Asymptomatic Pediatric Patient and Idiopathic Ventricular Tachycardia: Case Report

Asemptomatik Pediatrik Hasta ve İdiopatik Ventriküler Taşikardi

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Geliş Tarihi/*Received:* 26.09.2008
Kabul Tarihi/*Accepted:* 26.03.2009

This case report is presented as TARK 2007.

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ABSTRACT We present a pediatric case with no previous cardiac problem that developed idiopathic ventricular tachycardia (VT) during anesthesia induction. A 12 years old boy was scheduled for surgery of bilateral tube insertion due to chronic dacryocystitis. Sedation was achieved with 2 mg i.m. midazolam administered in the operation room. Following pre-oxygenation, 2.5 mg/kg of propofol and 0.75 mg/kg atracurium was administered and the patient was intubated. Anesthesia was maintained by sevoflurane 2% in a 40% O_2 + 60% N_2O mixture. Shortly after administration of sevoflurane, monomorphic couplet or triplet ventricular extrasystoles (VES) with wide QRS waves were observed followed by sustained VT (135-140 pulse/min). Lidocaine 40 mg /min i.v was administered, but VT did not resolve and sevoflurane inhalation was discontinued. Normal sinus rhythm was restored within 1 minute. Sevoflurane inhalation was resumed, but once more led to a series of VES within 1 minutes, therefore anesthesia was terminated, surgery was cancelled and patient was referred for detailed cardiovascular examination. Holter test revealed rare uniform benign VES, but no couplet, triplet VT. Result of invasive electrophysiological examination was normal. We believe that concomitant use of sevoflurane and phenylephrine might trigger VT since there was no cardiologic pathology.

Key Words: Sevoflurane; anestezin; tachycardia, ventricular; phenylephrine

ÖZET Anestezi indüksiyonu sırasında kardiyak problemi olmayan ve fenilefrin damla kullanılan bir pediatrik olguda oluşan idiopatik ventrikül taşikardi (VT) oluşumunu rapor ettik. 12 yaşında kr dakriosistit nedeniyle opere edilecek olgu 2 mg i.m midazolam ile premedike edildi. Preoksijenizasyon dan sonra 2.5 mg/kg propofol ve 0.75 mg/kg atrakuryum yapılıp, entübe edildi. İdame %40 O_2+ %60 N_2O karışımı içerisindeki %2 Sevofluran ile sağlandı. Dekonjestan amaçlı olarak fenilefrin damla her iki burun deliğine damlatıldı. Kısa bir süre sonra geniş QRS'li monomorfik ikili ve üçlü ventriküler ekstrasistol (VES) ve daha sonra kalıcı VT (135-140 atım/dk) atakları görüldü. 40 mg/dk i.v lidokain uygulandı. Ancak VT devam etti ve sevofluran inhalasyonuna son verildi. 1 dk sonra ritim sinüs ritmine döndü. Sevofluran inhalasyonuna başlandı ve tekrar VES atakları görülünce anestezi sonlandırılıp hastanın operasyonu iptal edilerek detaylı kardiovasküler incelemeye alındı. Holter testin de nadir uniform bening VES'ler görüldü ve couplet ve triplet VT saptanmadı. Olgunun invaziv elektrofizyolojik çalışma sonucu normal olarak değerlendirildi. VT'nin kardiyak bir patolojiye değil sevofluran ve fenilefrin etkileşmesine bağlı olduğunu düşündük.

Anahtar Kelimeler: Sevofluran; anestezi; ventriküler taşikardi; fenilefrin

Turkiye Klinikleri J Anest Reanim 2009;7(2):113-6

Ventricular tachycardia (VT) is most often associated with structural heart disease such as cardiomyopathy and myocardial infarction.[1] Heart rate is usually faster than 125/min. Ventricular tachycardia that is seen in a patient without an underlying cardiological pathology is

defined as idiopathic Ventricular tachycardia.[1,2] If the number of beats is more than 10 and the rhythm lasts longer than 30 seconds, then this condition is referred to as sustained VT. This is an extremely important and life-threatening condition which should be treated urgently. We present here a pediatric case with no previous cardiac problem that developed idiopathic ventricular tachycardia during anesthesia induction.

CASE

A 12 years old boy was scheduled for surgery of bilateral tube insertion due to chronic dacryocystitis. He has not experienced any symptoms like palpitation, pre-syncope, syncope or dyspnea that might indicate an arrhythmia. He had no medication used regularly, and his medical history revealed no remarkable finding attributable to cardiac disease. At admission his blood pressure was normal and he had a rhythmic pulse. No cardiac murmur, pathologic heart sounds or cyanosis was detected in physical examination. Pre-anesthesia laboratory results were: hemoglobin: 14.7 mg/dL, hematocrit: 43.1%, platelets: 240.000 /mm^3, WBC 9.900: /mm^3, glucose: 81 mg/dl, K: 3.3 mmol/L, SGOT: 40 U/L , SGPT: 5 U/L, Na: 131 mmol/L , Ca: 9.7 mg/dL, Mg: 1.9 mg/dL, Cre: 0.6 mg/L, TBil: 2.6 mg/dL, DBil: 0.4 mg/dL, Albumine: 3.3 mg/dL, CK: 371. Sedation was achieved with 2 mg i.m. midazolam administered in the operation room. Blood pressure, Pulse Oximeter Oxygen Saturation (SpO_2), end-tidal carbon dioxide levels ($EtCO_2$) were continuously monitorized. Standard DI-I derivation of ECG showed a pulse rate 90 per minute with sinusoidal rhythm; mean arterial pressure was 90-110 mmHg; SpO_2 was measured as 98% by pulse oximeter and $EtCO_2$ was 35-45 mmHg. Following pre-oxygenation with 100% oxygen for 5 minutes, 2.5 mg/kg of propofol and 0.75 mg/kg atracurium was administered and patient was intubated. Phenylephrine HCL %2.5 (mydfrine) was administered into both nostrils as decongestant. Anesthesia was maintained by sevoflurane 2% in a 40% O_2+ 60% N_2O mixture. Shortly after administration of sevoflurane, monomorphic couplet or triplet ventricular extrasystoles (VES) with wide QRS waves developed, followed by sustained VT (135 -140 pulse/min) (Figure1). Lidocaine 40 mg/min i.v was administered as he still had pulsation at the carotid arteries. However, VT did not resolve and sevoflurane inhalation was discontinued. Normal sinus rhythm was restored within 1 minute. Sevoflurane inhalation was resumed, but once more led to a series of VES within 1 minute, thus anesthesia was terminated, surgery was cancelled and patient was referred for detailed cardiovascular examination. Normal sinus rhythm was observed on electrocardiography (ECG). All cardiac structures and functions appeared normal by echocardiography. Holter test revealed rare uniform benign VES with no couplet, triplet or VT. Result of invasive electrophysiological examination was evaluated as normal. Although no cardiac pathology that may impede surgery was detected, his parents refused and the patient was discharged.

DISCUSSION

The case described a pediatric patient who developed ventricular tachycardia after concomitant use of sevoflurane and phenylephrine. Arrhythmias during the course of anesthesia mostly occur during intubation. Tachycardia and hypertension provoked by tracheal intubation reduce myocardial perfusion and increase oxygen consumption.[3,4] Deep hypoxemia is highly correlated with ventricular arrhythmias. Mechanism of these hypoxia-provoked arrhythmias is not completely elucidated, but consumption of high-energy phosphate sources together with alteration of local acid-base and

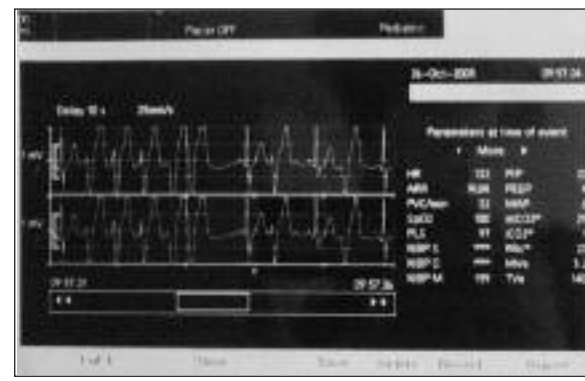

FIGURE 1: Idiopathic ventricular tachycardia.

electrolyte balance are believed to be responsible. In our case, blood pressure and pulse were within normal limits (110/70 mmHg, 80 beats/minute). Partial oxygen pressure (SpO_2) was around 98-100%. Disruption of acid-base balance either by hypercarbia or hypocarbia is associated with increased risk of arrhythmia. Respiratory acidosis during hypercarbia is a potent stimulator of sympathetic system; resultant catecholamine release enhances the arrhythmic effects of hypercarbia.[5] Hypocarbia is associated with respiratory alkalosis where plasma potassium concentration is decreased; thus hypocarbia is accused for hyperventilation-provoked arrhythmias.[5] Disturbances in potassium, calcium, or magnesium balance are the major electrolyte imbalances that may lead to cardiac arrhythmias. Particularly an underlying magnesium deficiency together with high levels of catecholamine increases the risk for sevoflurane- and halothane-induced arrhythmias.[5,6] But in our case $EtCO_2$ was 35-45 mmHg and there was no electrolyte imbalance.

Sevoflurane is usually preferred for anesthesia induction in children. Inhalation anesthetic agents are known to cause arrhythmias either alone or by interaction with other drugs or by potentializing the arrhythmic effects of circulating catecholamines.[7,8] However, the issue of whether intrinsic effects of inhalation anesthetics is arrhythmogenic or anti-arrhythmic is still controversial.[7,8] There are major differences between the inhalation anesthetics in terms of their interaction with catecholamines. This effect is highest with halothane and lowest with enflurane. Sevoflorane reduces myocardial contractility; this effect is similar to desflurane and isoflurane. However it does not potentialize epinephrine-induced cardiac arrhythmias.[9] Bernard et al[10] and Harkin et al[11] have reported a 30-40% increase in heart rate compared to awake-state after administration of 1.2-2 MAC sevoflurane.

Phenylephrine is a synthetic non-catecholamine agent used as a decongestant. Phenylephrine is absorbed through the mucosa. It leads to arteriolar and venous constriction by its strong alfa-1 agonistic effect.[12] Arteriolar vasoconstriction increases arterial pressure which might lead to reflex bradycardia.[12] The net effect of this agent on cardiac output depends on the interactions between afterload, preload and pulse rate. It is used in concentrations of up to 10% as a decongestant. Side effects in adults include hypertension, tachycardia and arrhythmias.[13] Tachycardia has been reported in the elderly and premature babies due to overdose.[12,13]

There is no universal agreement on phenylephrine dosage and concentration. The 2.5% phenylephrine eye drops are licensed for use in all age groups. Our case was a 12-year-old boy with no history of drug use. One drop of 2.5% phenylephrine was dropped into both nostrils in our patient. Bouts of VES were observed in about two minutes. Following 3-4 bouts of VES in a row, sevoflurane was discontinued and lidocaine was given, which turned the rhythm to normal sinus rhythm. Arrhythmic effects of phenylephrine are not uncommon in infants with low-birth weight and elderly hypertensive individuals.[12] However, our patient did not have any cardiac disorder. There was no overdose involved and concentration was within the normal limits. Sevoflurane was re-instated when the rhythm returned to normal sinus rhythm, but anesthesia was terminated again after bouts of VES repeated. Patient was taken under cardiologic examination but considered normal after holter, echocardiography and electrophysiologic. In our case, concomitant use of sevoflurane and phenylephrine was considered to be responsible for the arrhythmia since wide QRS complexes followed by sustained VT were observed after induction with sevoflurane and VT resolved spontaneously following discontinuation of sevoflurane and reoccurred when sevoflurane was resumed.

In conclusion, we believe that concomitant use of sevoflurane and phenylephrine might trigger VT since there was no cardiologic pathology or a condition that might provoke arrhythmia during the induction of anesthesia in our patient.

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