Pseudoephedrine-induced Ventricular Tachycardia

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ABSTRACT

Here, we report an unusual cause of ventricular tachycardia which had developed following pseudoephedrine intake. A 55 year old male patient was admitted to the emergency department with complaints of sustained palpitation. Monitorization records revealed ventricular tachycardia of 214 beats per minute. He had been suffering upper respiratory tract symptoms for the last two days. Palpitation had started an hour later following the ingestion of cold remedy drug, which included pseudoephedrine, and serious dyspnea gradually occurred. He had ischemic heart disease and peripheral arterial disease but he did not have a history of arrhythmia, any palpitation, diabetes mellitus or hypertension. Initially, lidocaine and later amiodorone infusion was administered but medical cardioversion was not successful. Sinus rhythm was provided after electrical cardioversion with 200 joule. Thereafter the patient was stable.

Key words: *Ventricular tachycardia, pseudoephedrine, side effect*

Psödoefedrine Bağlı Gelişen Ventriküler Taşikardi

ÖZET

Burada psödoefedrin kullanımına bağlı nadir bir yan etki olan ventriküler taşikardi olgusu sunulmuştur. 55 yaşında erkek hasta, acil kliniğine çarpıntı şikayeti ile başvurdu. Monitörizasyonunda 214/dk hızında ventriküler taşikardi saptandı. Hasta son iki gündür üst solunum yolu infeksiyonu geçirmekteydi. Çarpıntısı, soğuk algınlığı için aldığı ve psödoefedrin içeren ilaçtan yaklaşık bir saat sonar başlamış ve giderek artan şiddetli nefes darlığı ortaya çıkmış. İskemik kalp hastalığı ve periferik arter hastalığı anamnezi olmasına rağmen aritmi, hipertansiyon veya diabetes mellitus anamnezi yoktu. Önce lidokain, sonra amiodaron infüzyonu ile uygulanan medikal kardiyoversiyon başarılı olmadı. 200 joule ile yapılan elektiriki kardiyoversiyon ile sinüs ritmi elde edildi. Daha sonra hasta stabil seyretti.

Anahtar kelimeler: *Ventriküler taşikardi, psödoefedrin, yan etki*

INTRODUCTION

Palpitation is a common chief complaint among emergency department patients, and is often associated with a tachydysrhythmia. Ischemic heart disease, cardiomyopathy and congenital diseases are organic based reasons for arrhythmia, but other conditions such as metabolic disorders, electrolyte imbalance and drug intoxications should be assessed for non-cardiac arrhythmia etiology (1, 2).

This is a potentially life-threatening arrhythmia because it may lead to ventricular fibrillation and sudden death (2). The diagnosis of ventricular tachycardia (VT) is made based on the rhythm of sequential 3 or more ectopic ventricular complexes (broad QRS), with the rate at least 100-250 beat /minute, seen on either a 12 lead electrocardiography (ECG) rhythm strip (3).

Pseudoephedrine is a synthetic isomer of ephedrine. The main effects of pseudoephedrine are on the cardiovas-

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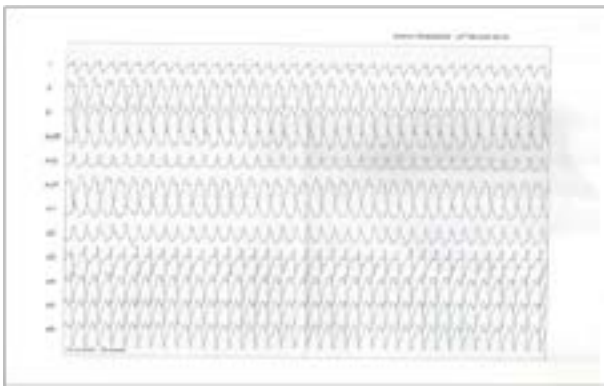

***Figure 1**. 12 lead ECG showed broad QRS complexes with the rate of 214 beat /minute*

cular system via alpha and beta- adrenergic activities. Hypertension, tachycardia, tremor, sleeping disorders, abdominal cramps and diarrhea can be seen due to alpha and beta-adrenergic activities. Particularly, individuals with an underlying predisposing disease, multidrug users, stimulator drug and herbal based compound users frequently experience these adverse effects (4, 5).

We presented a case with ventricular tachycardia requiring electrical cardioversion due to pseudoephedrine intake.

CASE

A 55-year-old male was admitted to our hospital with complaints of palpitation and dyspnea which had begun 7-8 hours ago. He had been suffering of weakness, sneezing, and mild headache in the last few days and was given a prescription of cold remedy drugs with the diagnosis of upper respiratory tract infection. He reported that his palpitation started one hour later following the ingestion of a decongestant containing 60 mg pseudoephedrine. He thought this condition could be temporary and did not refer to a hospital. However, his complaints continued and dizziness with dyspnea was added.

In his medical history he had a myocardial infarction in 1997. Coronary angiography revealed pathology in only one vessel. He had peripheral arterial disease diagnosed one year ago and a stent was implanted in the left lower extremity. His family history was non-significant. He smoked 1pck cigarette/day for 40 years, and he drank 35 cc/day alcohols for 20 years. Following prior MI, enteric coated aspirin 150 mg and atorvastatin 20 mg were prescribed. He denied any use of cocaine or other sympathomimetic drugs.

His general appearance was moderate; he was conscious, cooperating and oriented. The vital signs were as follows; blood pressure 80/40 mm Hg, pulse 200 beats/minute and irregular, respiratory rate 25 beats/min, axillary body temperature 36.2°C and oxygen saturation was 85% in room air. There was no murmur, rub, click, or S3 and S4 gallop rhythm but he had tachycardia. Peripheral pulses of distal left upper and lower extremity were not palpable. Other system examinations were unremarkable. All biochemical and hematological values were within normal range. Arterial blood sampling revealed: pH; 7.49, arterial CO_2 pressure: 30.7 mmHg, arterial O_2 pressure: 80 mmHg, O_2 saturation; 97%. 12 lead ECG showed broad QRS complexes with the rate of 214 beat /minute (Figure). His echocardiograpy showed; advanced left ventricular systolic function deterioration and mild mitral insufficiency. The ejection fraction was 30%.

Initially, therapy was started to treat the patient with intravenous lidocaine. 100 mg of intravenous lidocaine was administered. Subsequently, amiodorone (bolus 150 mg/IV) was administered. Afterwards we continued with 1 mg/kg amiodorone infusion. But both drugs failed in restoring a sinus rhythm. Due to the beginning of poor peripheral perfusion along with syncope, hypotension, and lack of radial pulses and having failed with medical cardioversion, we decided to perform electrical cardioversion.15 mg of etomidate and 50 mg of fentanyl were administered intravenously to establish sedation and analgesia. Synchronized electrical cardioversion was performed three times (100-150-200 joule, respectively). Following 200 joule sinus rhythm was obtained. When he achieved stable condition he was transferred to coronary intensive care unite.

On the upper and lower extremity Doppler USG; subclavian, axillary, brachial, radial, ulnar arteries were clear, flow rates were decreased. Vascular wall structure of the left main profound and superficial femoral and popliteal arteries was irregular and there were calcific plagues. All flow rates of left peripheral arteries were decreased, flow volume was lower.

His treatment with amiodorone and heparin infusions

continued in the coronary intensive care unit. There was no other arrhythmia for 72 hours following the transfer to coronary intensive care unit. Cardiac markers were not elevated. He was discharged on the fifth day of the hospitalization without any symptoms.

DISCUSSION

Ventricular tachycardia is an extremely hazardous rhythm. It refers to any rhythm with wide QRS complexes faster than 100-250 beats per minute arising distal from the bundle of His. VT lasting over 30 seconds is called sustained tachycardia (1). The most common setting for VT is ischemic heart disease, in which myocardial scar is the substrate for electrical reentry. Commonly it is associated with hemodynamic compromise, particularly if the left ventricle is impaired. Besides the structural and valvular diseases of the heart, utilization of drugs such as isoproterenol, digoxin, quinidine, epinephrine, physostigmine, theophylline, cyclic antidepressants, antihistaminic, and thyroxin, metabolic problems such as hypoxia, electrolyte disturbances, alkalosis or deep acidosis could cause ventricular tachycardia (2). VT may cause important symptoms such as acute regular or irregular palpitations, dyspnea and syncope. Weakness, chest pain and dizziness can occur (2). Similarly our case presented to the emergency unit with weakness, dizziness, and dyspnea.

The main effects of pseudoephedrine are on the cardiovascular system via alpha and beta- adrenergic activities. Hypertension, tachycardia, tremor, sleeping disorders, abdominal cramps and diarrhea can be seen due to increased alpha and beta-adrenergic activities (4). Particularly, individuals with an underlying predisposing disease, multidrug users, stimulator drug and herbal based compound users frequently experience these adverse effects. Mores et al have reported that consumption of pseudoephedrine within recommended doses is not a threat in healthy subjects (6). However, if there are underlying predisposing conditions, utilization of these drugs should be avoided. Cigarettes lead to coronary vasospasm by both increasing thrombocyte aggregation and catecholamine release. Intensive smokers are at high risk especially when they take anticold drugs with tea and coffee (7). In our case, the patient had smoked for years, after myocardial infarction he only decreased smoking but still was a smoker.

Data from a trial showed that six patients had acute myocardial infarction out of 160000 pseudoephedrine users; this ratio was two times the general population (8). Furthermore, a case of acute myocardial infarction has been reported due to pseudoephedrine intake within the recommended doses (9). Bektaş et al. (10) also presented a young female patient without any disease who had developed supraventricular tachycardia after taking 60 mg pseudoephedrine.

Anticholinergics should be considered as causative agents because they have cardiac effects, although they are not as potent as pseudoephedrine. Such as pseudoephedrine, phenylephrine, phenylpropanolamine, xilometazoline HCL drugs decrease congestion on upper respiratory tract through vasoconstriction and provide symptomatic relief (4). Kayrak et al. (7) reported a case with a prior history of hospitalization for acute coronary syndrome but had normal coronary angiography. One month later the patient was referred to a hospital for ST elevated myocardial infarction after high-dose xilometazoline ingestion.

In the presented case VT could be associated with the existing ischemic heart disease. However, the patient did not have a prior VT attack till aftermath of taking pseudoephedrine and he did not have further VT episodes after discontinuation of pseudoephedrine strongly suggesting a relation between the VT episode and pseudoephedrine use. Considering the underlying heart disease, VT is often associated with an increased risk of sudden death after fibrillation but patients can also be asymptomatic. Especially in younger, sportive individuals it can lead to sudden deaths. Parasympathetic activity diminishes and sympathetic activity increases during exercise. These alterations provide excessive norepinephrine (NE) release from sympathetic postganglionic nerve endpoints. Hypercoagulability and atherogenic effect of pseudoephedrine can lead to premature coronary artery disease and sudden death (11). Bright et al reported a case of paroxysmal supraventricular tachycardia (PSVT) that was induced during exercise after an intake of 120 mg of pseudoephedrine (8).

If a patient with organic heart disease tolerates the VT well, pharmacologic therapy may be initiated. The risk benefit ratio of treating each specific type of VT should be considered before beginning the therapy. This is important because antiarrhythmic agents can produce or exacerbate the arrhythmias (5x 12). Non-sustained VT

may not be treated because its prognosis is not affected. However; such patients have recurrent polymorphic VT and a high mortality from sudden death if they are left untreated.

If the patients have hypertension, organic heart disease, and hemodynamic compromise or if there is an evidence of ischemia or central nervous system hypoperfusion, the rhythm should be promptly terminated by (DC) cardioversion (5x12).

When our patient presented to the emergency department, he was clinically stable. Therapy was started with 100 mg I.V. lidocaine. Subsequently I.V. amiodorone therapy was administered. But both drugs failed to restore a sinus rhythm. Due to the beginning of poor peripheral perfusion along with near syncope, hypotension, and lack of radial pulses and having failed with medical cardioversion, was decided to perform electrical cardioversion. Prior to deteriorate three times (100-150-200 joule, respectively) synchronized electrical cardioversion which was performed three times. Following 200 joule sinus rhythm was returned.

Oral alpha adrenergic agonist agents are the most prescribed drugs especially in fall and winter. Pseudoephedrine is a common ingredient in many cold remedies. Although a completely satisfactory proof of causation was not obtained, the VT in the present case could be attributed to pseudoephedrine. It is interesting that the drug was used within the recommended dosage.

We believe that our patients' history of coronary and peripheral arterial disease and his smoking habit provoked this life-threatening tachyarrhythmia. Therefore, it is important to question every patient about his/her previous medical history and concomitant drug use before prescribing any cold remedy drugs.

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