

Case report

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Acute myocardial infarction after botulinum toxin injection

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Case presentation

A 56-year-old male patient with a history of Friedreich ataxia was referred to the cardiac catheterization laboratory after electromechanical resuscitation due to ventricular fibrillation. Two hours before, trans-urethral intra-vesical botulinum toxin A injection (300 U) had been performed because of neurogenic bladder dysfunction. His past medical history was positive for hypertension and smoking, but negative for any cardiovascular event.

On admission, laboratory analysis had been unremarkable and baseline ECG showed incomplete right bundle branch block and T-wave inversions in leads V1–V2. Postresuscitation, new preterminal T-wave inversions were noted in the inferior leads and in leads I, and V3–6, along with troponin T elevation (0.72 µg/l). In the course, dynamic discrete ST segment elevations developed in leads V1–V3. Unfractionated heparin, acetylsalicylic acid and clopidogrel were administered for suspected acute coronary syndrome. Coronary angiography revealed thrombotic occlusion of the right coronary artery with a large thrombus extending from the proximal to the mid segment (Figure 1A). Of note, there were no coronary artery spasms observed; in particular, vascular tone was unchanged after intracoronary nitroglycerine administration. Immediate percutaneous coronary intervention was performed and the vessel reopened by means of thrombus aspiration using a diver catheter and the utilization of two drug-eluting stents (Biomatrix). Final angiographic

documentation revealed complete restoration of flow and normal left ventricular wall motions with preserved left ventricular systolic function (Figure 1B). The post-interventional course on the intensive care unit (ICU) was unremarkable and the patient was transferred to the regular ward 1 day after admission.

Discussion

We describe a case of acute myocardial infarction in a patient with Friedreich ataxia following intra-vesical botulinum toxin A injection.

Cardiac involvement is frequently observed in Friedreich ataxia. However, acute coronary syndromes have rarely been described in these patients. Typically, hypertrophic cardiomyopathy, which was excluded in our patient using echocardiography, and an abnormal repolarization phase and arrhythmias are detected.¹

Botulinum toxin A impedes neuromuscular transmission causing muscle weakening. It blocks acetylcholine release in nerve terminals by cleaving SNARE proteins, thereby preventing fusion of acetylcholine vesicles with the cell membrane. Botulinum toxin A is widely used for cosmetic applications, and in the treatment of muscle spasms, chronic pain syndroms or bladder dysfunction.²

Botulinum toxin A is assumed to have mainly local effects. However, systemic side effects have been described. Hence, botulinum toxin A might also affect vasoreactivity or interact with the

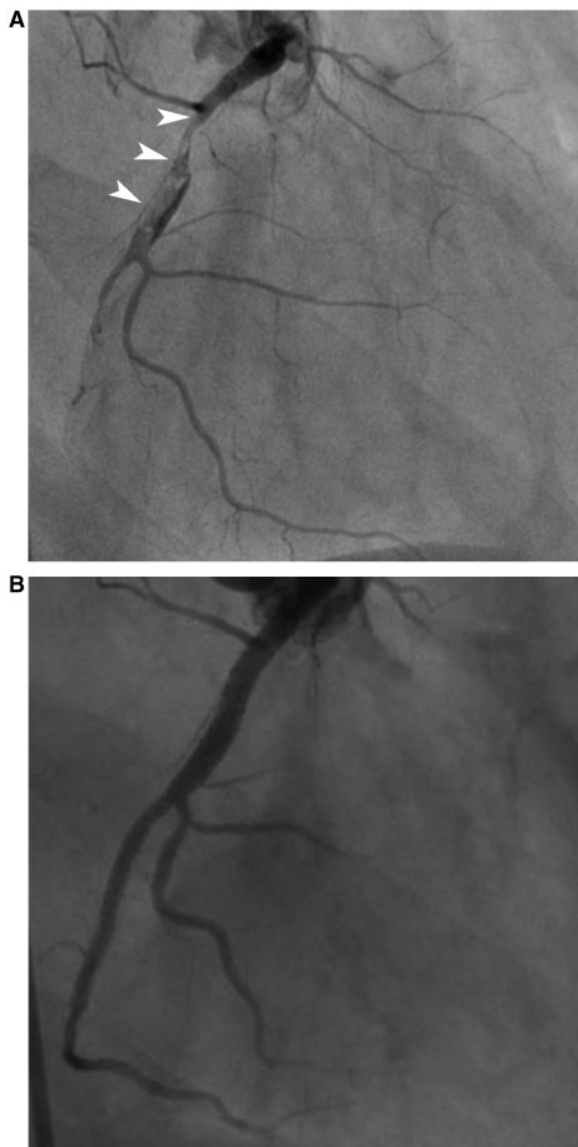


Figure 1. (A) Coronary angiography revealing thrombotic occlusion of proximal and mid segments of the right coronary artery with collateral flow from the left anterior descending artery. (B) Coronary angiography of the right coronary artery following percutaneous coronary intervention.

coagulation cascade, endothelial cells or platelets and in turn promote thrombus formation. Indeed, single cases of myocardial infarction, pulmonary embolism, and even death have been reported after botulinum toxin A injection.³ The effect of

botulinum toxin A on vasoreactivity is not fully understood. In rat, aortic rings suspended in organ chambers, contractions to potassium chloride (KCl) and norepinephrine were completely inhibited after incubation with botulinum toxin.⁴ Furthermore, in Sprague Dawley rats, femoral vessel diameter was increased after subcutaneous botulinum toxin injection.⁵ Hence, and in line with the coronary angiogram, vasospasms as the primary cause of the acute myocardial infarction appear unlikely in this patient. Rather, the extensive thrombus burden suggests a pro-thrombotic state. As we did not exclude a patent foramen ovale in our patient, paradoxical embolism cannot be ruled out completely. However, such events are rare and typically present with abrupt distal coronary occlusion suggestive of embolism on angiogram. In any case, pro-thrombotic effects of botulinum toxin A have not been described so far, both, *in vitro* and *in vivo*, and may be assumed.

The temporal coincidence of botulinum toxin injection and the onset of myocardial infarction in our patient suggest a causal relationship. Importantly, as botulinum toxin injections are widely performed, also in elderly patients with cardiovascular disease, and as acute coronary syndromes are serious complications, clinicians should be cautious using botulinum toxin A because of the risk of serious side effects and patients have to be monitored carefully after botulinum toxin injections.

Conflict of interest: None declared.

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