J Anesth (1995) 9:96–98

Journal of
Anesthesia

Anesthetic management for cesarean section in a patient with paroxysmal ventricular tachycardia: a case report

RYU OKUTANI[1], AYUMI FUJITA[2], and TAEKO FUKUDA[2]

[1] Department of Anesthesia, Hyogo Prefectural Nishinomiya Hospital, 13-9 Rokutanji-cho, Nishinomiya city, Hyogo, 662 Japan
[2] Department of Anesthesiology, Hyogo College of Medicine, 1-1 Mukogawa-cho, Nishinomiya city, Hyogo, 663 Japan

Key words: Cesarean section, Paroxysmal ventricular tachycardia, General anesthesia

Introduction

Paroxysmal ventricular tachycardia is usually associated with organic heart disease, but can occur in young persons without evidence of organic heart disease. Ventricular tachycardia (VT) in parturients in the absence of organic heart disease has also been reported [1–4], although its incidence is unknown. The anesthetic management of pregnant women with serious cardiac arrhythmias is an important aspect in the decision of whether or not to continue the pregnancy and medication. We report our experience in a patient with a history of VT who underwent cesarean section and discuss her anesthetic management.

Case report

The patient was a 28-year-old, 64-kg parturient who presented for cesarean section. She had undergone surgical repair of atrial septal defect (ASD) at the age of 8 years. Thereafter, she had experienced two syncopal episodes, at the ages of 18 and 26 years. Although she had undergone neurological and cardiological examinations, no abnormalities had been found. Her family history was unremarkable. At 37 weeks of gestation, she suddenly developed general fatigue and exertional dyspnea, and she was noted to have transient tachycardia but was not treated with medication. At 40 weeks of gestation, the patient was admitted for management of incessant ventricular tachycardia (heart rate: 265 beats/min) and fetal distress. Electrocardiography (ECG) revealed paroxysmal VT (Fig. 1). The arrhythmia did not respond to an intravenous injection of 100 mg of lidocaine, which caused chest discomfort and nausea. Consequently, procainamide (400 mg) was given intravenously, and the heart rate then declined to 100 beats/min and the blood pressure settled to 110/70 mmHg. About 15 min after this treatment, echocardiography revealed diffusely hypokinetic left ventricular wall motion, left atrial dilation, and an ejection fraction of 51%. No regurgitation of the mitral or aortic valve was found. Fetal echocardiography revealed a normal heart rate (144 beats/min). When the maternal episode of VT ceased, the fetal distress disappeared. At that time, emergency cesarean section was postponed. The results of blood chemistry and thyroid and adrenal hormonal analysis were within normal limits. Treatment with mexiletine (300 mg/day) and digoxin (0.125 mg/day) was commenced prior to cesarean section. No further VT episode was noted. Cesarean section was scheduled for the 3rd day, when it was assumed that the blood concentrations of the prescribed drugs would be sufficiently high.

No premedication was given. The preoperative ECG and electrolyte (including plasma potassium) findings were normal. In the operating room, a pulse oximeter and 5-lead ECG monitor were applied, and a right radial artery catheter was inserted under local anesthesia for continuous blood pressure monitoring. Anesthesia was induced with thiamylal (150 mg), and vecuronium bromide (8 mg) was given for neuromuscular blockade. An endotracheal tube was then inserted, and anesthesia was maintained with a low concentration of isoflurane plus 66% nitrous oxide in oxygen. At the beginning of the operation, ECG revealed transient atrial flutter, with a heart rate of 120–140 beats/min, while the blood pressure (125/68 mmHg) was not markedly changed. A

Address correspondence to: R. Okutani
Received for publication on July 14, 1993; accepted on September 30, 1994

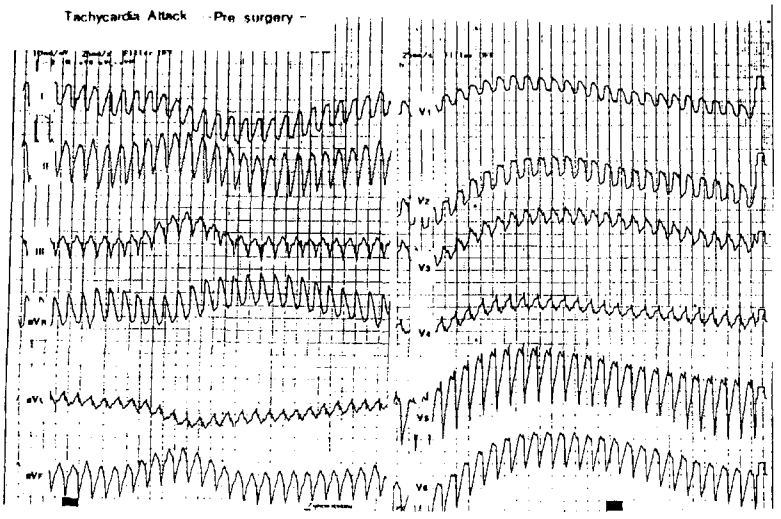

Fig. 1. Electrocardiogram demonstrating ventricular tachycardia at a rate of about 250 beats per minute

healthy 3054-g neonate was delivered about 8 min after induction of anesthesia; the Apgar score was 8 and 10 at 1 and 5 min after birth, respectively. After safe delivery of the neonate, the patient's heart rate remained elevated at 135 beats/min, while the blood pressure was 125/72. Diltiazem 5 mg was administered to decrease the heart rate, but it also elicited an attack of Wolff-Parkinson-White (WPW) syndrome and first degree A-V block. Although only transiently, her heart rate declined to 60–70 beats/min. The operation was completed without any complications. The neuromuscular blockade was reversed with slow administration of neostigmine 3 mg and atropine 1.5 mg, and the tracheal tube was removed.

Postoperatively, the patient developed an episode of VT, at the rate of 170–230 beats/min on days 2 and 10, even though she was receiving mexiletine 150 mg/day and digoxin 0.25 mg/day orally. Each episode of VT, like the preoperative episode, was treated with a bolus administration of procainamide and disappeared quickly. She was discharged 20 days after delivery, still requiring medication. After discharge, no further VT episode has been noted 3 months after cesarean section.

Discussion

Since McMillan and Bellet first reported a case of development of VT in a pregnant woman [1], several reports have been published [2–6]. However, in all the reported cases the fetus was delivered vaginally. Pregnancy is frequently associated with atrial and/or ventricular tachycardia due to increased ectopic activity. In general, thyrotoxicosis has been recognized as a major cause of atrial fibrillation during pregnancy. Structurally abnormal heart diseases such as mitral valve prolapse, cardiomyopathy, and long QT syndrome, are recognized to cause VT. However, there are many reports documenting paroxysmal VT in pregnant patients without evidence of organic heart disease [4,5], as occurred in our case. The following factors are reported to induce idiopathic VT: emotional stress, fear, exercise, coffee consumption, heavy smoking, alcohol, trauma, changes of posture, hypokalemia, and autonomic nervous system imbalance [7].

The principles of management in patients with arrhythmia include treatment of underlying disease such as hyperthyroidism or mitral stenosis [8], administration of appropriate antiarrhythmic drugs, and elimination of exacerbating factors such as pain, stress, and postural changes. In pregnancy, the use of antiarrhythmic drugs must be considered with caution, not only due to the possible proarrhythmic effects [9], such as the association of quinidine, procainamide, and disopyramide with marked prolongation of the maternal Q-T interval, but also due to the possible effects on the fetus both before and after birth [10]. However, among the various antiarrhythmic drugs, quinidine [11], procainamide [10], lidocaine [11], and mexiletine [12] can be used safely with exposure of the developing fetus. There is also no contraindication for digitalis and Ca^{++} antagonists [13] during pregnancy and labor. Amiodaron [13,14], a benzfuran derivative and a type II (Vaughan-Williams classification) antiarrhythmic agent, affects thyroid function and may cause hypothroidism or hyperthyroidism in the fetus. β-Blocker therapy during pregnancy has been utilized in patients with hypertension [15], hyperthyroidism, ideopathic hypertrophic subaortic stenosis [16], and cardiac arrhythmia. The use of propranolol may result in intrauterine growth retardation and fetal apnea, bradycardia, hypoglycemia, hypocalcemia, hyperbilirunemia,

and polycythemia [17,18]. Phenytoin is an alternative choice as an antiarrhythmic drug in patients with digitalis toxicity. Fetal hydantoin syndrome [19,20] is characterized by motor and mental retardation, microcephalia, nail hypoplasia and congenital abnormalities, such as heart defects and cleft palate.

Schroeder and Harrison [21] reported a case in which multiple cardioversions were performed to treat refractory paroxysmal atrial and ventricular tachycardia during pregnancy. It appears that this therapy is safe and effective in the treatment of serious arrhythmia even during pregnancy. However, we believe that the effects of cardioversion are still not well understood. In the anesthetic management of a patient with life-threatening ventricular arrythmia, if a VT episode occurs intraoperatively, an attempt should first be made to deliver the fetus, employing intravenous administration of lidocaine, mexiletine and/or procainamide, and cardioversion should be conducted only when the patient no longer responds to the medication.

It is also important to control the patient's anxiety, pain, and stress, since pain stimulation increases endogenous catecholamine release, which promotes the occurrence of severe arrhythmia.

In regard to the selection of the anesthetic technique, epidural or spinal anesthesia is most commonly used. However, spinal or epidural anesthesia often increase patient anxiety, which increases endogeneous catecholamine release with the associated increased risk of severe arrhythmia. Furthermore, the incidence of supine hypotension syndrome is higher under spinal or epidural anesthesia. For these reasons, we selected general anesthesia in our patient. Care must be taken when atropine and neostigmine are administered to antagonize the effect of neuromuscular blocking agents, since both can induce arrhythmia. We administered these drugs slowly and under close ECG monitoring, and no arrhythmia was induced. In this patient, however, two VT episodes occurred postoperatively, even though the patient was receiving antiarrhythmic drugs. Reexamination by cardiologists did not reveal any organic heart disease, and no episodes of VT were noted during the 3-month follow-up period after cesarean delivery without drug therapy. Thus, the VT was assumed to have been transient, accompanying the pregnancy.

In conclusion, we report our experience in the anesthetic management of a patient who had a VT episode during attempted cesarean section. Antiarrhythmic drugs against VT were given first, and the cesarean section was performed under general anesthesia 3 days later, when the blood concentration of the drugs was thought to have reached sufficiently high levels. The fetus was delivered safely.

References

1. McMillan TM, Bellet S (1931) Ventricular paroxysmal tachycardia: Report of a case in a pregnant girl of sixteen years with an apparently normal heart. Am Heart J 7:70–78
2. Kinugawa T, Fujimoto Y, Miyakoda H, Ogino K, Shigemasa C, Hasegawa J, Kotake H, Mashiba H (1989) A case of paroxysmal ventricular tachycardia during pregnancy. Jpn Circ J 53:807–812
3. Brodsky MA, Sato DA, Oster PD, Schmidt PL, Chesnie BM, Henry WL (1986) Paroxysmal ventricular tachycardia with sycope during pregnancy. Am J Cardiol 58:563–564
4. Yamada T, Kasanuma H, Yuri A (1987) Changes in arrhythmias during pregnancy using Holter ECG. Jpn J Electrocardiol 7:442–450
5. Chandra NC, Gates EA, Thamer M (1991) Conservative treatment of paroxysmal ventricular tachycardia during pregnancy. Clin Cardiol 14:347–350
6. Brodsky M, Doria R, Allen B, Sato D, Thomas G, Soda M (1992) New-onset ventricular tachycardia during pregnancy. Am Heart J 123:933–941
7. Menon KPS, Mahapatra RK (1984) Paroxysmal ventricular tachycardia associated with Bell's palsy in a teenager at late pregnancy. Angiology 35:534–536
8. Burch GE (1977) Heart disease and pregnancy. Am Heart J 93:104–116
9. Ruskin JN, McGovern B, Garan H, Di Margo JP, Kelly E (1983) Antiarrhythmic drugs. A possible cause of out-of-hospital cardiac arrest. N Engl J Med 309:1302–1306
10. Tamari I, Eldar M, Rabinowitz B, Neufeld HN (1982) Medical treatment of cardiovascular disorders during pregnancy. Am Heart J 104:1357–1363
11. Morishima HO, Heymann MA, Rudolph AM, Barrett CT, James LS (1975) Transfer of lidocaine across the sheep placenta to the fetus. Hemodyanamic and acid-base response of the fetal lamb. Am J Obstet Gynecol 122:581–588
12. Timmis AD, Jackson G (1980) Mexiletine for control of ventricular dysrhythmias in pregnancy Lancet 2:647–648
13. Rey E, Duperron L, Gauthier R, Lemay M, Grignon A, LeLorier J (1985) Transplacental treatment of tachycardia-induced fetal heart failure with verapamil and amiodarone: a case report. Am J Obstet Gynecol 153:311–312
14. Rey E, Bachrach LK, Burrow GN (1987) Effects of amiodarone during pregnancy. Can Med Assoc J 136:959–960
15. Eliahou HE, Silverberg DS, Reisen E, Romen I, Mashiach S, Serr DM (1978) Propranolol for the treatment of hypertension in pregnancy. Br J Obstet Gynaecol 85:431–436
16. Sabom MB, Curry CR, Wise DE (1978) Propranolol therapy during pregnancy in a patient with ideopathic hypertrophic subaortic stenosis: is it safe? South Med J 71:328–329
17. Schoenfeld N, Epstein O, Nemesh L, Rosen M, Arsmon A (1978) Effects of propranolol during pregnancy and development of rats. I. Adverse effects during pregnancy. Pediatr Res 12:747–750
18. Lieberman BA, Stirrat GM, Cohen SL, Beard RW, Pinker GD, Belsey E (1978) The possible adverse effect of propranolol on the fetus in pregnancies complicated by severe hypertension. Br J Obstet Gynaecol 85:678–683
19. Hanson JW, Myrianthopoules NC, Harvey MAS, Smith DW (1976) Risks to the offspring of women treated with hydantoin anticonvulsants, with emphasis on the fetal hydantoin syndrome. J Pediatr 89:662–668
20. Hanson JW, Smith DW (1975) The fetal hydantoin syndrome. J Pediatr 87:285–290
21. Schroeder JS, Harrison DC (1971) Repeated cardioversion during pregnancy. Treatment of refractory paroxysmal atrial tachycardia during 3 successive pregnancies. Am J Cardiol 27:445–446