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Anesthetic management of a patient with aortocaval fistula

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Abstract
Aortocaval fistula is a rare complication of ruptured abdominal aortic aneurysm (AAA), and patients with an aortocaval fistula show multiple symptoms. We report an 87-year-old man who was diagnosed as having an AAA with aortocaval fistula and who developed refractory hypotension after induction of anesthesia. Following a phenylephrine injection for slight hypotension induced by anesthetic induction, he developed severe hypotension and bradycardia, and his skin became cyanotic. Vasopressor agents had no immediate effect on the hypotension, but blood pressure gradually increased in about 30 min with continuous infusion of dopamine and noradrenaline. Transesophageal echocardiography (TEE) showed right ventricle (RV) hypokinesis and massive tricuspid regurgitation (TR). Central venous pressure (CVP) showed a remarkably high value. After the repair of the aortocaval fistula, the hemodynamics became stable, RV motion was improved, TR was reduced, and CVP became normal. Anesthetic management of the repair of an aortocaval fistula is very difficult. The hemodynamics changed dramatically throughout anesthesia in our patient with this disorder, even though low-dose anesthetics were used. For the successful treatment of this disorder, preparation for the operation is required before the induction of anesthesia, and urgent closure of the fistula is necessary after the induction of anesthesia. TEE is a useful tool for monitoring hemodynamics in such patients.

Key words Aortocaval fistula · Shock · Abdominal aortic aneurysm · Cardiac failure

Introduction

Aortocaval fistula is a rare complication of ruptured abdominal aortic aneurysm (AAA), but shows very severe clinical features [1,2]. We report the anesthetic management of a patient with ruptured AAA with aortocaval fistula who developed severe refractory hypotension, bradycardia, and cyanosis immediately after the induction of anesthesia despite the use of very low-dose anesthetics; transesophageal echocardiography (TEE) showed marked changes in right ventricle (RV) wall motion and the degree of tricuspid regurgitation (TR) before and after closure of the aortocaval fistula.

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Case report

An 87-year-old man, height 160 cm and weight 42 kg, was admitted as an emergency to our hospital with lower abdominal pain. Heart rate (HR) was 101 bpm and blood pressure (BP) was 90/50 mmHg. Laboratory findings were as follows: hemoglobin, 11.8 $g \cdot dl^{-1}$; blood urea nitrogen, 27 $mg \cdot dl^{-1}$; creatinine, 1.7 $mg \cdot dl^{-1}$; potassium, 5.4 $mEq \cdot l^{-1}$; and C-reactive protein, 3.19 $mg \cdot dl^{-1}$. Arterial blood gas (ABG) showed compensated metabolic acidosis (pH 7.417; Pa_{O_2} 87 mm Hg; Pa_{CO_2} 28 mmHg; bicarbonate 18.7 $mEq \cdot l^{-1}$; and base excess (BE) −5 $mEq \cdot l^{-1}$). The electrocardiogram showed incomplete right bundle branch block and left ventricle (LV) hypertrophy, and transthoracic echocardiography showed ejection fraction 68% and TR grade II-III. The chest radiogram showed congestion in the upper lung field. Enhanced computed tomography showed calcification of the abdominal aorta and AAA, with early enhancement of the inferior vena cava (IVC; Fig. 1). He was diagnosed with a ruptured AAA with aortocaval fistula and prepared for emergency abdominal aorta replacement grafting. We planned anesthetic induction for this patient using minimal doses of a benzodiazepine (midazolam), an opioid (fentanyl), and a muscle relaxant, with sevoflurane for maintenance, and monitoring of central venous pressure (CVP) and direct arterial pressure.

Vital signs in the operating room before induction of anesthesia were: HR, 116 bpm; BP, 96/48 mmHg; and

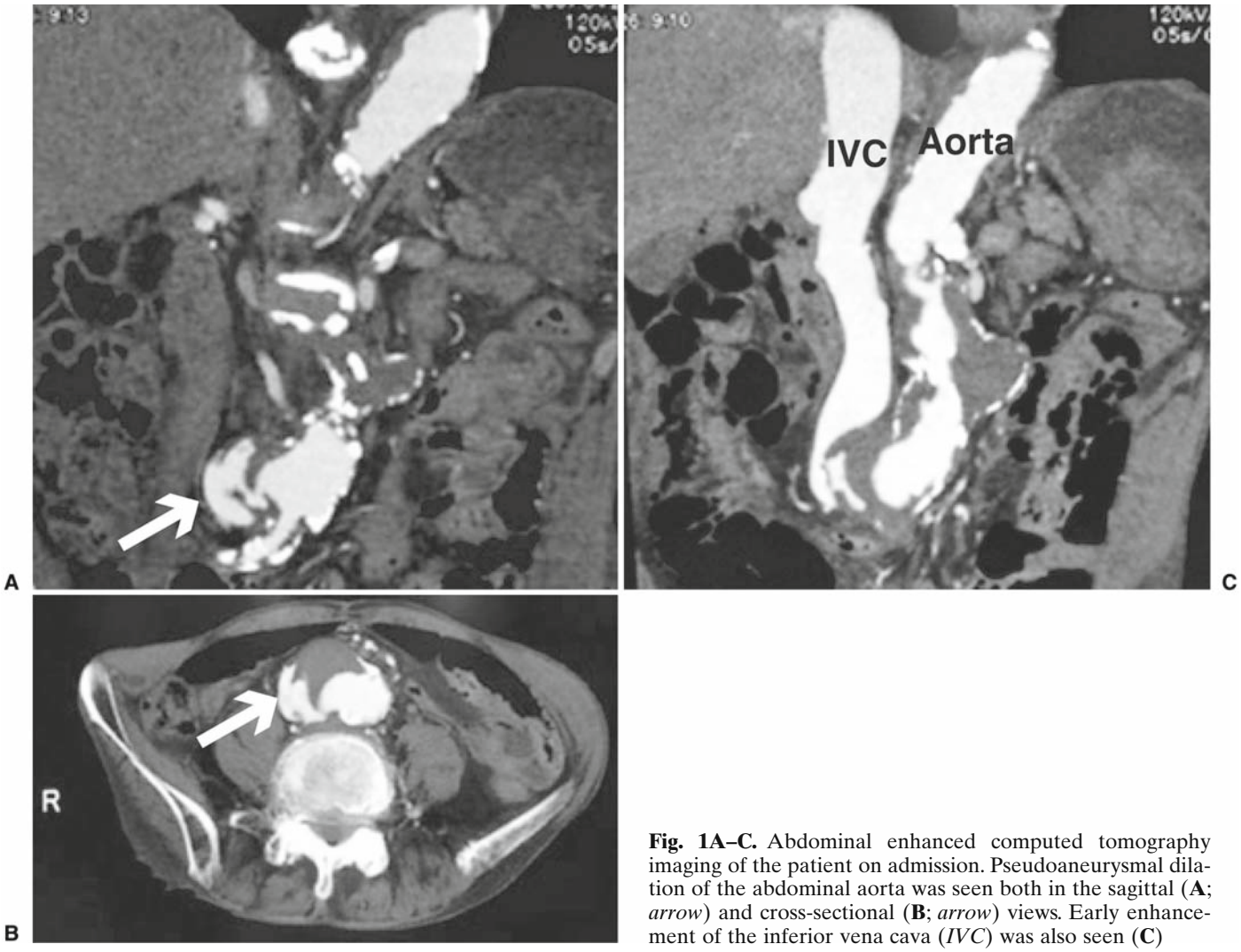

Fig. 1A–C. Abdominal enhanced computed tomography imaging of the patient on admission. Pseudoaneurysmal dilation of the abdominal aorta was seen both in the sagittal (**A**; *arrow*) and cross-sectional (**B**; *arrow*) views. Early enhancement of the inferior vena cava (*IVC*) was also seen (**C**)

oxygen saturation by pulse oximetry (S_{pO_2}), 100% under 5 l·min^{-1} oxygen mask. We secured peripheral venous access (20 gauge and 18 gauge), and volume resuscitation was performed using lactate Ringer solution with dextran 40 (Saviosol; Otsuka Pharmaceutical, Tokyo Japan) and normal saline. Anesthetic induction was performed with 50 µg fentanyl, 2 mg midazolam, and 5 mg vecuronium. Immediately after the injection of anesthetics, his BP dropped to 70/40 mmHg. After intravenous injection of 0.1 mg phenylephrine, further reduction of the BP and the HR occurred, to 40/20 mmHg and 60 bpm, respectively, and his skin became cyanotic. We intravenously injected a total dose of 20 mg ephedrine (4 mg, 8 mg, and 8 mg in separate doses), 1 mg noradrenaline (0.25 mg, 0.15 mg, 0.6 mg), and 4.5 mg adrenaline (1 mg, 0.5 mg, 1 mg, 1 mg, 1 mg), but BP did not increase immediately. Then the trachea was intubated and he was given continuous infusion of dopamine at 3 µg·kg^{-1}·min^{-1} and noradrenaline at 0.3 µg·kg^{-1}·min^{-1}. We tried to insert a central venous (CV) catheter from the internal jugular vein, and noticed the external jugular vein was swollen. After insertion of the CV catheter, CVP showed a remarkably high value, of 28 mmHg. TEE showed enlargement of the right atrium (RA) and RV; RV hypokinesis; normal LV wall motion; and color Doppler showed massive TR (Fig. 2). ABG showed further metabolic acidosis (pH 7.30; Pa_{O_2} 452 mmHg; Pa_{CO_2} 24 mmHg; bicarbonate 11.8 mEq·l^{-1}; and BE –13.3 mEq·l^{-1}), so we infused sodium bicarbonate. His BP rose gradually in about 30 min after the first reduction of his BP, and TEE showed improved RV wall motion, slightly decreased RV size, and unchanged TR. CVP values ranged from 12 to 18 mmHg.

The operation was begun as soon as possible. Before clamping of the abdominal aorta, the BP again decreased to 40/20 mmHg, but immediately after the clamping of the abdominal aorta, BP rose to 108/60 mmHg and CVP decreased from 18 to 10 mmHg. Then BP and other vital signs were stable with continuous infusion of both dopamine at 6–8 µg·kg^{-1}·min^{-1} and noradrenaline

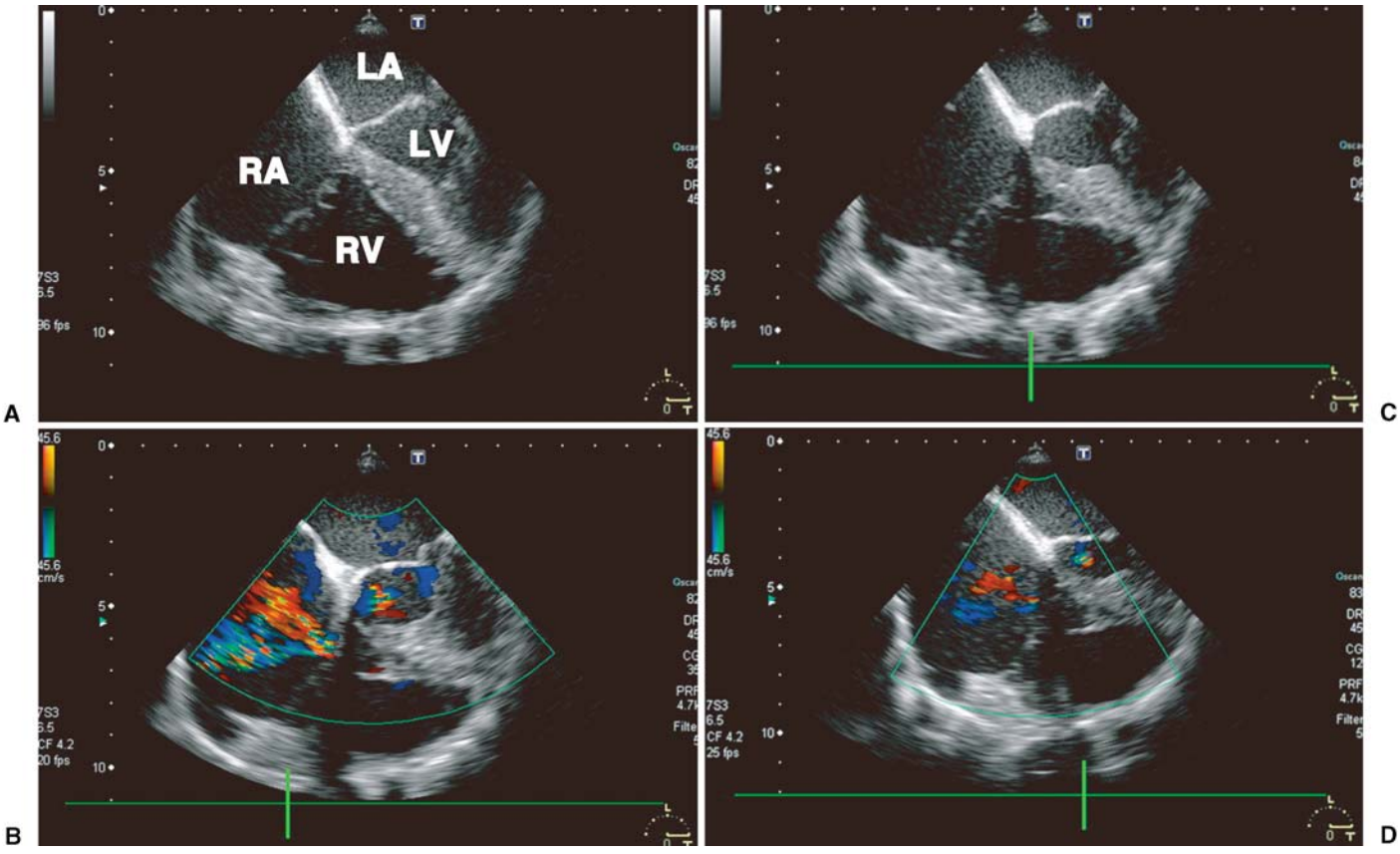

Fig. 2A–D. Four-chamber view of transesophageal echocardiography (TEE) imaging performed before (**A**, **B**) and after (**C**, **D**) aortocaval fistula repair. After the induction of anesthesia, TEE showed enlarged right atrium (*RA*) and right ventricle (*RV*; **A**) and color Doppler showed massive tricuspid regurgitation (**B**). After the repair of the fistula, enlargement of the RA and RV remained (**C**), but the tricuspid regurgitation was significantly reduced (**D**). *LA*, Left atrium; *LV*, left ventricle

at 0.1 $\mu g \cdot kg^{-1} \cdot min^{-1}$. After the repair of the fistula, TEE showed reduced, trivial TR and further improvement of RV wall motion, although enlargement of the RA and RV remained (Fig. 2C, D). CVP values ranged from 3 to 8 mmHg.

Anesthesia was maintained by continuous infusion of fentanyl at 2 $\mu g \cdot kg^{-1} \cdot h^{-1}$ and ketamine at a rate of 50 $mg \cdot h^{-1}$, not by sevoflurane, for more stable hemodynamics. His vital signs were kept stable until the end of the operation. He was transferred to the intensive care unit with an open wound, which was closed on postoperative day 4.

Discussion

Aortocaval fistula is a rare disease arising mostly from atherosclerotic changes of the aorta [1,2], and occasionally from traumatic [3], mycotic [4], iatrogenic [5], or connective tissue disorders [1,2]. Its reported incidence was 3%-4% in all ruptured aneurysms [1]. Its morbidity has been reported to be significantly high (10%–71%) [1,2]. Its clinical presentation depends on the amount of the shunt and progressive rates of hemodynamic changes [2,6], acute [2] or chronic presentation [7].

An aortocaval fistula increases venous return, which results in a hyperdynamic state, including increased cardiac output (CO) and HR and subsequently high-output cardiac failure, often with TR [2]. Increased peripheral venous pressure induces congestion of the peripheral circulation [2]. Renal dysfunction is very common [1,2]. Lack of a diagnosis before surgery is not rare [8].

The only effective treatment for this disease is operative closure of the fistula [1] or endovascular repair of the fistula [9,10]. An aortic cross clamp may normalize vital signs because of a decreased venous return, but also increases the afterload of the heart and may result in excessive hypertension [10,11]. In that case, vasodilating drugs should be carefully administered. In addition, hypotension due to decreased venous return should be managed.

In our patient, refractory hypotension occurred following a phenylephrine injection for the slight hypoten-

sion induced by anesthetic induction. Phenylephrine is an alpha-stimulating vasoconstrictive drug without positive inotropic effect and it may raise SVR excessively. The total amount of shunting and venous return would increase in consequence, and this would worsen RV function and induce refractory hypotension. TEE findings in our patient supported this hypothesis, showing an enlarged, hypokinetic RV, and relatively small LV, with normal systolic function and massive TR. The remarkably elevated CVP of 28 mmHg also supported this hypothesis. His cyanosis may have been the result of congestion or hypoperfusion of the peripheral circulation. Therefore, the use of phenylephrine was not indicated in this situation.

The reasons for the gradual increase in BP were unclear, but one possible reason was improved RV systolic function brought about by the infused catecholamines and the injected vasoactive drugs, or a decreased amount of shunting. These considerations were confirmed by the TEE findings at the time, in which the size of the RV was slightly decreased and the RV wall motion had improved compared with the initial findings. The decreased CVP would also support this hypothesis. After closure of the fistula, the hemodynamics became stable with the continuous infusion of catecholamine. TEE showed a markedly reduced degree of TR and further improvement in RV wall motion, although the RA and RV remained enlarged. CVP was further decreased after the closure of the fistula. These results confirmed the improvement of RV failure by the repair of the aortocaval fistula.

It was very difficult to keep hemodynamics stable in our patient, especially after the induction of anesthesia, despite the use of very low dose anesthetics. Severe hypotension, bradycardia, and asystole have been reported after the induction of anesthesia for the closure of an aortocaval fistula [11]. If we could have performed induction of anesthesia after the abdomen had been disinfected and draped, the duration of cardiovascular collapse would have been shorter. This is a very important point to note in hindsight in our case. Also, we should have secured a CV catheter before the induction of anesthesia, because this would have been useful, especially in an emergent vascular operation.

After our patient's recovery of BP, the hemodynamics was stable, with continuous infusion of fentanyl and ketamine, which seemed to be suitable for maintenance. A pulmonary artery (PA) catheter has been reported to be useful for measuring PA pressure, PA occlusive pressure, SVR, and CO [7], but in this emergency case there would have been no time to insert one. If we had inserted a PA catheter or collected blood samples from the central vein, elevated oxygen saturation would likely have been observed because of the influx of arterial blood into the IVC [2]. In this case, continuous central venous saturation ($ScvO_2$) monitoring, using a PreSep Oximetry Catheter (Edwards Lifesciences, Irvine, CA, USA) would have been useful to measure changes in $ScvO_2$ before and after closure of the fistula.

In a patient with aortocaval fistula, before the induction of anesthesia, it is necessary to secure large-diameter venous lines and a CV catheter, to prepare for the injection of all kinds of vasoactive drugs, and to disinfect and drape the abdomen. Induction of anesthesia is to be performed carefully with minimal doses of opioids and benzodiazepines, and immediately after induction, the surgeon should repair the fistula as soon as possible.

In summary, anesthetic management of the closure of an aortocaval fistula is very complicated and difficult. Careful induction and maintenance of anesthesia and urgent closure of the fistula are required for the patient's survival. TEE is a useful tool for monitoring hemodynamic changes in such patients.

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