Anesthetic Management for a Patient with Renal Arterio-venous Fistula

Yuko URATSUJI, Natsu IKEGAKI, Hironori KITADA and Seizo IWAI*

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Arteriovenous fistula of the renal vessel is an uncommon complication of nephrectomy. Since the first case was reported by Hollingsworth in 1934¹, 62 cases have been reported in the world². These fistulas are variable in size and their clinical manifestations. When a lesion is large and chronic, the cardiovascular system may compensate with significant hemodynamic changes, but gradually the large sized fistulas were reported to cause high output failure with or without cardiac decompensation. Accepted treatment is surgical closure of the arteriovenous communication. During the surgical treatment of this fistula, sudden arterial occlusion might result in life-threatening fluxes of vascular volumes and pressures. However there is no available report describing the anesthetic management for the operation of renal arterio-venous fistulas. One case was reported which was maintained under GOF anesthesia (Ogasawara et al.³), though precise description had not been found. In this report, we have traced several hemodynamic parameters such as arterial blood pressure, heart rate, cardiac output, systemic vascular resistance, pulmonary vascular resistance, prior to, during, and after operation. These data are presented and safe and sta-

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ble anesthetic management for this surgical treatment is discussed.

Case Report

A fifty-year-old female was admitted to our hospital for the evaluation of her elevated blood pressure and abdominal murmur. Prior to this hospitalization, her right kidney had been removed because of pyeronephritis and renal stone at the age of 27-year-old. Shortly after the urologic procedure, hypertension was found and had been treated medically using the calciumblocking agent (nifedipine). Twelve years



Fig. 1. Large arteriovenous fistula between right renal artery and renal vein with almost immediate opacification of inferior vena cava.

Department of Anesthesiology, Kobe Rosai Hospital, Kobe, Japan

^{*}Department of Anesthesiology, Kobe University, School of Medicine, Kobe, Japan

Address reprint requests to Dr. Uratsuji: Department of Anesthesiology, Kobe Rosai Hospital, Kagoike-dori 4-1-23, Chuo-ku, Kobe, 651 Japan

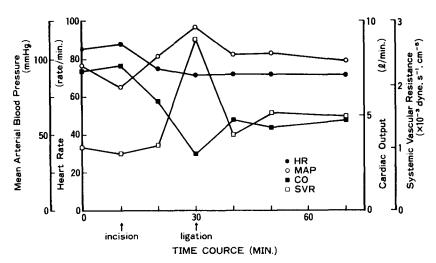


Fig. 2. Perioperative hemodynamic summary. Connecting lines are for visual ease. Solid circles represent heart rate (HR); open circles, mean arterial pressure (MAP); solid squares, cardiac output (CO); and open squares, systemic vascular resistance (SVR).

after nephrectomy, she was treated with digoxin due to hypertensive cardiac failure. At the age of 42-year-old, she was treated her hypertensive renal failure. Medications on admission included nifedipine 20 mg per day, which kept her blood pressure around $140 \sim 160/100 \sim 110$ mmHg.

Physical examination revealed a thrill and a continuous bruit in the right flank and lumbar fossa with a regular pulse rate of 84/min and a blood pressure of 164/110 mmHg on both arms. Routine chest xray examination showed a moderate cardiac enlargement (CTR = 62%) and slight pulmonary congestion. Electrocardiogram revealed left ventricular hypertrophy, but masters double test showed no ischemic change. An echocardiogram revealed right renal arteriovenous fistula fed by right renal artery. Angiography confirmed the above diagnosis (fig. 1), also blood gas data pre and postfistula showing sudden increase of O₂ saturation from 75.1% to 92.5%. Hemodynamic parameters by Swan-Ganz catheter were: Cardiac output, 7.38 $\ell \cdot \min^{-1}$, cardiac index, 5.77; right atrial pressure, 3 mmHg; pulmonary artery pressure of 21/8 mmHg with mean capillary wedge pressure, 6 mmHg. Laboratory investigation demonstrated normal electrolytes and serum creatinine of 0.5 mg, urine catecholamine and serum aldosterone levels, $81.3 \ \mu g \cdot day^{-1}$ and $33 \ pg \cdot ml^{-1}$, respectively. Serum renin activity was 0.8 ng $\cdot ml^{-1} \cdot hr^{-1}$. Urinalysis was negative. Further preoperative work up showed normal hemogram, serum chemistries, and coagulation studies, as well as a room air blood gas of pH 7.362, Pa_{CO_2} 41.7 mmHg, Pa_{O_2} 83.7 mmHg, and pulmonary functions tests of FEV₁ = 1.18 ℓ (1.50 predicted), FVC = 1.53 ℓ (2.36 predicted).

She was scheduled for surgical repair of this fistula and received diazepam, 10 mg p.o. and atropin, 0.5 mg i.m., for premedication. Epidural catheter was inserted at the level of L 1-2 interspace, and the patient received a total of 25 ml of 1% mepivacaine. General anesthesia was induced with thiopental and succinvlcholine and the trachea was intubated. Anesthesia was maintained with oxygen, nitrous oxide, halothane and pancuronium. After laparotomy inferior vena cava was exposed and the thrill was felt at the right renal vein with almost immediate opacification of the inferior vena cava with the hand of surgeon. The fistula was formed between right renal vein and renal artery, which appeared considerably

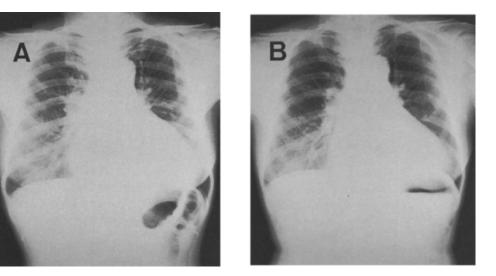


Fig. 3. Chest x-ray films obtained before 21 days operation (A) and after 7 days operation (B) showing cardiac enlargement and slightly pulmonary congestions (A) and slight improvement of cardiomegaly (B).

dilated and hypertrophic. The thrill disappeared with the compression of this fistula. After ligation of the right renal vein with almost immediate opacification of inferior vena cava, the thrill was not felt anymore.

A summary of the perioperative hemodynamic course is presented in figure 2, beginning with preanesthetic values at time "0". In attempt to effect preload and afterload reduction, especially after ligation of the fistula, nitroglycerin, phentolamine and epidural block were prepared. At the beginning of the operation, 20 ml of 1% mepivacaine was administered through epidural tubing. Before administration of 20 ml of 1% mepivacaine, mean arterial pressure, mean pulmonary artery pressure, systemic vascular resistance were 107 mmHg, 12.3 mmHg, 1440 dyne·cm⁻¹·s⁻⁵, respectively and this epidural block reduced these pressures to 81 mmHg, 11 mmHg, 872 dyne \cdot cm⁻¹ \cdot s⁻⁵, respectively. Throughout the entire procedure, TNG infusion of approximately 0.1-0.3 $\mu g \cdot k g^{-1} \cdot min^{-1}$ was continued. Before ligation of the right renal vein, TNG infusion was increased to 0.5 $\mu g \cdot kg^{-1} \cdot min^{-1}$ and phentolamine was prepared for the abrupt increase of afterload due to ligation of right

renal vein. Just after ligation of the right renal vein, systemic vascular resistance and mean arterial pressure both increased 2900 dyne·cm⁻¹·s⁻⁵, 120 mmHg, respectively. So iv bolus of phentolamine, 2 mg, and 5 ml of 1% mepivacaine to epidural tubing were given quickly to reduce abrupt increase of afterload with a cardiac output of 3.0 $\ell \cdot \min^{-1}$. Once these increases returned to normal range with a systemic vascular resistance of 1288 dyne $cm^{-1} s^{-5}$, a cardiac output of 4.8 $\ell \cdot \min^{-1}$ and a mean arterial pressure of 103 mmHg within 10 min, TNG infusion was waned. At this time no abnormal findings was found in EKG monitoring. Prior to and after the ligation of right renal vein, blood specimens were obtained through Swan-Ganz catheter at the site of right ventricle orifice, and these two oxygen saturation data were compared and confirmed of successful ligation of the shunt. Oxygen saturation data after ligation of right renal vein decreased to 82.4% compared to that of 92.7% before ligation. Her urine output was greater than 1 $ml \cdot kg^{-1} \cdot h^{-1}$ throughout the entire procedure. Since the beginning of the operation, Swan-Ganz catheter data showed no pulmonary hypertension although pulmonary

vascular resistance increased transiently up to 107 dyne·cm⁻¹·s⁻⁵ after ligation of right renal vein. Within one hour operation the surgical procedure and anesthesia were uncomplicated and 2 mg of morphine was administered through the epidural catheter for postoperative pain. She returned to the surgical unit. Her postoperative course was uneventful with stable blood pressure and heart rate. No pain complaint was observed.

The cardiomegaly found before operation still remained, but some improvement was observed. Chest x-ray films obtained before 1 month and after 7 days operation were compared in figure 3. After 14 days operation, cardiac catheterization was done, obtained cardiac output was $3.75\ell \cdot min^{-1}$; CI, 2.93. Patient had no complications and discharged to the hospital 21 days after the operation.

Discussion

Postnephrectomy renal arteriovenous fistulas are rare, considering the number of nephrectomies performed. Symptoms include a bruit over the nephrectomy incision in 80% of the cases, cardiomegaly in 80%, high output heart failure in 50% and pain in 40% (Maldonado et al.⁴). These symptoms develop over a period of time ranging from days to years. Increased cardiac output is a compensatory mechanism of the steal of arterial blood toward the venous return to the right heart. The increased volume load affects both ventricles and leads to cardiomegaly both through hypertrophy of the wall and dilatation of the cavities. Gradually this situation induces cardiac failure, though this tolerance of the heart to the fistula is extremely long. The steal of arterial blood into a low-pressure accounts for the blood pressure abnormalities, such as low diastolic pressure and wide pulse pressure. But arterial hypertension and the presence of the fistula are not always clear⁵. The definitive diagnosis is made by renal angiography. Fistulas with hemodynamic disturbances sufficient to cause symptoms have to be surgically repaired.

Generally surgical treatment of renal arteriovenous fistulas is indicated for congestive heart failure, hypertension, hematuria, and pain. The surgical treatment gave satisfying results in most cases^{6,7}.

The risk factors for the management of anesthesia are 1) management of cardiac failure, which is caused by volume overload to both ventricles, this condition is variable from just hypertention to severe cardiac failure with cardiomegaly, pulmonary congestion which needs treatment with inotrope and diuretics. 2) management of sudden increase of afterload after ligation of the fistula. This is critical in the case of hemodynamically significant shunt because it causes profound circulatory decompensations. Sullivan and Goldenberg⁸ emphasized the marked effect of a large fistula on peripheral vascular resistance and relative fluid volume. They reported such a case of large renal arteriovenous fistula with congestive heart failure. In their report, they recommended combined, controlled approach, which consists of intra-arterial balloon to stabilize the hemodynamic change and surgical treatment after arterial occlusion by inflating balloon. According to these reasons, sometimes surgical correction becomes contraindicated to the patients in poor general condition in spite of the fistulas with hemodynamic disturbances sufficient to cause symptoms have to be surgically corrected. For such a case nonsurgical closure of fistula was also reported using the embolization technique (Castaneda et al.⁹).

In our case, preoperative cardiac failure was not so severe, no symptoms of pulmonary congestion was shown, although hyperdynamic state of the heart was observed. In addition to circulatory problems, her right kidney was removed before, the risk factors were 1) management of cardiac failure and hemodynamic change. 2) management of urine output which reflects her residual renal function. During the operation the use of TNG, phentolamine and epidural block for the reduction of both preload and afterload was felt to work very well except one point increase of afterload. About this increase, we should have estimated this increase more properly and should have administered phentolamine iv, TNG and epidural mepivacaine

more quickly than we did to prevent sudden increase of afterload. We could not expect this increase so properly which induced sudden decrease of cardiac output. Applying Swan-Ganz catheter made 1) careful observation of the hemodynamic movement, 2) maintenance of stable hemodynamics, 3) determination of proper infusion volume for urine output and 4) estimation of successful operation technique possible, 4) means that taking samples at the site of RV orifice and comparing their oxygen saturation data before and after ligation of right renal vein, the decreased oxygen saturation data confirmed that the shunt was closed successfully. These technique is not special but well used for left to right shunt disease, but this case has volume overload to both ventricles and needs careful, safe management during operation. As renal arteriovenous fistula itself is very rare, no precise reports to keep this operation successful had not been found. For surgical treatment of such case our anesthesia method will work very well with improved technique of using vasodilator to keep stable hemodynamics, and also epidural catheter can be used for post operative pain.

In summary this report describes the hemodynamic changes during perioperative period of the anesthesia for the renal arteriovenous fistula, which was combined with hyperdynamic hemodynamics. Potential advantages of monitoring data from Swan-Ganz catheter and using vasodilating techniques during operation include keeping hemodynamic change stable. For the safer and more stable anesthetic management, more experience with using vasodilator technique and better understanding the circulation status during operation should be needed.

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