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Postoperative Life-Threatening Recurrent Ventricular Arrhythmia Triggered by the Swan-Ganz Catheter in a Patient Undergoing Off-Pump Coronary Artery Bypass Surgery

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Recurrent ventricular arrhythmia can be fatal and cause serious complications, particularly when it is caused immediately after an operation. Incorrect placement of a Swan-Ganz catheter can trigger life-threatening ventricular arrhythmia, but even intensive care specialists tend to miss this fact. Here, we report a case of recurrent ventricular arrhythmia causing a severe hemodynamic compromise; the arrhythmia was induced by a severely angulated Swan-Ganz catheter. The recurrent ventricular arrhythmia was not controlled by any measures including repositioning of the catheter, until the complete removal of the Swan-Ganz catheter. It is necessary to keep in mind that the position of the pulmonary artery catheter should be promptly checked if there is intractable recurrent ventricular arrhythmia.

Key words: 1. Arrhythmia
2. Swan-Ganz catheter

CASE REPORT

A 72-year-old male patient visited SMG-SNU Boramae Medical Center with exertional chest pain, which had been aggravated during the most recent 3 months. The initial electrocardiogram revealed sinus bradycardia with a heart rate of 50 beat per minute, and there was no ST segment elevation. The serum cardiac enzyme level was within the normal range, and there were no abnormal findings except mild functional mitral regurgitation in transthoracic echocardiography, which showed a left ventricular ejection fraction of 67% and no regional wall motion abnormality. Myocardial single-photon emission computed tomography showed a reversible perfusion decrease at the basal inferior wall. Preoperative coronary angiography showed 70% to 80% stenosis of the left anterior descending coronary artery (LAD), 90% stenosis of the proximal ramus artery, 50% stenosis of the proximal left circumflex artery, 95% stenosis of the distal right coronary artery, and 90% stenosis of the posterolateral branch artery (PLB). According to the patient's past medical history, he had undergone right upper lobectomy for squamous lung cancer 3 years ago. In addition, arteriosclerosis obliterans of both the lower extremities had been treated medically for 6 years.

Diagnosed with unstable angina with three-vessel disease, the patient underwent off-pump coronary artery bypass graft surgery (CABG). Under general anesthesia, an advanced venous access catheter and a Swan-Ganz (SG) catheter were in-

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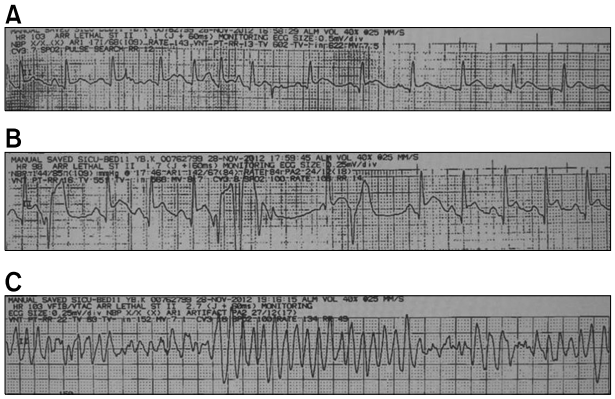

Fig. 1. Continuous electrocardiography monitoring shows the frequent development of malignant ventricular arrhythmia. (A) No ST segment elevation but unstable rhythm. (B) Development of ST segment elevation and frequent premature ventricular contraction. (C) Frequent ventricular fibrillation.

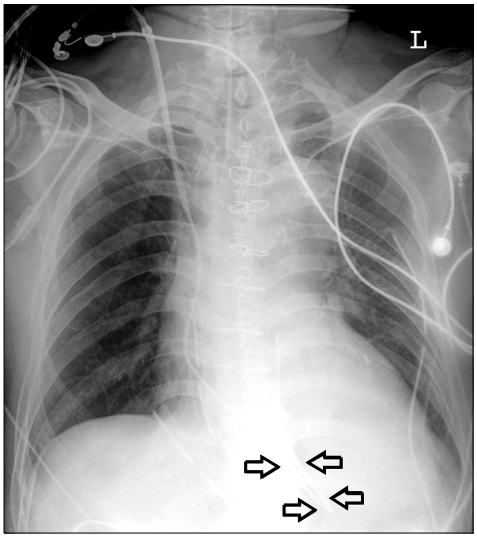

Fig. 2. Chest radiography shows the Swan-Ganz catheter located too deep in the right ventricle.

serted via the right internal jugular vein by the anesthesiologist. Several premature ventricular contractions (PVCs) occurred during the insertion of the SG catheter. We used the left internal mammary artery as a pedicled graft and anastomosed it to the mid-LAD. We also harvested the saphenous vein from the left lower leg and anastomosed it to the left internal mammary artery to make a Y-shaped composite graft. Subsequently, the vein graft was anastomosed to the ramus artery and PLB sequentially. During the operation, there were no events, although frequent PVC occurred.

As soon as the patient was transferred to the intensive care unit, frequent ventricular tachyarrhythmia began to occur and the hemodynamics became unstable. We tried our best to control the unstable ventricular arrhythmia with the administration of antiarrhythmic drugs like lidocaine and amiodarone as well as a meticulous correction of the fluid and electrolyte imbalance, but none of the medical measures worked. Furthermore, ventricular fibrillation (VF) occurred three times at intervals of about 1 hour (Fig. 1).

Considering reperfusion injury to be the cause of VF, we inserted an intra-aortic balloon pump to provide hemodynamic support, but failed to stop recurrent VF, and the patient's condition worsened considerably. In fact, we did not notice the severely angulated SG catheter demonstrated by the serial chest radiographs until around 6 hours after the patient was brought to the intensive care unit. We found that the SG catheter was located too deep in the right ventricle and relocated the catheter to remove the acute angulation (Fig. 2). However, the catheter still seemed to readily stimulate the ventricle; thus, we decided to remove the catheter. During the removal of the SG catheter, VF occurred once again. The removal of the catheter finally resulted in prompt and complete cessation of recurrent ventricular arrhythmia. There were, in total, 5 events of VF and defibrillation was performed 9 times. Unfortunately, the delayed removal of the problematic catheter led to considerable myocardial damage. The myocardial contractility, which was shown to be good by transesophageal echocardiography at the end of the operation, worsened and led to a left ventricular ejection fraction of 43%. The serum troponin-I level was also markedly elevated from an immediate postoperative value of 2.0 ng/mL to a peak value of 45.7 ng/mL on postoperative day 1.

Despite this event, the patient recovered and was transferred to the general ward on postoperative day 6 without any significant complications. Myocardial function was fully recovered without any regional wall motion abnormality, but the saphenous vein harvest site was dehisced and the wound problem had to be addressed. The patient was discharged in a good condition 21 days after the operation.

DISCUSSION

The first presentation of sustained ventricular arrhythmia in the recovery period after CABG is uncommon, and its incidence in the previous studies ranged from 1.6% to 3.1%. The occurrence of ventricular tachycardia (VT) or VF after CABG has been reported to have a strong impact on in-hospital mortality, which ranged from 21.7% to 25% [1,2]. Recurrent ventricular arrhythmia was not infrequent, observed in 33% of the patients, and was associated with a high mortality; 75% of the patients with these characteristics did not survive to hospital discharge [1]. The known risk factors for ventricular arrhythmia after CABG are age under 65 years, female gender, body mass index under 25 kg/m^2, unstable angina, moderate or poor ejection fraction, and a need for preoperative inotropic support or an intra-aortic balloon pump [2].

As an SG catheter allows the measurement of hemodynamic variables that cannot be assessed reliably or continuously by less invasive means, it is widely used in the perioperative monitoring of cardiac surgical procedures in many centers [3]. However, a significant number of patients who undergo pulmonary artery catheterization experience catheter-related complications such as cardiac arrhythmia, pulmonary infarction, catheter knotting, catheter entanglement with other endovascular structures, pulmonary artery rupture, thrombosis, and tricuspid valve rupture [3,4].

Various types of arrhythmia related to the SG catheter placement have been described and include premature atrial contraction, atrial and ventricular tachycardia, PVC, and conduction abnormalities. The incidence of arrhythmia has been reported to range from 12.5% to more than 70% during SG catheter insertion [3]. Some cite a 52% to 68% incidence of PVC, the most commonly seen type of arrhythmia [3,5]. Non-sustained VT is also common [5]. These resolve either with the SG catheter advancement from the right ventricle into the pulmonary artery, or with the prompt SG catheter withdrawal into the right atrium or the superior vena cava. Fortunately, clinically significant VT or VF requiring treatment occurs in $<1\%$ of the patients, although fatal VT has been observed [4,5]. The risk factors for ventricular arrhythmia during the insertion of an SG catheter include prolonged catheterization time, a predisposition to PVC, liver transplantation, myocardial infarction or ischemia, shock, acidosis, sepsis, electrolyte disturbances, and increased sympathetic tone [3,4].

Baldwin and Heland [6] reported a 2% incidence of VT associated with the removal of the SG catheter. In this report, the onset of VT was associated with a brief period of hypotension, and the duration of VT and hypotension was longer than the period of catheter removal, which implies that the activity is triggered by the procedure and has the potential to be prolonged beyond an initial stimulus.

In patients with the Wolff-Parkinson-White (WPW) syndrome, pre-excited atrial fibrillation with a rapid ventricular response can induce potentially life-threatening arrhythmia [7,8]. A previous prospective study reported that the incidence of VF with cardiac arrest among the WPW syndrome patients was 1.6% (3 of 184 patients) [8]. Although it is not relevant to our case, the WPW syndrome is thought to be one of the predisposing factors of VF.

In our case, recurrent VF, which was intractable with medical treatment, occurred after CABG. There was no risk factor for ventricular arrhythmia except unstable angina, and VF was thought to be triggered by the malpositioned SG catheter. The inserted length of the SG catheter was within the usual range because the kinking of the catheter masked the malposition. Checking the pulmonary capillary wedge pressure could be helpful in detecting the malposition of the catheter, but we did not try it until we realized the cause of the VF.

In conclusion, it is basic and imperative to check the position of the SG catheter and to keep in mind that life-threatening and otherwise intractable recurrent ventricular arrhythmia can be triggered by a malpositioned catheter.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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