Cardiac and Respiratory Arrest following Aortocaval Fistula

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Aortocaval fistula is an uncommon complication of lumbar disk surgery or is secondary to rupture of an abdominal aortic aneurysm. We treated abrupt asystole in a patient with aortocaval fistula which resulted from rupture of an infrarenal aortic aneurysm into the vena cava. The sudden onset of extreme bradycardia or asystole has been reported to occur during dissection of an aneurysm associated with aortocaval fistula[1,2]. It is postulated that acute decompensation due to a marked change in venous return to the heart or pulmonary embolism through the fistula may be the cause. We discuss here that reflex may be a potential mechanism for abrupt circulatory collapse.

Case Report

A 73-yr-old man with sudden onset of nausea, abdominal discomfort and weakness of lower limbs which occurred six hours before admission, was transferred to the emergency department for evaluation of ruptured abdominal aortic aneurysm. On admission, the blood pressure was 70/50 mmHg and the pulse rate 154 beats·min^{-1} with irregular rhythm. The jugular veins were distended, and the face and upper and lower extremities were cyanosed. At auscultation bibasilar moist rales and a loud S_3 gallop at the apex could be heard. The abdomen was distended but soft, and a pulsating abdominal mass was palpable. A bruit with systolic accentuation was audible at 2 qfb under the umbilicus and there was no pulse distal to the femoral arteries.

A chest roentgenogram showed perihilar markings with slight pulmonary congestion and a cardiothoracic ratio (CTR) of 0.55. A 12-leads electrocardiogram revealed atrial fibrillation, nonspecific ST segment changes in ${}_aV_f$, V_5 and V_6, and left ventricular hypertrophy. The echocardiogram showed a hyperdynamic wall motion. The abdominal ultrasonogram and the computed tomography revealed the presence of an aortic aneurysm but there was no evidence of fluid collection in the abdomen. A tentative diagnosis of unruptured abdominal aortic aneurysm was made and the patient was transferred to the intensive care unit (ICU).

On arrival in the ICU, the patient

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was alert and oriented. The blood pressure was 66/44 mmHg under continuous infusion of dopamine at a rate of 5 $\mu g \cdot kg^{-1} \cdot min^{-1}$ and the heart rate was about 158 beats·min^{-1} with atrial fibrillation. The patient was given oxygen at a concentration of 40% by a Venturi mask. Treatment in the ICU was initiated with rapid infusion of 5% albumin. However, no significant change in blood pressure was observed. Approximately 15 min after initiation of albumin infusion and while performing a follow-up echocardiogram, the heart rate abruptly decreased to 52 beats·min^{-1} followed by a loss of consciousness, respiratory arrest, progressive bradycardia and eventually asystole. External cardiac compression and endotracheal intubation were immediately begun and atropine 0.5 mg and epinephrine 1.0 mg were administered intravenously. There was immediate restoration of a heart rate of about 145 beats·min^{-1} with atrial fibrillation and a blood pressure of 80/60 mmHg. The patient regained consciousness, but controlled ventilation was begun. Dopamine, dobutamine and noradrenaline were started at a rate of 12 $\mu g \cdot kg^{-1} \cdot min^{-1}$, 5 $\mu g \cdot kg^{-1} \cdot min^{-1}$ and 0.5 $\mu g \cdot kg^{-1} \cdot min^{-1}$, respectively. Arterial blood drawn immediately after resuscitation showed a pH of 7.209, a Pa_{CO_2} of 32 torr and a Pa_{O_2} of 582 torr (F_{IO_2}=1.0) and a BE of −13.0. The metabolic acidosis was corrected by sodium bicarbonate. There was no evidence of myocardial ischemia on the echocardiogram at that time.

To assess the hemodynamic status, a pulmonary artery catheter was inserted; the central venous pressure was 12 mmHg, pulmonary artery pressure 33/12 mmHg, pulmonary capillary wedge pressure 22 mmHg and cardiac output 10 $l \cdot min^{-1}$ with the systemic pressure of 75/56 mmHg and the heart rate of 144 beats·min^{-1}. As an aortocaval fistula was suspected from venous hypertension, arterial insufficiency in the lower extremities and an abdominal bruit, blood gas was examined simultaneously both in the pulmonary artery and the femoral vein. The partial pressure of oxygen in the pulmonary artery was 68 torr, whereas that in the femoral vein was 23 torr. This indicated the presence of a left to right extracardiac shunt above the distal site of the aneurysm.

Six hours after the episode of cardiac arrest, emergency laparotomy was performed. Before the induction of anesthesia, the blood pressure was 95/50 mmHg and the heart showed normal sinus rhythm with a rate of 130 beats·min^{-1}. After aortic clamping, the aneurysm was incised. An arteriovenous fistula of 1.0 cm in diameter was found between the right common iliac artery and inferior vena cava. The fistula was closed with a patch, and the aorta was then reconstructed with a ducron bifurcation graft. Postoperatively, the hemodynamics remained stable for two days. The patient subsequently developed circulatory shock due to a massive bleeding from gastric cancer which had not been diagnosed on admission to the hospital. On the 3rd postoperative day, the patient underwent a partial gastrectomy. The course was further complicated by wound dehiscence and acute renal failure. The patient eventually died of multiple organ failure.

Discussion

Hemodynamic changes associated with aortocaval fistula are related to increases in venous return and decreases in total peripheral resistance due to diversion of aortic flow into the inferior vena cava. This produces a marked increase in preload and a marked decrease in afterload to the heart. The progression from a hyperdynamic state to cardiac decompensation depends on the size of the fis-

tula, shunt flow and cardiac reserve[3]. In our patient a fistula was thought to exist on admission, as an abdominal bruit and jugular vein distention were evident. However, it was speculated from both findings on the chest roentgenogram and the echocardiogram that the heart was not necessarily decompensated at that stage.

Asystole and respiratory arrest which suddenly occurred in the ICU may result from several causes, but the differential diagnosis can be divided conveniently into cardiac and noncardiac origins. A major cardiac cause is acute decompensation due to myocardial ischemia following increased cardiac work. A potential cause of noncardiac orgin is embolization of thrombus or atheromatous debris through the fistula. In our patient, data on blood gas and hemodynamic parameters obtained immediately after resuscitation excluded the possibility of pulmonary edema due to acute cardiac decompensation or massive pulmonary embolism. Myocardial ischemia was also unlikely from the findings on serial echocardiograms.

Abrupt onset and rapid recovery of circulatory collapse in our patient strongly suggest that reflex could be the cause. The circulatory collapse was probably preceded by a sudden increase in shunt flow. Administration of 5% albumin might promote an increase in preload. Right atrial stretch receptors produce a stretch-induced alteration in pacemaker activity[4]. An increase in venous return to the heart stimulates atrial stretch receptors and increases heart rate. This response, in conjunction with afterload reduction due to extracardiac shunt, might contribute to earlier hemodynamic changes, including tachycardia and hypotension in our patient. Similar stretch receptors are present in the pulmonary vascular beds and the left ventricle, and apnea, hypotension and bradycardia can occur through the chemoreflex or stretch reflex[5]. Afferent fibers in the vagal nerves innervate these stretch receptors and relay to the vasomotor and cardioinhibitory areas as well as the respiratory center in the medulla. It is likely that a marked distention of the pulmonary vascular beds or the left ventricle due to a sudden increase in venous return resulted in reflex bradycardia, respiratory arrest, hypotension and asystole as seen in our patient. Our postulation may be supported by the fact that the patient responded rapidly to resuscitation with atropine and epinephrine.

Aortocaval fistula formation was reported to occur in 0.2% to 1.3% of patients with atherosclerotic aortic aneurysms, and in 3% to 4% of patients with ruptured aneurysms[3]. Compression or surgical closure of an arteriovenous fistula is associated with an increase in systemic arterial pressure and results in reflex bradycardia (Nicoladoni sign) triggered by stimulation of the arterial baroreceptors[6]. As bradycardia or asystole can occur during surgery in a patient with aortocaval fistula, the effect of changes in venous return on various baroreceptors should be considered in the hemodynamic management of a patient with aortocaval fistula.

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