

CASE REPORT

“Double arrest” - Amphetamine fatality in a 31-year-old male: a case report

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Abstract

Amphetamine abuse in the general population in the Netherlands is relatively limited and stable. Approximately 0.2% of the Dutch population used amphetamine in 2005.

Fatal cases of amphetamine-associated cardiotoxicity (myocardial infarction or necrosis, acute cardiac failure or cardiac arrhythmia) are rarely reported in medical literature.

This report describes the case of a 31-year-old male standing in front of his bedroom window, swinging an axe and threatening to jump. During a struggle with the police in which pepper spray was used, the patient suddenly collapsed and lost consciousness. Police officers immediately started with basic life support. After twenty-one minutes of resuscitation there was a sinus tachycardia and output. The evening prior to his struggle with the police, the patient had smoked 1.5 gram of speed.

Toxicological analysis in the patient's urine revealed a concentration of 26 mg/L of amphetamine (toxicological level >0.2 mg/L) and 18 µg/L of methamphetamine (toxicological level >0.2 mg/L). No traces of pepper spray were found. Initial electrocardiogram revealed diffuse ischemia. On our ICU, refractory cardiogenic shock developed and was eventually followed by death, approximately 30 hours after admission.

Introduction

Amphetamine abuse in the general population in the Netherlands is relatively limited and stable. Approximately 0.2% of the Dutch population used amphetamine in 2005.¹

Amphetamine-associated cardiotoxicity (myocardial infarction or necrosis, cardiomyopathy, and cardiac arrhythmia) cases are rarely reported in medical literature.

We describe a case of sudden cardiac arrest after amphetamine abuse, followed by refractory cardiogenic shock and eventually death.

Case

A 31-year-old male was observed by neighbours standing in front of his bedroom window on the first floor shouting,

threatening to jump and swinging an axe. Neighbours called in the emergency services. Police officers forced the door and tried to arrest the male. Pepper spray and physical force were used to overwhelm him. During the struggle, he suddenly collapsed and lost consciousness. Police officers immediately started basic life support. Seven minutes later, a team of paramedics arrived on the scene. The first heart rhythm found was asystole. After twenty-one minutes of resuscitation, and after the administration of 5 mg adrenaline and 3 mg atropine, the patient had a sinus tachycardia and cardiac output. After this successful resuscitation he was transported to the emergency department of our hospital.

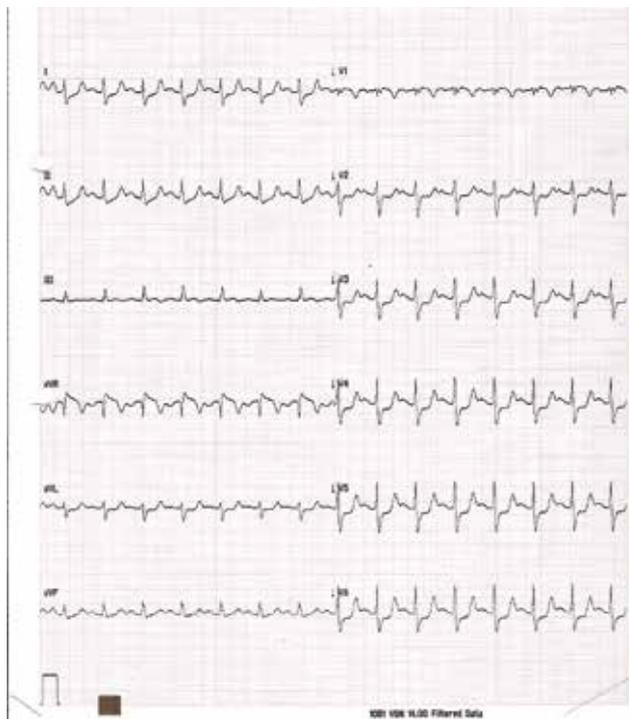
On arrival at our emergency department, the patient's vital signs were: blood pressure 80/40 mm Hg, heart rate 120/minute, SpO₂ 97%, temperature 36.4° C and Glasgow Coma Scale score E1 M1 V tube. His pupils were dilated and did not respond to light. Heart and lung sounds were normal and the abdomen showed no abnormalities. The patient's right wrist and left tibia were extensively bruised. Initial ECG showed diffuse ischemia and chest X-ray showed no pulmonary pathology, however, the heart appeared to be enlarged (*figure 1*).

Routine laboratory investigations were performed: arterial blood gas measurements showed a severe metabolic acidosis (*table 1*).

At that time it remained unclear on what grounds sudden cardiac arrest had occurred. Taken into account the young age and the circumstances in which the sudden cardiac arrest had occurred, blood and urine samples were obtained for toxicological analysis. Because it was unclear at the time what the origin of the sudden cardiac arrest and the first presented rhythm were, we decided to start a 24 hours hypothermia protocol.^{2,3} During the hypothermia protocol, the patient showed persisting hypotension, so fluid resuscitation and noradrenalin were started.

Four hours after admission to the ICU, hetero-anamnesis revealed that the patient used speed on a regular basis. He started to smoke speed at the age of fifteen and was not a daily user, but

Figure 1. Initial ECG showing a sinus tachycardia of 112 beats/minute, intermediary axis, ST elevation in aVR with a negative T-top and ST depression in I, aVL, II, III, aVF, and V2-V6.



used three to four times per month. Quantity of this usage could not be specified by his family. His girlfriend admitted that on this occasion he had smoked 1.5 gram of speed.

Toxicological screening of his urine was positive for amphetamine, and quantitative analysis showed a concentration of 26 mg/L of amphetamine (toxicological level >0.2 mg/L) and 18 µgr/L of methamphetamine (toxicological level >0.2 mg/L).⁴ No traces of the contents of pepper spray (capsaicine, dihydrocapsaicine or nordihydrocapsaicine) were found.

During admission to the ICU the patient developed a severe rhabdomyolysis (*table 1*).

Creatinekinase rose to a maximum of 10,440 U/L. For renal protection, forced diuresis was started with an infusion of sodium bicarbonate at a concentration of 1.4% at a rate of 4 litres per 24 hours.

Thirteen hours after admission the patient became haemodynamically unstable, with a blood pressure of 60/40 mm Hg. The shock was unresponsive to fluid resuscitation and noradrenalin up to a rate of 0.6 microgram/kg/min. It was therefore necessary to terminate the 24 hours hypothermia protocol. One hour later, the patient appeared more and more dependent on inotropic drugs. Therefore, it was decided to add adrenalin at a rate of 0.2 microgram/kg/min and phenylephrine at a rate of 1 mg/hour. Despite changes in ventilator settings and an infusion of sodium bicarbonate, the patient became more and more acidotic.

The patient remained haemodynamically unstable, and did not respond to gradually increased adrenalin up to a rate of 1.2 microgram/kg/min. Fluid resuscitation with colloids

Table 1. Laboratory results.

	Hours (h) after admission							Reference values
	ER	1.5 h	7 h	13 h	19 h	25 h	27.5 h	
BUN (mmol/L)	5.3	6.2	7.3			12.9		2.5-7.5
Creatinine (mm/L)	187	202	167			345		65-115
Sodium (mmol/L)	137	134	140	138	146	149		135-145
Potassium (mmol/L)	5.2	5.9	2.4	3.3	2.2	2		3.2-4.7
ASAT (U/L)	326	318	811	890	619	760		<40
ALAT (U/L)	441	349	568	515	329	509		<45
CK (U/L)	422	773	8,490	10,440	7,777			10-200
CK-MB (U/L)	51	60	263	380	279			0-14
Troponine (ug/L)	0.04	1.05	14.4					<0.04
pH	6.61	7.26	7.33	7.18	6.94	6.94	6.98	7.37-7.42
pCO ₂ (kPa)	14.5	3.8	3.2	4.6	4.4	4.6	3.8	4.8-5.9
HCO ₃ (mmol/L)	11	12	12	12	7	7	7	22-26
Base excess (mmol/L)	-29.6	-13.4	-11.6	-15.4	-24.2	-24	-23.7	-2.5-2.5
pO ₂ (kPa)	13.6	52.5	21.4	11.1	21.4	12.6	12	9.3-13.3
O ₂ saturation (%)	86	100	99	94	98	95	97	95-100
Lactate (mmol/L)	11.8	11.3	6.7			18.9		0.4-1.2

did not have any effect. At this point, the patient's blood pressure dropped to 55/35 mm Hg and could not be raised either by increasing the inotropic drugs or fluid resuscitation with colloids. Despite all these efforts the patient died as a consequence of cardiac failure.

Discussion

Our patient sustained a sudden cardiac arrest after amphetamine abuse. Amphetamines are a class of noncatechol sympathicomimetic amines that produce central nervous stimulation.⁵ Amphetamines induce euphoria, increase alertness, intensify emotions and boost self-esteem. Because of these effects amphetamines are a popular party drug. A research study conducted among visitors of bars and cafes in Amsterdam showed that 17 percent of the visitors had experience with amphetamine.¹

In excessive doses amphetamines cause anxiety, hallucinations, coma, seizures, cardiotoxicity and agitation.⁵⁻⁷ Amphetamines can be used orally, per inhalation, and intravenously. After inhalation, amphetamine has a strong and direct effect.⁴ Amphetamines are metabolized in the liver.⁸ Twenty to 65% of amphetamines are eliminated through the kidneys in unchanged form, and the remaining percentage is excreted as metabolites.⁴ Physical half-life is 10-30 hours in normal urine, but is strongly prolonged in alkali urine (up to 140 hours).⁴

To date, reports about amphetamine-associated cardiotoxicity remain scarce.

Cardiotoxicity of amphetamine and its synthetic derivatives can manifest itself as: acute myocardial infarction or necrosis, arrhythmias, cardiomyopathy and acute heart failure.⁹⁻¹⁵

There is a number of possibilities that could have induced the sudden cardiac arrest in this case. Firstly, amphetamine is an indirectly acting sympathicomimetic that stimulates the release of noradrenaline from sympathetic nerves. This release of noradrenaline has a pressor effect on the coronary circulation and may precipitate vascular spasm and therefore cause ischemic infarction. Myocardial ischemia causes a massive efflux of potassium. This efflux of potassium can in turn lead to cardiac arrhythmias.

Secondly, amphetamines themselves can induce myocardial necrosis. For example, it has been demonstrated in rats that administration of methamphetamine results in the loss of myoglobin in the ventricular myocardium, swelling and degeneration of mitochondria in affected myocytes, sarcolemmal damage, myocytolysis and fibrosis.¹⁶⁻¹⁸ Thirdly, it has been described that amphetamine can induce acute as well as chronic cardiomyopathy.^{19,20}

It is known that patients placed in physical restraints can suffer sudden cardiac arrest due to a combination of dehydration, adrenergic neurotransmitters, and metabolic acidosis.²¹ Heavy muscle contraction during the struggle most certainly

attributed to the rise in creatinine kinase and also the severe (lactate) acidosis.

Autopsy showed an 80% stenosis of the left anterior descending artery, and a 50% stenosis of the left circumflex coronary artery. No valve vitia were found. The weight of the heart was 430 gram. Macroscopically, there were multiple infarctions visible in the lateral and inferior wall of the left ventricle, with a maximal diameter of 0.5 cm x 0.5 cm. Microscopic LDH colouring showed a concentric decolouring of the left ventricle. It is difficult to establish which of the above described mechanisms led to the sudden cardiac arrest in our patient. Most likely it was a combination of pathophysiological mechanisms and, in his agitated state, coronary vascular spasms occurred, which led to diffuse infarction. Probably, infarction was speeded up as a consequence of increased oxygen demand, caused by the struggle with the police. Also the pre-existent coronary artery stenosis in two coronary arteries may have speeded up the infarction. Massive efflux of potassium, caused by diffuse infarction could have led to an arrhythmia.

The irritating substances in pepper spray used by the Dutch police are capsaïcine, dihydrocapsaicine and nordihydrocapsaicine.²² No traces of these substances were found at toxicological analysis. However, the use of pepper spray on our patient could have induced the feeling that breathing was impossible. Combined with the physical struggle this could have led to a decreased availability of oxygen to the myocardium.

In conclusion, amphetamine can cause cardiotoxicity in different ways. In our patient, amphetamine intake together with physical exertion most likely caused diffuse coronary vascular spasms, resulting in diffuse infarction and eventually arrhythmia.

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NVIC CURSUSSEN LUCHTWEGMANAGEMENT OP DE IC

Woensdag 4 en donderdag 5 juni 2014
Maandag 17 en dinsdag 18 november 2014

LOCATIE: Hotel Houten en Skillscenter OSG te Houten

Luchtwegmanagement

Luchtwegmanagement is een van de belangrijkste taken van de intensivist. Regelmatig zijn spoedintubaties noodzakelijk. Vaak is sprake van gecombineerde problematiek, is er minder tijd en zijn de omstandigheden minder gunstig dan in electieve situaties. Dit vereist een brede aanpak met aandacht voor oxygenatie, circulatie en medicatie.

Opzet van de tweedaagse cursus

De tweedaagse NVIC Cursus Luchtwegmanagement op de IC is een intensieve en praktijkgerichte cursus. In kleine groepen worden verschillende luchtwegtechnieken geoefend en diverse scenariotrainingen gedaan. Aan de hand van ingestuurde casuïstiek wordt ruim aandacht besteed aan luchtwegstrategieën.



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