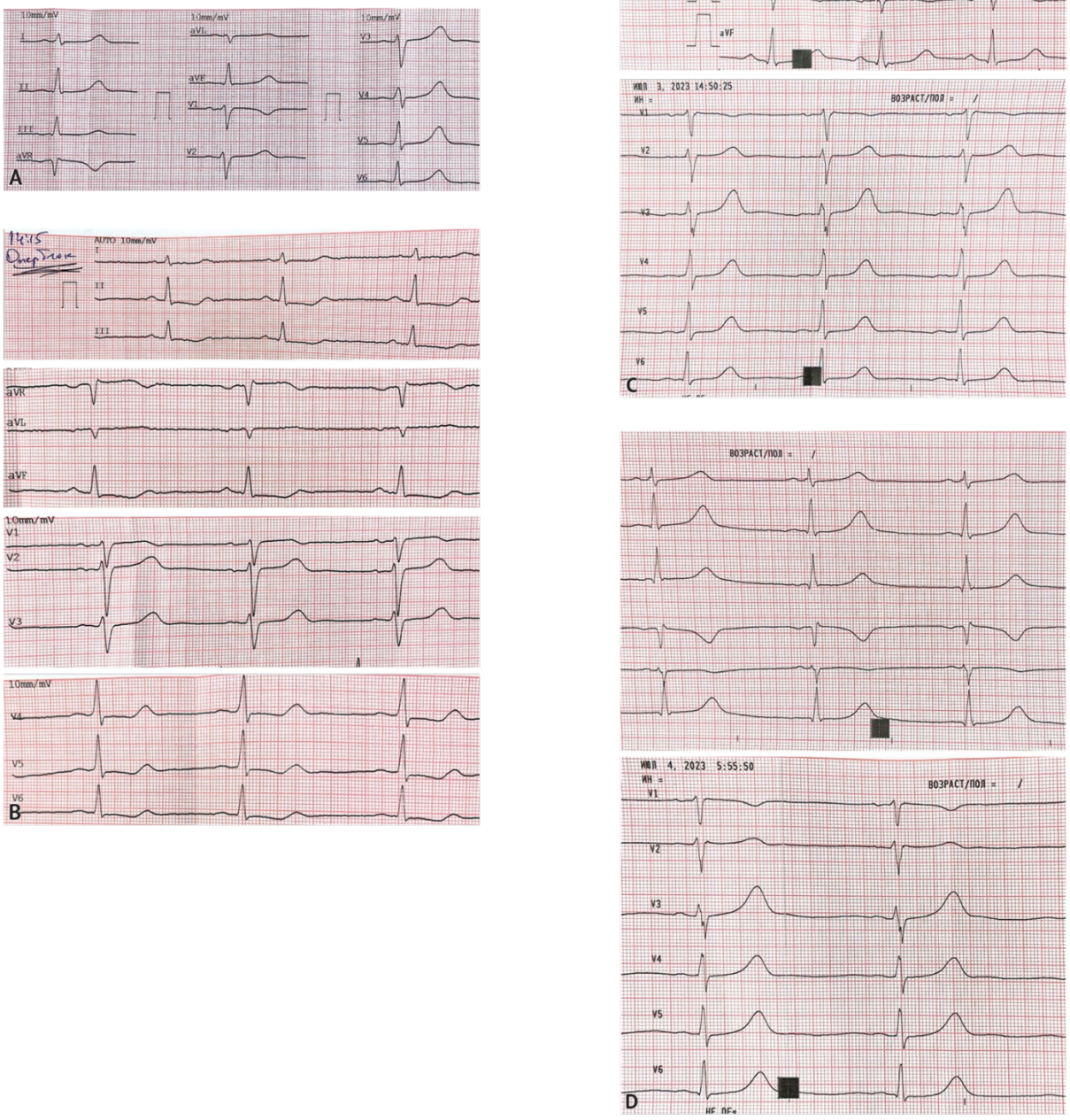

Figure. Electrocardiogram. A — upon admission to the hospital; B - after the administration of atropine and the development of chest pain in the operating room; C—in the intensive care unit; D—24 hours after the development of chest pain

Nitrospray was administered sublingually (1 dose), with a positive effect: a rapid significant reduction in chest pain and its cessation within 5 minutes were noted.

Due to the development of an attack of chest pain and an ischemic ECG change, a decision was made to refrain from surgery. The patient was transferred to the intensive care unit for dynamic observation.

According to the dynamic ECG data, the *ST segment returns* to the isoelectric line within an hour (see figure). According to echocardiography (EchoCG): there are no hypo- and akinesia zones, the ejection fraction (EF) of the left ventricle (LV) (according to Simpson) is 63%.

Troponin I concentration upon transfer to the intensive care unit was 0.04 ng/ml, 0.12 ng/ml in 2 hours, 0.04 ng/ml in 12 hours. Other laboratory parameters were normal.

The patient was transferred to the cardiology department for observation.

According to 24-hour ECG monitoring: 41 supraventricular and 1 ventricular extrasystoles were recorded.

According to the exercise test on a bicycle ergometer: tolerance to physical activity is high, no chest pain or ECG changes were noted, the test is negative.

In this case, performing coronary angiography to exclude hemodynamically significant stenosis of the coronary arteries could have been justified, but given the low pre-test probability of hemodynamically significant damage to the coronary arteries, as well as the patient's negative attitude (refusal) to invasive interventions, it was decided to refrain from performing it.

DISCUSSION

The development of chest pain and subsequent development of ischemic ECG changes, which were accompanied by a slight dynamic increase in the concentration of troponin I in the blood, was probably associated with the administration of atropine for premedication.

In the literature, we found two clinical observations of the development of chest pain after the administration of atropine [1, 2]. The first observation was presented by authors from South Korea. A 44-year-old woman was prepared for varicectomy and ligation of the communicating veins due to bilateral varicose veins. There were no indications of any aggravated anamnesis in the anamnesis [1]. At the time of hospitalization, no pathological changes were noted on the ECG, the heart rate was 57 beats/min with a normal duration of all intervals. At the time of delivery to the operating room, BP reached 134/72 mm Hg and HR 90–93 beats/min. The patient was given spinal anesthesia in the lateral decubitus position using a 25 *G Quincke needle* with 12 mg bupivacaine in the *L3 – L4 interval*. After that, the patient was placed in the supine position, in which she remained for 10 minutes. During this period, BP was 120/70 mmHg and HR was in the range of 82–85 bpm. Maximum sensory block was noted at the T8 level.

Midazolam 2.5 mg was then administered intravenously to achieve sedation and reduce anxiety. Blood pressure was stabilized at 110–90/65–45 mmHg. One hour after the start of the operation, the heart rate began to gradually decrease, since blood pressure remained stable, sedation was maintained, and a decision was made to continue observation without further assessment of the level of sensory block. Two hours after the start of spinal anesthesia, the heart rate decreased to 42–45 bpm, and therefore 0.5 mg atropine was administered intravenously. Within 5 minutes, the heart rate increased to 60 bpm. Soon, the ECG recorded a ventricular extrasystole followed by the development of ventricular tachycardia with blood pressure maintaining 120/60 bpm.

The patient complained of chest discomfort. At that time, the upper level of sensory block to cold reached T10. Esmolol 10 mg was administered intravenously. After 2–3 minutes, the ECG showed an increase in the *ST segment* with a heart rate of 62

bpm. The operation was stopped and the patient was transferred to the intensive care unit. An increase in the concentration of troponin in the blood to 6.38 ng/ml was noted, followed by a return to normal levels within 2 days.

Coronary angiography revealed stenosis of up to 40% in the middle segment of the anterior descending artery in the absence of other coronary artery lesions. According to echocardiography performed immediately after the operation, LVEF was 40% and hypokinesia was noted in the area of blood supply to the circumflex artery with signs of mild LV dysfunction or impaired myocardial relaxation. Based on the analysis of all clinical data, a diagnosis of ventricular tachycardia due to coronary artery spasm was established. After the administration of calcium antagonists, there were no clinical manifestations of angina, and the patient was discharged after 3 days [1].

As in our observation, the authors associated the development of coronary artery spasm with the administration of atropine. The second clinical observation [2] described the development of coronary artery spasm in a 10-year-old child after the administration of atropine (also for the purpose of premedication), but the spasm was due to the development of Kounis syndrome (acute coronary syndrome of allergic origin). So atropine could cause coronary artery spasm not due to its direct pharmacological action, but as a provoking factor that caused an allergic reaction with damage to the coronary arteries and their spasm.

Atropine is a powerful anticholinergic drug that is widely used for premedication to reduce secretion in the gastrointestinal tract and, accordingly, prevent aspiration of gastric contents during surgery. In addition, the administration of atropine can stop the development of bronchospasm and the development of severe bradycardia.

How can the development of coronary artery spasm be explained with the administration of atropine? The action of atropine is characterized by several phases: 1) the initial vagotonic effect; 2) an intermediate period of imbalance of the sympathetic and parasympathetic divisions of the autonomic nervous system (ANS) at different levels of the cardiac conduction system and; 3) the final period of prolonged blockade of the parasympathetic division [3]. Thus, like the authors of another clinical observation [1], we assume that the initial increase in the activity of the sympathetic division of the ANS immediately after the administration of atropine could cause coronary artery spasm.

Coronary arteries are innervated by both sympathetic and parasympathetic neurons, so changes in the activity of each of the ANS divisions can cause coronary artery spasm. It is believed that many factors can influence the development of coronary artery spasm in the perioperative period, including increased activity of the sympathetic division of the ANS, alkalosis, and the administration of calcium preparations [1, 4].

CONCLUSION

The clinical observation presented in the article confirms the possibility of developing coronary artery spasm immediately after the administration of atropine. Thus, anesthesiologists and cardiologists should remember that the administration of atropine, although extremely rare, can cause coronary artery spasm, and also be especially attentive to the complaints of patients who are administered atropine for premedication.

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