Satisfactory recovery of consciousness after prolonged cardiopulmonary resuscitation due to unexpected hemorrhage during a conduit ileum examination

MARIKO HAGA, KATSUMI HARASAWA, YUJI MORIMOTO, and OSAMU KEMMOTSU

Department of Anesthesiology and Critical Care Medicine, Hokkaido University Graduate School of Medicine, N-15, W-7, Kita-ku, Sapporo 060-8638, Japan

Key words Cardiac arrest · Massive hemorrhage · Hypothermia

Introduction

Intraoperative cardiac arrest is rare but is directly lifethreatening [1–3]. Anesthesiologists usually prepare for massive bleeding in cases in which such a situation is likely. We report a case of a fiberoscopic urological examination complicated by cardiac arrest due to unexpected, uncontrolled bleeding, which resulted in satisfactory recovery of consciousness after prolonged cardiopulmonary resuscitation.

Case report

A 61-year-old male patient was admitted to our university hospital to undergo surgical repair of a stenosis between the left ureter and a conduit ileum anastomosis. Total cystectomy had already been performed for the bladder tumor, and the ileum had been used as a conduit. One and a half months after the surgical repair, a fiberoscopic examination was scheduled for unidentified bleeding from the conduit. The patient had also been suffering from idiopathic interstitial pneumonia and allergic vasculitis that had been treated with 20 mg of prednisolone per day.

After placement of a venous cannula in the left hand, general anesthesia with fentanyl, sevoflurane in oxygen, and nitrous oxide was induced and the trachea was intubated after being facilitated by vecuronium. To monitor arterial pressure and to analyze arterial blood gases, the left dorsalis pedis artery was cannulated after the patient was put in the dorsosacral position with both arms fixed along the trunk. Three hours later, massive bleeding occurred and the arterial pressure fell suddenly when the urologists were examining a protuberance in the conduit. Intravenous administration of 1 mg of epinephrine and concentrated red cells were used to restore circulation. The rectal temperature fell slightly (to 33.9°C at the lowest) after the onset of bleeding and then recovered to 35.7°C. The urologists planned to control the bleeding by asking for interventional angiography. One hour after the event, a radiologist found a bleeding point at the right external iliac artery. During subsequent urgent embolization, the arterial pressure critically dropped again due to rebleeding, and the cardiac rhythm deteriorated to ventricular fibrillation. Manual chest compression under controlled ventilation was immediately started for cardiac arrest, followed by head surface cooling with ice packing. Although intravenous epinephrine was given repeatedly in doses of 1 to 5mg and electrical defibrillation was performed several times, the monitored ECG continued to show ventricular fibrillation. At the time of the second shock, the rectal temperature was 35.7°C, which was followed by a gradual decrease in temperature to a low of 33.1°C. Vascular surgeons were requested to help and soon began to control the bleeding; however, it was difficult to detect the bleeding point because of the previous laparotomy. At one and a quarter hours after the second bleeding event, the QRS complexes were transiently restored on the ECG, and end-tidal carbon dioxide pressure waveforms showed temporary recovery of the pulmonary circulation. This recovery did not continue for more than 10min. Both pupils were 9mm in diameter. After 2h of resuscitation, the vascular surgeons finally succeeded in controlling the bleeding, and a spontaneous arterial pulse was regained on the monitor. A femoro-femoral bypass was placed to perfuse the right lower extremity after the right external iliac artery was ligated. The total amount of bleeding

Address correspondence to: K. Harasawa

Received: July 25, 2002 / Accepted: February 25, 2003

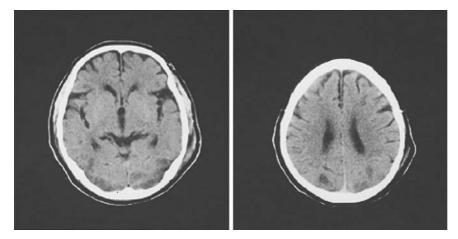


Fig. 1. Brain computed tomographic (CT) scan 4 days after the surgery shows infarction areas mainly in the occipital lobes. The left side of each image is the right side of the patient

reached 12940 ml. Severe acidosis continued after the successful resuscitation, and the lactate level was $22.7 \text{ mmol} \cdot l^{-1}$ at the end of surgery.

The patient was transferred to the intensive care unit (ICU), and hypothermic therapy targeted around 34°C was initiated with the use of a cooling blanket. Dopamine and dobutamine were continuously infused to maintain circulation and to enhance diuresis. Human atrial natriuretic peptide was also used to facilitate urine production; however, continuous hemodiafiltration was required until the patient left the ICU. Intravenous lidocaine was also administered for 5 days to prevent lethal ventricular arrhythmias. The day after the surgery, the patient was sedated with $100 \mu g \cdot h^{-1}$ of fentanyl, followed by infusion of thiamylal $(1-3 \text{ mg} \cdot \text{kg}^{-1} \cdot \text{h}^{-1})$ for 5 days thereafter. About 12h after the patient was admitted to the ICU, the presence of cerebral blood flow was confirmed by Doppler ultrasonography. Between the 2nd and 6th postoperative days (PODs), the body temperature remained around 35°C. Light reaction of the eyes was regained on the 3rd POD; however, cerebral infarction was detected mainly in the occipital lobes at POD 4 (Fig. 1). On the 7th POD the patient began to frown during oral care, and spontaneous eye opening was recognized on the 11th POD. On POD 13 he started to react to verbal commands, for example, by opening his mouth and putting his tongue in front of his lips. On POD 14 he was discharged to the urology ward. On the 21st POD he went into hemorrhagic shock again on the ward, and surgical control was again performed. After the last surgery, he still could communicate using a "character panel" until he died from complicated pulmonary bleeding on POD 64.

Discussion

We could not expect that this patient's consciousness would return to a satisfactory level after a prolonged, 2-h resuscitation process. It is speculated that an important factor that allowed this patient to regain consciousness was the lowered body temperature during the resuscitation period [4,5]. After the first massive bleeding from the iliac pseudoaneurysm, a large quantity of plasma expander kept at room temperature and cooled red cell transfusions were administered to treat the shock. The patient's rectal temperature was around 35°C when he underwent the second episode of shock. The temperature further decreased during the resuscitation period down to 34°C. Sterz et al. [5] reported that a better neurological outcome was obtained in mildly hypothermic dogs than in normothermic dogs during and after resuscitation. Kuboyama et al. [6] reported that hypothermia induced immediately at the onset of a critical event was necessary for neuronal protection to be expected. Incidental hypothermia already existed in this case at the time of cardiac arrest, and that may have been the most beneficial factor. Internal body cooling, such as that produced during induction of cardiopulmonary bypass or by other external circulatory support systems, is effective to control body temperature but requires special techniques and equipments. External body cooling is not as rapidly effective but is easy to perform. Although we cooled the patient's head in this case [7], its effectiveness for protection of the brain was not clear. In fact, Corbett and Laptook [8] reported that localized head cooling failed to reduce the brain temperature. Moreover, in the study of Takasu et al. [9], surface cooling failed to reduce the core temperature rapidly and hastened death. However, target brain temperatures may be achieved by surface cooling in small children [4,10].

Massive bleeding is sometimes unpredictable yet often unavoidable. This case indicates that hypothermia, whether it is incidental or not, may be effective with regard to cerebral resuscitation in the treatment of massive hemorrhagic shock. Acknowledgment. The authors are grateful to Edward Bertaccini, M.D., of VA Palo Alto Health Care System and Stanford University for his kind revision of this manuscript.

References

- Olsson GL, Hallen B (1988) Cardiac arrest during anaesthesia. A computer-aided study in 250,543 anaesthetics. Acta Anaesthesiol Scand 32:653–664
- Keenan RL, Boyan CP (1991) Decreasing frequency of anesthetic cardiac arrests. J Clin Anesth 3:354–357
- Tikkanen J, Hovi-Viander M (1995) Death associated with anaesthesia and surgery in Finland in 1986 compared to 1975. Acta Anaesthesiol Scand 39:262–267
- Laptook AR, Corbett RJT, Sterett R, Burns DK, Garcia D, Tollefsbol G (1997) Modest hypothermia provides partial neuroprotection when used for immediate resuscitation after brain ischemia. Pediatr Res 42:17–23

- Sterz F, Safar P, Tisherman S, Radovsky A, Kuboyama K, Oku K (1991) Mild hypothermic cardiopulmonary resuscitation improves outcome after prolonged cardiac arrest in dogs. Crit Care Med 19:379–389
- Kuboyama K, Safar P, Radovsky A, Tisherman SA, Stezoski SW, Alexander H (1993) Delay in cooling negates the beneficial effect of mild resuscitative cerebral hypothermia after cardiac arrest in dogs: a prospective, randomized study. Crit Care Med 21:1348– 1358
- Tadler SC, Callaway CW, Menegazzi JJ (1998) Noninvasive cerebral cooling in a swine model of cardiac arrest. Acad Emerg Med 5:25–30
- Corbett RJT, Laptook AR (1998) Failure of localized head cooling to reduce brain temperature in adult humans. NeuroReport 9:2721–2725
- Takasu A, Ishihara S, Anada H, Sakamoto T, Okada Y (2000) Surface cooling, which fails to reduce the core temperature rapidly, hastens death during severe hemorrhagic shock in pigs. J Trauma 48:942–947
- Gelman B, Schleien CL, Lohe A, Kuluz JW (1996) Selective brain cooling in infant piglets after cardiac arrest and resuscitation. Crit Care Med 24:1009–1017