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# Is Botulinum Toxin A a Universally Safe Agent? A Case of Myocardial Infarction Following Injection

CASE REPORT

## INTRODUCTION

Botulinum toxin is a neurotoxin produced by the anaerobic bacterium *Clostridium botulinum*. Commonly referred to as "Botox," this agent is widely used for both medical and cosmetic purposes. Among its 7 serotypes, type A is the most commonly used form. When injected intramuscularly, it acts on nerve terminals to induce temporary paralysis, thereby inhibiting excessive muscle contraction. Various commercial formulations containing either type A or B are available on the market, each tailored for individual use and not interchangeable.[1]

Although popularly recognized for its role in cosmetic wrinkle reduction, botulinum toxin has long been employed therapeutically. It has demonstrated clinical efficacy in conditions such as cervical dystonia, strabismus, hyperhidrosis, chronic muscle spasms, laryngospasm, migraine, sphincter spasms, myofascial pain, and several movement disorders.[1] In this case report, the authors present a patient who developed myocardial infarction following a botulinum toxin A injection administered for cosmetic purposes.

## CASE REPORT

A 41-year-old woman with no known history of chronic illness presented to the emergency department with dizziness, fatigue, light-headedness, followed by a sensation of impending syncope and subsequent collapse. The patient reported no loss of consciousness, suggesting a presyncopal episode. Twelve hours prior, she had received a 100-unit intramuscular injection of botulinum toxin A into her forehead and periorbital region for cosmetic reasons. The product used was Dysport, which contains 500 units of botulinum toxin A per vial. The physician stated that the vial was diluted with 2.5 cc of saline, and 0.5 cc of the solution was injected, corresponding to an approximate dose of 100 units. Her medical history included a 15-pack-year smoking habit, but no previous cardiovascular events.

Electrocardiography performed in the emergency department revealed ST-segment elevation in leads D1 and aVL, accompanied by reciprocal ST depression in leads D2, D3, and aVF, consistent with lateral myocardial infarction (Figure 1). A high-sensitivity troponin I level was elevated at 170 µg/L. The patient was immediately loaded with 300 mg of acetylsalicylic acid and 180 mg of ticagrelor. She was then urgently transferred to the catheterization laboratory.

Right coronary angiography performed with a Judkins-4 catheter revealed no abnormalities. Subsequently, the left main coronary artery was cannulated with a Medtronic Launcher guide catheter. Angiographic imaging identified a 70-80% hazy lesion in the proximal segment of the circumflex artery (Figure 2A). The lesion was crossed with a floppy guidewire, followed by the implantation of a 4.0 × 23 mm everolimus-eluting stent (Figure 2B). Final angiography confirmed successful reperfusion with preserved left ventricular wall motion and systolic function (Figure 2C).

The patient was admitted to the coronary intensive care unit for further monitoring. Transthoracic echocardiography revealed a preserved ejection fraction of

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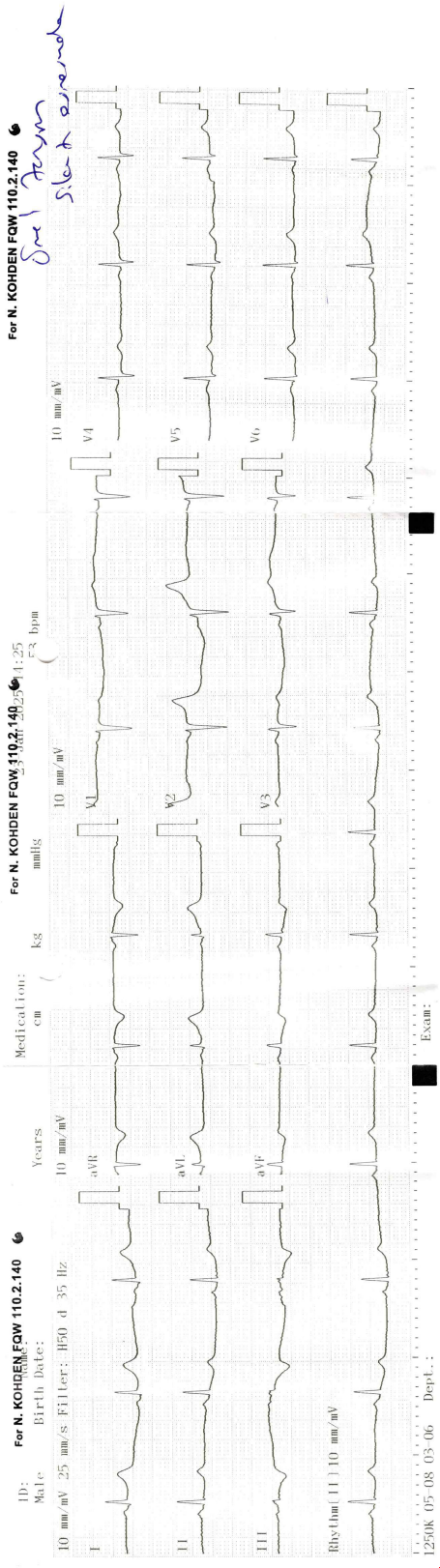


**Figure 1. The patient's electrocardiogram at the time of hospital admission.**

60%. A repeat ECG showed resolution of ST-segment elevations. She remained hemodynamically stable and was discharged after 48 hours of observation.

## DISCUSSION

The cosmetic application of botulinum toxin A is gaining increasing popularity. The neurotoxin functions by blocking the release of acetylcholine at presynaptic neuromuscular junctions, resulting in flaccid paralysis of skeletal muscle. Although its effects are considered to be predominantly local, systemic adverse events (AEs) have also been reported. It is therefore hypothesized that botulinum toxin A may influence vascular reactivity or promote thrombogenesis by interacting with the coagulation cascade, endothelial cells, or platelets.[2]

Botulinum toxin is a potent neurotoxin that inhibits synaptic transmission by blocking the release of acetylcholine. Although it is typically confined to local effects, systemic absorption may occur in rare cases due to high doses, improper injection techniques, or individual susceptibility. Once absorbed systemically, it can induce widespread parasympathetic blockade within the peripheral autonomic nervous system, potentially affecting multiple organ systems including the cardiovascular, gastrointestinal, and respiratory systems. However, such systemic effects are exceedingly rare at the doses commonly used for cosmetic purposes and are generally associated with predisposing factors or procedural complications.

Isolated case reports have described serious adverse outcomes such as myocardial infarction, pulmonary embolism, venous thromboembolism, and even death following botulinum toxin A injections.

The study conducted by Coté et al[3] aimed to evaluate AEs reported to the U.S. Food and Drug Administration (FDA) following botulinum toxin type A (BTA) injections. Data were reviewed from the FDA's MedWatch system, focusing on serious AEs reported between December 1989 and May 2003, and non-serious AEs reported between December 2001 and November 2002. The analysis included both therapeutic and cosmetic uses of BTA, with attention given to both serious and non-serious AEs. A total of 1437 AE reports were analyzed. Of these, 1031 reports were associated with cosmetic use, including 36 serious and 995 non-serious cases. Serious AEs referred primarily to systemic complications affecting the cardiovascular, respiratory, and nervous systems. Among cardiovascular-related events, 2 cases were identified: 1 involving arrhythmia and the other unspecified.[3]

In the case report presented by Wang et al,[4] a healthy 22-year-old woman received a total of 300 units of botulinum toxin A injected into both legs for aesthetic treatment of calf muscle hypertrophy. On the third day post-injection, she developed pain and swelling in the left leg. By the fifth day, the pain had intensified, rendering her unable to walk. Imaging studies revealed deep vein thrombosis (DVT) and bilateral pulmonary embolism (PE). During the course of treatment, an inferior vena cava filter was placed, and anticoagulant therapy was initiated.

In the case report by Mines et al,[5] a 67-year-old woman with spasticity of the right calf and posterior tibial dystonia secondary to hypoxic brain injury sustained during childbirth

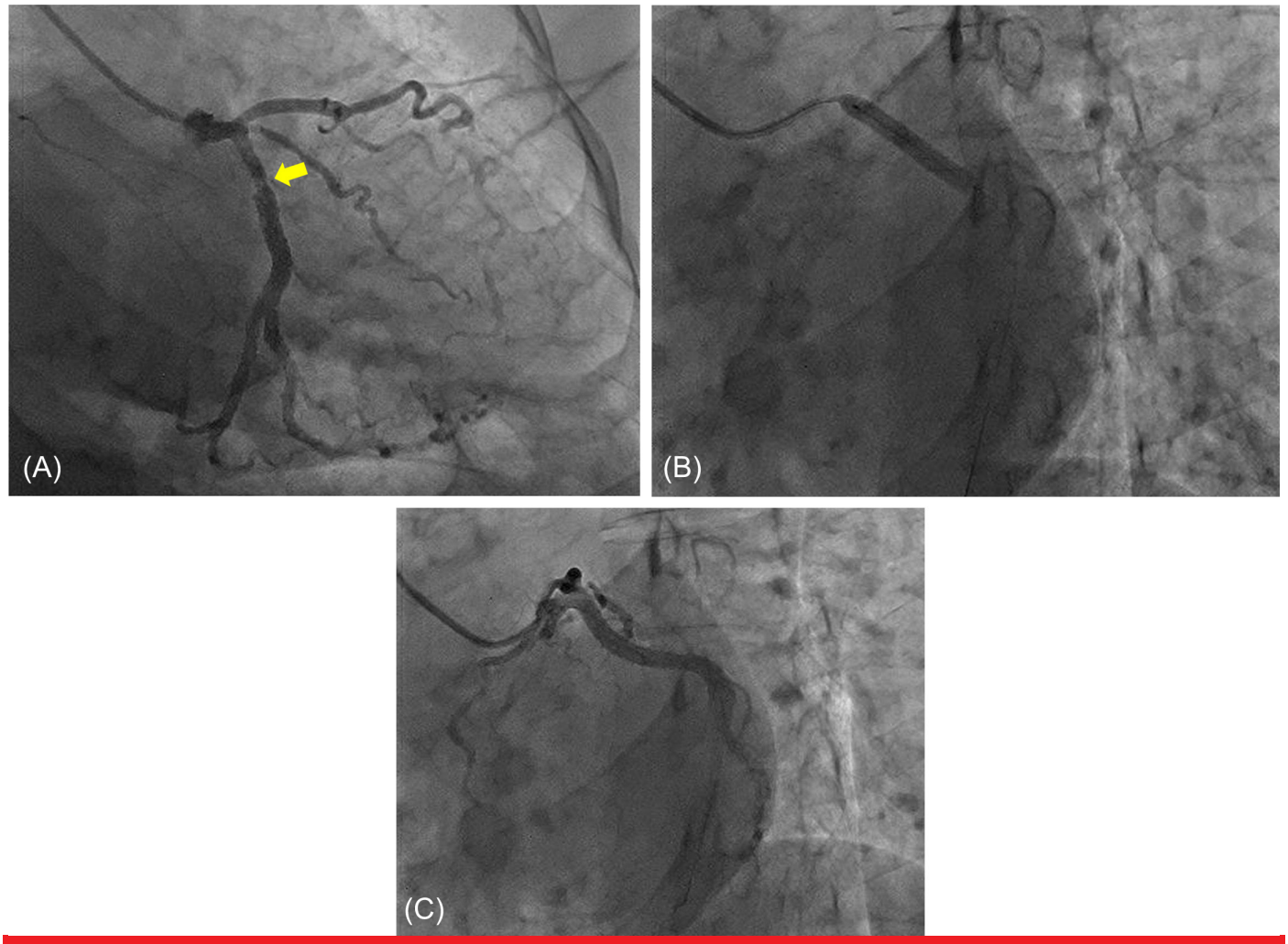


**Figure 2. (A) The angiographic image of the patient's circumflex artery and the hazy area in the proximal segment. (B) The image of stent implantation to the culprit lesion. (C) Successful post-stent implantation flow.**

was treated with 250 units of BoNT-A. Three days after the injection, the patient developed leg pain and edema. Venous Doppler ultrasound revealed a thrombus in the soleus vein. Apixaban therapy was initiated, and the symptoms resolved within 2 weeks.

In the case report by Stähli et al,[6] acute myocardial infarction developed in a 56-year-old patient following intravesical botulinum toxin A injection. Coronary angiography revealed a thrombotic occlusion in the right coronary artery, which was treated with drug-eluting stents. The authors emphasized that although rare, the systemic pro-thrombotic effects of botulinum toxin may lead to serious cardiovascular complications.

In the case reported by Jost et al,[7] a patient developed perianal venous thrombosis following BoNT-A injection into the anal sphincter region.

In the case reported by Pisani et al,[8] a 32-year-old individual received BoNT-A injections for the treatment of axillary hyperhidrosis and subsequently developed thrombosis in the superficial veins of the anterior chest wall (Mondor's disease). Symptoms resolved with enoxaparin therapy.

In this case, angiographic findings were consistent with a significant thrombus burden, suggesting an underlying prothrombotic state as a potential precipitating factor for acute myocardial infarction.

Although paradoxical embolism due to a patent foramen ovale cannot be entirely ruled out in the authors' patient, it remains an unlikely cause. Such embolic events typically present with abrupt distal coronary occlusions visible on angiography, which were not evident in this case. Moreover, the existing literature does not provide conclusive evidence for a systemic prothrombotic effect of botulinum toxin A. To date, *in vitro* and *in vivo* studies have not demonstrated significant effects on coagulation parameters, endothelial function, or platelet activation.[9] Therefore, the thrombogenic risk associated with botulinum toxin A remains theoretical.

Another possible cause that should not be overlooked is sympathetic activation triggered by anxiety or stress following botulinum toxin injection, which may precipitate myocardial infarction. However, the authors' patient is a nurse working in their hospital who is actively involved in performing such procedures and frequently undergoes them herself, with no

fear of hospitals or injections. Therefore, the authors did not consider this mechanism likely in her case. Additionally, according to the patient's statement, this was her third botulinum toxin injection, and she reported no anxiety or stress during the procedure.

Nevertheless, the close temporal relationship between botulinum toxin administration and the onset of myocardial infarction raises the possibility of a causal association. Considering that botulinum toxin is frequently used in elderly patients and those with cardiovascular comorbidities, clinicians should be cautious and monitor patients closely after administration, especially given the potential for acute coronary syndromes, which carry significant morbidity.

Although the possibility of a coincidental myocardial infarction independent of the injection and temporal association cannot be completely ruled out, several factors in this case make this less likely. The patient had no prior history of cardiovascular disease, her thrombotic parameters were within normal limits, and as a hospital staff member, she had been undergoing regular medical check-ups, all of which had been unremarkable up to that point. Given the temporal proximity between the botulinum toxin A injection and the onset of myocardial infarction, the event raises the possibility of a potential association between the injection and the infarction.

## CONCLUSION

Although myocardial infarction following botulinum toxin A injection is a rare event, the temporal association observed in this case is noteworthy. While the systemic prothrombotic effects of botulinum toxin remain unproven, mechanisms such as endothelial dysfunction or altered vascular reactivity remain plausible. Given the potential for serious complications like acute coronary syndrome, practitioners should exercise caution when administering botulinum toxin A, particularly in high-risk individuals, and ensure close post-procedural monitoring. This case highlights the need for further systematic research into the cardiovascular risks associated with botulinum toxin use.

**Informed Consent:** Detailed information was given to the patients regarding the possible contribution of the case report to the literature. The patients gave written and verbal consent for the publication of the case report.

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**Video 1:** Pre-percutaneous coronary intervention image of the circumflex artery.

**Video 2:** Post-stent implantation image of the circumflex artery.

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