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External manual compression of the abdominal aorta to control hemorrhage from a ruptured aneurysm

Nobuhide Kin, Masakazu Hayashida, Kyung-ho Chang, Kanji Uchida, and Kazuo Hanaoka

Department of Anesthesiology, School of Medicine, The University of Tokyo, 7-3-1 Hongo, Bunkyo-ku, Tokyo 113-8655, Japan

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Introduction

Abdominal aortic aneurysm is a common, lethal disease. Recent advances in operative techniques, anesthesia, and perioperative intensive care have reduced the mortality rate for elective repair of abdominal aortic aneurysm to less than 5% [1, 2]. For ruptured aortic aneurysm, however, intraoperative and postoperative survival has not been improved substantially. Most medical centers still report a mortality rate of more than 50% for repair of ruptured abdominal aortic aneurysm [1, 2]. We report a patient of advanced age who survived repeated episodes of rupture of abdominal aortic aneurysm. In the second rupture episode, a series of procedures, including external manual compression and subsequent direct digital occlusion of the abdominal aorta, was effective in controlling the hemorrhage.

Case report

An 83-year-old woman, weighing approximately 50 kg, who had been receiving medication for hypertension for 20 years, presented at the emergency room at our institute because of severe abdominal pain, nausea, and vomiting. Her systolic blood pressure (SBP) was less than 70 mmHg. Blood analyses revealed mild anemia (hemoglobin [Hb], 10.1 g/dl) and metabolic acidosis (base excess, −7.7 mEq/l). The radial artery was cannulated, and a central venous catheter was inserted. With fluid infusion and subsequent blood transfusion, her SBP gradually increased. She underwent radiological examinations, including abdominal computed tomography (CT), which revealed an infrarenal aortic aneurysm 10 cm in diameter. Retroperitoneal hematoma, as well as fluid collection in the peritoneal cavity, suggestive of intraperitoneal spread of hemorrhage, was noted. Because of continuous volume resuscitation, her SBP exceeded 100 mmHg at the end of the radiological examinations. By the time she arrived at the operating room, she had received fluid and homologous blood amounting to approximately 2100 ml and 720 ml, respectively.

On arrival at the operating room, her SBP exceeded 130 mmHg. Her heart rate (HR) was 90 beats per min (Fig. 1). After she was placed on the operating table, however, her SBP fell abruptly, to less than 50 mmHg. Bradycardia immediately followed. After complaining of abdominal pain, the patient became comatose. Her abdomen showed marked swelling. An instantaneous diagnosis of re-rupture of the aneurysm was made, and an anesthesiologist compressed her epigastrium hard with his fist in an attempt to occlude the abdominal aorta between his fist and the patient's vertebrae. Meanwhile, another anesthesiologist intubated the trachea, using topical lidocaine. Then, general anesthesia was induced with fentanyl (200 μg), midazolam (5 mg), and vecuronium (8 mg). Simultaneously, atropine (0.5 mg) and norepinephrine (25 μg) were injected en bolus. Blood and fluid were administered aggressively. The patient's anterial blood pressure (ABP) and HR promptly responded to these treatments. With the continuous external manual compression of the abdominal aorta, the femoral pulse was not palpable on either side, while the radial SBP exceeded 100 mmHg within 5 min of the circulatory collapse, and exceeded 150 mmHg during the next 5 min, necessitating the cessation of volume loading and the initiation of vasodilator therapy, with nitroglycerine (0.5–3.0 $\mu g \cdot kg^{-1} \cdot min^{-1}$) and prostag-

Address correspondence to: M. Hayashida
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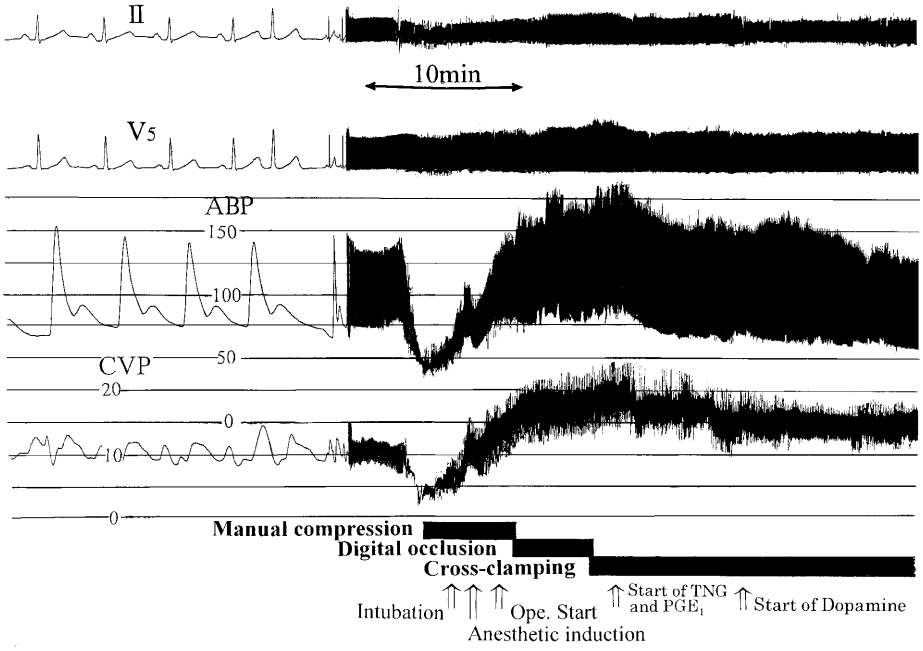

Fig. 1. Monitoring of trends in electrocardiogram (*II and V5*), arterial blood pressure (*ABP*), and central venous pressure (*CVP*) at the reccurrence of rupture of abdominal aortic aneurysm. *Manual compression*, External manual compression of the abdominal aorta; *digital occlusion*, direct digital occlusion of the suprailiac abdominal aorta via minilaparotomy; *cross-clamping*, cross-clamping of the infrarenal aorta; *TNG*, nitroglycerine; *PGE1*, prostaglandin E_1

landin E_1 (0.02–0.10 μg·kg^{-1}·min^{-1}) (Fig. 1). Anesthesia was maintained with isoflurane (1.0%–2.0%) in an air-oxygen mixture and fentanyl (1500 μg in total).

The whole abdomen was disinfected and draped, including the anesthesiologist's fist on the epigastrium. Then the anesthesiologist's fist was promptly replaced by a surgeon's gloved fist. A small midline incision was made between the xyphoid process and the surgeon's fist. Through this incision, another surgeon occluded the aorta directly and blindly, with his fingers, at the level of the diaphragmatic hiatus. Then the external manual compression was released. While the aorta was being digitally occluded, the midline incision was extended down to the pubic symphysis. Approximately 1200 ml of free blood was evacuated from the peritoneal cavity. During the isolation of the infrarenal aorta, active bleeding was not noted, although a massive hematoma was already present retroperitoneally. After cross-clamping of the infrarenal aorta, the digital aortic occlusion was released. The aneurysm was excised and replaced by a Dacron graft. The duration of cross-clamping and the duration of the surgery were 85 and 307 min, respectively. The total blood loss amounted to 7900 ml.

*P*CO2, *P*O2, base excess, pH, hematocrit, and arterial blood Hb changed from 57 mmHg, 570 mmHg (FIO2, 1.0), −14.1 mEq/l, 7.039, 15.2%, and 4.8 g/dl, respectively, at the beginning of surgery to 36 mmHg, 266 mmHg (FIO2, 0.5), −3.6 mEq/l, 7.373, 32.5%, and 12.7 g/dl, respectively, at the end of surgery, without the use of sodium bicarbonate. A total of 6800 ml of blood was transfused. Although anuria had persisted before surgery, diuresis resumed during surgery, after sufficient volume resuscitation and the IV administration of furosemide (100 mg), mannitol (40 g), dopamine (2–5 μg·kg^{-1} min^{-1}), prostaglandin E_1, urinastatin (300 000 units), and methylprednisolone (1 g). The patients urine output reached 2400 ml.

Postoperatively, she was transferred to the intensive care unit. The trachea was extubated on the third postoperative day. She recovered without major complications and was discharged from hospital, walking unaided, 3 weeks after the surgery.

Discussion

Ruptured abdominal aortic aneurysm is a major challenge for both anesthesiologists and surgeons. The survival rate of patients with ruptured abdominal aortic aneurysm still remains less than 50%. Advanced age (more than 80 years) and blood transfusion of more than 3000 ml are associated with a high mortality rate, of more than 70%. Further, hemorrhage into the peritoneal cavity is associated with an extremely high mortality rate (60%–97%) [1, 2]. When these unfavorable conditions were considered, our patient appeared to represent rare case of survival.

Even if patients survive the operation for ruptured aneurysm, the development of postoperative complications is highly probable [3]. In general, sufficient fluid resuscitation is crucial to prevent postoperative organ

failure in patients in circulatory shock [2, 3]. In the patient with ruptured aortic aneurysm, however, fluid resuscitation should be limited to avoid recurrent or progressive hemorrhage resulting from elevated blood pressure. Actually, some reports recommend that volume resuscitation should be commenced only when SBP falls below 70–80 mmHg [4–6]. In our patient, it was highly likely that elevated blood pressure, resulting from excessive fluid infusion, as well as blood transfusion, contributed to the reccurrence of the rupture. We should have monitored the volume resuscitation more strictly.

Without the application of the external manual compression of the abdominal aorta at the recurrence of the rupture, cardiac arrest might have ensued in the presence of hemorrhage into the peritoneal cavity. In children with tetralogy of Fallot, external compression of the abdominal aorta proved to be effective in damping pressure transmission within the aorta [7]. Also, in our patient, we did not feel a pulse over either femoral artery, while proximal arterial pressure increased beyond our expectation, even after the induction of deep anesthesia, which should have diminished sympathetic tone. Presumably, bleeding from the ruptured aneurysm was slowed or even stopped by the compression maneuver, through a marked reduction in the distal aortic blood pressure, and, because of this successful control of hemorrhage, the excessive increase in proximal arterial pressure ensued.

In the presence of such arterial hypertension, in particular, hemorrhage may recur after laparotomy, through the acute loss of the tamponading effect of the tight abdominal wall [2]. To prevent such a catastrophic event, we applied direct digital occlusion of the abdominal aorta, via a mini-laparotomy, before we extended the incision on the abdominal wall. With the digital aortic occlusion, the bleeding was controlled effectively during the isolation of the abdominal aorta. We continued this maneuver until the infrarenal aorta was cross-clamped.

Thus, hemorrhage from the ruptured aneurysm was effectively controlled, using all the consecutive procedures described above, i.e., external manual compression of the abdominal aorta, mini-laparotomy proximal to the site of the compression, digital occlusion of the suprailiac aorta via the mini-laparotomy, followed by the release of the external manual compression, the extension of the midline incision on the abdominal wall while the aorta was digitally occluded, and cross-clamping of the infrarenal aorta, followed by the release of the digital occlusion.

In conclusion, we have reported a woman of advanced age who survived repeated episodes of rupture of an abdominal aortic aneurysm. In this patient, a series of procedures, including external manual compression and subsequent direct digital occlusion of the abdominal aorta, was effective in controlling the hemorrhage.

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