Cardiothoracic Anesthesia, Respiration and Airway

Perioperative management of biventricular failure after closure of a long-standing massive arteriovenous fistula

[Le traitement périopératoire d'une défaillance biventriculaire suivant la fermeture d'une ancienne fistule artério-veineuse massive.]

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Purpose: To report the perioperative management of arteriovenous fistula (AVF) closure in a patient with high-output heart failure and pulmonary hypertension.

Clinical features: In a 71-yr-old man, closure of a long-standing massive AVF between the right femoral artery and vein was performed. After closure of the AVF, his pulmonary artery pressure (PAP) increased from 52/21 mmHg to 68/26 mmHg, his cardiac index decreased from $5.27 \text{ L}\cdot\text{min}^{-1}\cdot\text{m}^{-2}$ to $3.18 \text{ L}\cdot\text{min}^{-1}\cdot\text{m}^{-2}$, and his pulmonary wedge pressure increased from 15 mmHg to 32 mmHg due to an acute increase in afterload. Co-administration of prostaglandin E_1 and a phosphodiesterase III inhibitor improved the cardiac index and the PAP.

Conclusions: Surgical closure of the fistula may not always lead to resolution of the high output cardiac failure. In this case, afterload management using arterial dilators (prostaglandin E_1 , phosphodiesterase III inhibitor), use of inotropic drugs (phosphodiesterase III inhibitor), and close attention to volume status was crucial for a successful outcome after surgical AVF closure.

Objectif : Décrire le traitement périopératoire de la fermeture d'une fistule artério-veineuse (FAV) chez un patient atteint d'insuffisance cardiaque, avec débit cardiaque élevé, et d'hypertension artérielle pulmonaire.

Éléments cliniques : On a réalisé la fermeture d'une ancienne FAV massive entre l'artère et la veine fémorales droites chez un homme de

71 ans. Après la fermeture, la pression de l'artère pulmonaire (PAP) a augmenté de 52/21 mmHg à 68/26 mmHg, l'index cardiaque a diminué de $5,27 \text{ L}\cdot\text{min}^{-1}\cdot\text{m}^{-2}$ à $3,18 \text{ L}\cdot\text{min}^{-1}\cdot\text{m}^{-2}$ et la pression capillaire pulmonaire bloquée est passée de 15 mmHg à 32 mmHg à cause d'une hausse aiguë de la postcharge. La co-administration de prostaglandine E_1 et d'un inhibiteur de phosphodiesterase III a amélioré l'index cardiaque et la PAP.

Conclusion : La fermeture chirurgicale d'une fistule ne permet pas toujours la résolution d'une défaillance cardiaque avec débit cardiaque élevé. Dans le cas cité, la gestion de la postcharge à l'aide de dilateurs artériels (prostaglandine E_1 , inhibiteur de phosphodiesterase III), l'utilisation de médicaments inotropiques (inhibiteur de phosphodiesterase III) et une surveillance soutenue de l'état volumique ont contribué de façon capitale à l'évolution positive de la fermeture chirurgicale d'une FAV.

LONG-STANDING massive arteriovenous fistulas (AVF) caused by trauma are infrequent, and surgical repair is even more infrequent.¹⁻⁵ The perioperative management of patients undergoing closure of large AVF is difficult, especially in those complicated by high output cardiac failure and/or pulmonary hypertension.⁶ In such patients, preoperative cardiac failure may not

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TABLE Hemodynamics

	<i>Preinduction</i>	<i>Preclamp</i>	<i>Clamp</i>	<i>with PGE₁</i>	<i>Declamp</i>	<i>In the ward</i>	<i>PGE₁ & PDEIIIi</i>	<i>POD1</i>
HR	74	55	52	53	46		82	72
CO		7.65	4.62	5.75	5.09		7.62	5.88
CI		5.27	3.18	3.96	3.51		5.12	4.05
SAP	128/69(101)	91/43(61)	167/67(99)	141/59(86)	108/48(68)	160/60(99)	112/56(75)	119/60(80)
PAP		52/21(33)	68/26(41)	49/13(29)	28/14(16)		25/12(16)	30/16(20)
PCWP		15	32	20	15	25	10	8
CVP		15	25	10	4		7	4
SVRI		697	1819	1535	1458		1035	1510
PVRI		266	221	181	22		91	243

Hemodynamic changes during operation and postoperation. HR=heart rate (beats·min⁻¹); CO=cardiac output (L·min⁻¹); CI=cardiac index=(L·min⁻¹·m⁻²); SAP=systemic arterial pressure (mmHg); PAP=pulmonary arterial pressure (mmHg); PCWP=pulmonary capillary wedge pressure (mmHg); CVP=central venous pressure (mmHg); SVRI=systemic vascular resistance index (dynes·sec·cm⁻⁵·m⁻²); PVRI=pulmonary vascular resistance index (dynes·sec·cm⁻⁵·m⁻²); PGE₁=prostaglandin E₁; PDE IIIi=phosphodiesterase III inhibitor; POD 1=postoperative day1.

resolve immediately after closure of the AVF because the intervention induces an acute increase in afterload secondary to the sudden reduction of shunt flow. In this report, we describe the perioperative management of a patient with a large traumatic femoral artery to vein fistula. Administration of prostaglandin E₁ (PGE₁) and a phosphodiesterase III (PDE III) inhibitor significantly improved cardiac failure after closure of the AVF.

Case report

A 71-yr-old man, 53 kg in weight and 153 cm tall, had sustained a traumatic injury to his right groin 52 yr previously. He was first treated by compression of the bleeding site and later received blood transfusions. Otherwise, his history was unremarkable and he took no medication. Three months prior to his most recent hospitalization, he presented with a gradual onset of cold sensation and pain in the right leg. He was admitted to our hospital with mild exertional dyspnea, palpitations, and moderate swelling of the right lower limb. His blood pressure was 130/70 mmHg, heart rate 74 beats·min⁻¹, and respiratory rate 18 breaths·min⁻¹. A loud continuous murmur was audible upon auscultation and a strong thrill was palpated in the right groin. Both pedal pulses were palpable, but the pulse was weak on the right side. Auscultation of the heart revealed a prominent systolic murmur over the apex and over the second right intercostal space.

Angiography demonstrated a wide fistula between the right femoral artery and the circumflex vein, and there was no evidence of thrombosis in either vessel. The segment between the right external iliac artery and the femoral artery was tortuous. Both the femoral artery and vein were dilated. Chest radiography revealed cardiomegaly (cardiothoracic ratio: 64%),

mild congestion of the lung fields, and calcification of the aorta. Left ventricular end-diastolic and end-systolic dimensions were 69 mm and 44 mm (normal range; 37–55 and 26–36 mm) respectively, and ejection fraction was 65% by echocardiography. The electrocardiogram (ECG) met voltage criteria for left ventricular hypertrophy.

Midazolam (2 mg) was injected intramuscularly 30 min before induction of anesthesia. Radial and pulmonary artery catheters were inserted under local anesthesia. An epidural catheter was inserted via the second lumbar interspace. After a successful test dose (1% mepivacaine 2 ml), anesthesia was established with 1.5% mepivacaine (10 ml every 90 min). Thirty minutes after the initial dose of 1.5% mepivacaine, anesthesia to cold sensation was obtained between Th6–S2 dermatome. Before surgery, the cardiac index (CI) was increased and pulmonary hypertension was also present. The systemic vascular resistance index (SVRI) was very low preoperatively. All perioperative hemodynamic data are shown in the Table. After clamping the right external iliac artery, systemic arterial pressure (SAP), pulmonary artery pressure (PAP), and SVRI increased immediately. The CI decreased to 3.18 L·min⁻¹·m⁻², and the pulmonary wedge pressure (PCWP) increased to 32 mmHg. The height of the v-wave on the PCWP tracing wave did not change and ECG showed no remarkable change. These data suggested acute left ventricular dysfunction and, therefore, a continuous infusion of PGE₁ (0.05 µg·kg⁻¹·min⁻¹) was initiated in order to decrease SVR and PVR. The SAP, PAP, and SVRI decreased and the CI increased 15 min after the administration of PGE₁. After closure of the AVF and release of the iliac artery clamp, the PAP decreased to 28/14 mmHg, while the SVRI remained within normal limits. PGE₁ administration (0.025 to 0.05

$\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) was continued until the end of the operation. The patient received 700 ml crystalloid during the operation. Total blood loss was 400 g, and urine output was 800 ml, for an estimated intraoperative fluid balance of -500 ml.

After the patient's return to the ward, administration of PGE_1 was discontinued because the patient had no clinical symptoms of heart failure. However, the patient complained of dyspnea 30 min after the PGE_1 infusion was discontinued and 80 min after the final injection of 1.5% mepivacaine was given through the epidural catheter. The patient's PCWP increased to 25 mmHg and his chest radiogram revealed pulmonary edema. The PGE_1 ($0.05 \mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) infusion was reinstated and an infusion of the PDE III inhibitor amrinone ($0.5 \mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) was begun. Furosemide (10 mg) was also administered intravenously. The patient's congestive heart failure improved dramatically 30 min after co-administration of the drugs, (PCWP=10 mmHg, CI= $5.12 \text{ L}\cdot\text{min}^{-1}\cdot\text{m}^{-2}$). On postoperative day one, PGE_1 and the PDE III inhibitor were tapered while digitalization (digoxin, 0.125 mg/day) and anticoagulant therapy were started.

Discussion

This report documents a marked decline in cardiac function after closure of an AVF in a patient with a high output state from long-standing and massive shunting through the fistula. The patient presented with mild exertional dyspnea and palpitations. Chest radiography revealed cardiomegaly with mild pulmonary vascular congestion. These clinical data suggest that the patient had high output heart failure and volume overload preoperatively. Contrary to a previous report showing rapid reversibility of heart failure with closure of an AVF,⁷ our patient exhibited significant cardiac depression and pulmonary edema after the closure of his fistula.

High output cardiac failure due to arteriovenous shunting is a complication seen in elderly patients with large, chronic fistulas, and is frequently accompanied by other cardiac abnormalities.⁸ In such patients, cardiac failure may not resolve after closure of the fistula.⁹ In our patient, the SVRI increased (2.6 fold) and the CI decreased (0.6 fold) following clamping of the iliac artery, and the PCWP increased from 15 to 32 mmHg. The patient had no chest pain and ECG showed no ST-T change. In addition, an increase in the height of the v-wave was not observed. These data ruled out ischemic left ventricular failure and mitral regurgitation, and suggested that the sudden increase in afterload resulted in acute left ventricular dysfunction. Subsequent administration of PGE_1 decreased

the afterload of both the left and right ventricle¹⁰ and improved cardiac function.

Volume overload is frequently seen in patients with high output cardiac failure.¹¹ Although the patient's intraoperative fluid balance was negative, he developed pulmonary edema postoperatively. Sudden termination of the PGE_1 infusion increased the afterload of both ventricles and this may have led to the second episode of cardiac failure. In addition, return of a normal peripheral vascular resistance, as the effect of the epidural anesthetic receded may have also facilitated congestive heart failure and pulmonary edema. Use of PGE_1 , a PDE III inhibitor, and a diuretic were very effective. The PDE III inhibitor amrinone produces pulmonary and systemic vasodilation due to an increase in cyclic nucleosides in vascular smooth muscle, or a decrease in calcium entry and positive inotropic effects.¹²

Ingram *et al.*⁶ reported similar findings in a patient with a hemodialysis fistula. The patient suffered progressive depression of left ventricular function. He was extremely sensitive to such afterload reducing agents such as nifedipine and captopril. Patients with high output cardiac failure and a chronically low SVRI may be very sensitive both to increases in afterload and to vasodilators.

Epidural blockade is frequently used for vascular surgery. In general, patients undergoing vascular surgery potentially benefit from epidural anesthesia, because the associated sympathetic blockade enhances blood flow to the lower limbs. In patients with AVF, since SVR is low preoperatively, sympathetic blockade further decreases SVR and may induce hemodynamic instability. On the other hand, volume overload is frequently seen in patients with high output cardiac failure, and epidural blockade may be safely induced in these patients. Since AVF may be accompanied by epidural venous engorgement, it is essential for the anesthesiologist to minimize the possibility of accidental intravascular puncture. In addition, the majority of these patients are receiving anticoagulation therapy preoperatively to prevent thrombosis. During surgery heparin is also usually given prior to arterial cross-clamping. Furthermore, in many cases, anticoagulation therapy is continued in the immediate postoperative period. Decisions to use epidural anesthesia with anticoagulation therapy should be made on a case-by-case basis after careful evaluation of the possible risks and benefits.

Surgical closure of an AVF with massive shunting may not always lead to immediate resolution of the high output cardiac failure and may result in a marked decline of cardiac function postoperatively. Afterload management using arterial dilators, "inodilators", and

close attention to volume status may be required to achieve a successful outcome after surgical closure of such AVF.

References

- 1 *Graham ANJ, Barros D'Sa AAB.* Missed arteriovenous fistulae and false aneurysms in penetrating lower limb trauma: relearning old lessons. *Injury* 1991; 22: 179–82.
- 2 *Dedichen H, Thaulow E, Naess A* Traumatic arteriovenous fistula. Case report. *Acta Chir Scand* 1989; 155: 297–300.
- 3 *Özcan F, Baki C, Piskin B, Katptanoglu M, Yavas D* Aneurysmic dilatation of popliteal and femoral artery due to long-standing traumatic arteriovenous fistula. *Vasa* 1990; 19: 79–81.
- 4 *Coy JD, Coy VL.* Delayed diagnosis of post-traumatic arteriovenous fistula from trip wire-detonated booby trap in Vietnam. *Mil Med* 1991; 156: 636–7.
- 5 *Schneider M, Creutzig A, Alexander K.* Untreated arteriovenous fistula after world war II trauma. *Vasa* 1996; 25: 174–9 .
- 6 *Ingram CW, Satler LF, Rackley CE.* Progressive heart failure secondary to a high output state. *Chest* 1987; 92: 1117–8.
- 7 *Rich NM, Hobson RW, Collins GJ Jr.* Traumatic arteriovenous fistula and false aneurysms: a review of 585 lesions. *Surgery* 1975; 78: 817–28.
- 8 *Smith VM, Hughes CW, Sapp O, Joy RJJ, Mattingly TW.* High-output circulatory failure due to arteriovenous fistula. *Arch Int Med* 1957; 100: 833–41.
- 9 *Pate JW, Shermann RT, Jackson T, Wilson H* Cardiac failure following traumatic arteriovenous fistula: a report of fourteen cases. *J Trauma* 1965; 5: 398–403.
- 10 *Lock JE, Olley PM, Cocceani F.* Direct pulmonary vascular responses to prostaglandins in the conscious newborn lamb. *Am J Physiol* 1980; 238: H631–8.
- 11 *Schrrier RW.* Pathogenesis of sodium and water retention in high-output and low-output cardiac failure, nephritic syndrome, cirrhosis, and pregnancy. (First of two part). *N Engl. J Med* 1988; 319: 1065–72.
- 12 *Lery JH, Bailey JM.* Perioperative experience with amrinone. *Eur J Anaesthesiol* 1992; (Suppl. 5): 15–9.