Myocardial Infarction After Taser Exposure

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We report a case of a 20-year-old man who had an acute inferior wall myocardial infarction after he was subdued with a Taser-conducted electrical weapon. Myocardial infarction was diagnosed by electrocardiographic findings of ST-segment elevation in the inferior leads, followed by the development of new Q waves. Cardiac enzymes were elevated. Coronary arteriography was normal, and left ventriculography showed hypokinesis of the distal inferior wall. This is the first reported case of ST-segment elevation myocardial infarction related to Taser exposure.

Figure 1. Electrocardiogram recorded soon after the patient developed chest pain while in the emergency department shows ST-segment elevation in leads II, III, aVF, V₅, V₆, with reciprocal depression in lead aVL, indicating inferior and interolateral injury.
A 20-year-old man and several of his peers had an altercation with another group of young men outside a bar. Police arrived, and a police officer discharged a Taser X26 (Taser International, Scottsdale, AZ), striking the man in the upper right posterior part of the thorax and in the right buttock with two five-second activations of the device. The man did not sustain a significant fall. He was brought to the hospital in custody one hour later to have the Taser barbs removed.

In the emergency department, the patient had no signs of agitation or delirium. Initially, he reported no chest pain, then, 30 minutes after arrival, he developed burning retrosternal pain, rated 5/10 in intensity, accompanied by dyspnea. He was treated with aspirin and nitroglycerin paste, and his symptoms subsided.

The initial electrocardiogram (ECG) showed ST elevation in the inferior leads, consistent with inferior injury (Figure 1). Urgent coronary arteriography was performed percutaneously via the right radial artery. The coronary arteries were widely patent and angiographically normal (Figure 2). Left ventriculography showed hypokinesis of the distal inferior wall, with an ejection fraction of 60%.

Four hours after the onset of chest pain, a second ECG showed evolution of an inferior infarct (Figure 3). The initial serum troponin was 0.66 ng/mL and later peaked at 10.73 (reference < 0.04); serum creatine kinase (CK) was 373 U/L and peaked at 1016 (reference < 230); and CK-MB was 7.3 ng/mL and peaked at 52.5 (reference < 7.7).

The patient was taking no prescribed medications, but he reported regular use of Finaflex (Redefine Nutrition), an over-the-counter anabolic steroid supplement for muscle building. He denied cigarette smoking and drug use. He denied recent use of alcohol. A urine toxicology screen was negative for cocaine metabolites and other drugs except benzodiazepines, which he received in the emergency department. Blood toxicology was negative for ethanol. His serum lipid profile was abnormal with a total cholesterol level of 336 mg/dL (reference < 200), an LDL cholesterol level of 289 mg/dL (reference < 130), an HDL cholesterol level of 18 mg/dL (reference 40-59), and a triglyceride level of 144 mg/dL (reference < 150).

Throughout his two-day hospital stay, the patient’s blood pressure was elevated in the range of 146-190/90-98 mmHg. He had no further symptoms, and he was discharged the next day on aspirin, a beta-blocker, an angiotensin-converting enzyme inhibitor, and simvastatin. He was advised to stop taking the anabolic steroid supplement.

### DISCUSSION

Conducted electrical weapons such as the Taser have been used by law-enforcement officers on several hundred thousand criminal suspects. The Taser X26 uses compressed nitrogen to fire two barbs attached to long copper wires that adhere to the subject and deliver electrical energy. The device delivers up to 50,000 volts over five seconds. The electrical discharge stimulates skeletal muscle and motor

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Figure 2. Right (A) and left (B) coronary arteriograms in the left anterior oblique and right anterior oblique projections, respectively, are normal, as they were in all other standard projections. The opaque dot in the center of the images represents a pellet shot; the patient reported a remote history of being shot in the chest with a pellet gun.
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nerve stimulation, temporarily rendering a subject immobile, and also stimulating sensory nerves causing pain. Although the Taser has been associated with various injuries in case reports, larger studies have suggested that significant injuries are rare. Increasing use of the Taser has generated concern about the possibility of electrical weapons causing cardiac arrhythmias. In three studies involving 165 human volunteers, no arrhythmia was detected during or after Taser exposure. However, the Taser was shown to cause ventricular fibrillation and ventricular tachycardia in swine, and a case report described rapid ventricular tachycardia associated with Taser exposure in a 53-year-old man with a dual-chamber pacemaker. Another case report described new-onset atrial fibrillation after Taser exposure in a healthy 16-year-old boy. Reports of sudden cardiac death after Taser use have generated significant news attention, and have led the manufacturer to advise against aiming the Taser at the chest. Risk factors for death after Taser exposure include stimulant use, combative behavior, physical restraint, and "excited delirium." Cardiovascular disease also may play a role in Taser-related deaths. In a review of 37 autopsy reports following Taser-related deaths, cardiovascular disease was present in 54% of cases.

Taser exposure has not been shown to cause myocardial infarction, but it is well-established that direct electrical injury, such as a lightning strike or electrocution, can cause myocardial necrosis. In a study of 66 volunteers, including subjects with heart disease, there were no increases in serum troponin I level six hours after exposure to a Taser X26 discharge. Similarly, there was no elevation in troponin level in 38 volunteers after heavy exercise and prolonged Taser exposure. These findings suggest that the Taser is unlikely to cause direct electrical injury to the heart.

Other causes of myocardial infarction include coronary artery thrombosis, cardiac trauma, stress cardiomyopathy, and coronary artery spasm. In this case report, coronary artery thrombosis is unlikely, as there is no evidence of coronary artery disease or thrombus on coronary arteriography. Traumatic myocardial injury is unlikely, as there is no significant anterior chest trauma. It has been speculated that stress cardiomyopathy (Takotsubo cardiomyopathy) may play a role in Taser-related deaths, but in this case report, the pattern of myocardial injury is not consistent with stress cardiomyopathy.

The pathogenesis of the myocardial injury in this case remains uncertain. There is evidence of an acute myocardial infarction in the right coronary artery distribution, with normal coronary arteriography. Transient spasm of the

Figure 3. Electrocardiogram (ECG) recorded four hours after the ECG shown in Figure 1. ST-segment changes have diminished; slight terminal T-wave inversion is noted in leads III, aVF, V5, V6; and Q waves have appeared or become larger in leads II, III, aVF. Thus, there has been evolution of the changes of acute inferior myocardial infarction.
right coronary artery after Taser exposure may be the most plausible explanation for myocardial infarction in this young patient with hypertension and hyperlipidemia.

This case report suggests that Taser exposure may precipitate ST-elevation myocardial infarction. Patients who complain of chest pain after exposure to a conducted electrical weapon should be evaluated for myocardial injury.

This case report also has implications for public policy decisions about the use of conducted electrical weapons. Use of conducted electrical weapons may reduce the need for use of more lethal weapons, such as firearms, and has been shown to reduce the risk of injury both to police officers and to suspects. Further research on injuries caused by conducted electrical weapons is warranted.

REFERENCES