

# The effect of lipid emulsion during advanced cardiac life support for Bonzai (Tetrahydrocannabinol) cardiovascular toxicity

Demet Doğan Erol<sup>1\*</sup>, Can Erdoğan<sup>1</sup>, Cem Koray Çataroğlu<sup>1</sup>, Süleyman Süner<sup>2</sup> and Zeynep Uluşan Özkan<sup>2</sup>

<sup>1</sup>Department of Anaesthesiology and Reanimation, Yıldırım Beyazıt Hospital, Ankara, Turkey

<sup>2</sup>Department of Cardiovascular Surgery, Yıldırım Beyazıt Hospital, Ankara, Turkey

## Objective

Worldwide, bonzai tetrahydrocannabinol is among the most widely used illicit drugs. The current wave of decriminalization may lead to more widespread use, and it is important that cardiologists be made aware of the potential for marijuana-associated adverse cardiovascular effects that may begin to occur in the population at a greater frequency. The main active substance, tetrahydrocannabinol, affects cannabinoid receptor 1 in the brain and cannabinoid receptor 2 in the periphery, with predominantly psychoactive effects [1]. There are several well-recognized complications associated with tetrahydrocannabinol use, including psychiatric, respiratory system, and cardiovascular disorders [2]. Published reports describe a temporal relation between tetrahydrocannabinol use and the development of acute myocardial infarction, cardiomyopathy, and sudden cardiac death. The mechanism underlying the association between tetrahydrocannabinol use and myocardial infarction is currently unknown. It is possible that cannabis has a deleterious effect on coronary microcirculation. Dahdouh, *et al.* reported the case of a 20 years old patient who abused tobacco and marijuana and developed cardiac arrest and massive myocardial infarction [3].

Lipid resuscitation refers to the use of an intravascular infusion of a lipid emulsion to treat severe, systemic drug toxicity or poisoning. Increasing evidence suggests that the intravenous (IV) infusion of lipid emulsions can reverse the cardiac and neurologic effects of local-anesthetic toxicity [4]. The role of lipid emulsion has expanded to treatment of cardiac toxicity due to other lipophilic drugs. Indeed, case reports support the early use of lipid emulsion at the first sign of arrhythmia, prolonged seizure activity, or rapid progression of toxic manifestations in patients with suspected local anesthetic toxicity. The similarity of several subsequent reports suggests that underlying ischemic heart disease or cardiac conduction defects may predispose to local anesthetic cardiac toxicity [5].

## Case report

23 years old man reported heart fluttering and near syncope a few hours after bonzai tetrahydrocannabinol, heroin smoking. In the emergency department, he was found to have a right bundle-branch-type ventricular tachycardia and had failed more than 40 mins of standard life support therapy including ventilation with oxygen, vasopressors, and force diuresis. Current guidelines suggest that 20% lipid emulsion initially be administered as a bolus of 1.5 mL/kg

over 1 min. Following completion of the bolus, a continuous infusion of 0.25 mL/kg/min should be started. Followed 20% lipid infusion at 16 hours rapidly restored normal cardiac markers and hemodynamic parameters. The patient recovered completely with no evidence of myocardial damage after 96 hours. (Table 1)

## Conclusions

While the exact mechanism of action is not known, several hypotheses exist as to how lipid emulsion works in the setting of local anesthetic toxicity. The first is the 'lipid sink' hypothesis that suggests that lipid-soluble local anesthetic molecules in the aqueous phase are extracted by the injected lipid. The lipid sequesters the local anesthetic and reduces the concentration of tissue bound local anesthetic. Alternatively, the metabolic hypothesis postulates that lipid impedes local anesthetic's inhibition of acyl carnitine, thereby improving mitochondrial metabolism. Finally, it has been proposed that lipid emulsion increases calcium concentrations in myocytes thereby improving contractility [6].

Table 1. Cardiac markers

	4. hour	8. hour	12. hour	16. hour	20.hour After lipid	24.hour After lipid	48.hour After lipid	72.hour After lipid	96.hour After lipid
Creatinin kinaz (U/L)	1384	2576	3137	4065	2324	1215	266	117	
CKMB (U/L)	64	78	99	95	55	60	19	22	6.6
Troponin I (ng/ml)		1.11	1.49	2.5	0.67		0.08	0.05	0.03
AST (U/L)	109	161	160	157	115	75	27	33	28
ALT (U/L)	85	134	138	135	129	105	71	56	48

**Correspondence to:** Demet Doğan Erol, Department of Anaesthesiology and Reanimation, Yıldırım Beyazıt Hospital, Ankara, Turkey, E-mail: demetdoganerol@myynet.com

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