J Anesth (2011) 25:263–266
DOI 10.1007/s00540-011-1104-9

CLINICAL REPORT

Anesthetic management of aortocaval fistula repair associated with aortic valve replacement, severe aortic regurgitation, and bacterial endocarditis

Jahan Porhomayon · Mahmoud A. A. Hassan · Padmavathi R. Perala · Nader D. Nader

Received: 22 September 2010 / Accepted: 31 January 2011 / Published online: 16 March 2011

Abstract We report a case of an adult male who had received a gunshot to the abdomen 12 years earlier. He presented with manifestations of high-output congestive heart failure (CHF), aortic regurgitation (AR), and pulmonary septic embolism. Further investigation revealed an aortocaval fistula (ACF). Following endovascular repair of the ACF, we observed an immediate rise in systemic vascular resistance (SVR), decrease in central venous pressure (CVP), increase in regurgitant flow across the aortic valve, and decrease in central mixed venous oxygenation. A combination of vasodilators and vasopressors was used to maintain hemodynamics. Milrinone infusion was necessary after cardiopulmonary bypass to maintain cardiac output. Even though local anesthesia and light sedation were used for ACF closure, the hemodynamics changed dramatically throughout the procedure. ACF closure under local anesthesia and sedation is preferred because the hemodynamics alterations under local anesthesia are less severe. The rise in SVR and regurgitant flow across aortic valve is less dramatic. As a result, hemodynamic management and separation from cardiopulmonary bypass are easier.

Keywords Aortocaval fistula · Hemodynamic · Endocarditis · Aortic valve

J. Porhomayon · N. D. Nader
VA Western New York Healthcare System, Division of Critical Care and Pain Medicine, Department of Anesthesiology, State University of New York at Buffalo School of Medicine and Biomedical Sciences, Buffalo, NY, USA

M. A. A. Hassan · P. R. Perala
VA Western New York Healthcare System, Division of Cardiothoracic Anesthesia, Department of Anesthesiology, State University of New York at Buffalo School of Medicine and Biomedical Sciences, Buffalo, NY, USA

J. Porhomayon (✉)
VA Medical Center, Rm 203C, 3495 Bailey Ave, Buffalo, NY 14215, USA
e-mail: jahanpor@buffalo.edu

Introduction

Chronic aortocaval fistula (ACF) is a rare complication of abdominal penetrating injuries. Presentation is variable in the presence of coexisting heart disease. We report the anesthetic management of a patient with ACF associated with aortic valve endocarditis and severe aortic regurgitation (AR). Management of these coexisting conditions is challenging in that correction of one disease may potentially worsen the other. The patient underwent repair of the ACF under local anesthesia and aortic valve replacement under general anesthesia. He experienced an immediate rise in systemic vascular resistance (SVR) and decreased central venous pressure (CVP) after ACF closure despite low-dose analgesia and sedation. He was managed with invasive hemodynamic monitoring and a combination of vasodilators and vasopressors.

Case report

A 50-year-old man was referred to the anesthesia preoperative clinic for aortic valve replacement (AVR) with severe aortic regurgitation (AR). He presented with worsening shortness of breath for a few days, 10-kg weight loss, lower extremity edema, and backache for a few weeks. His past medical history was significant for exploratory laparotomy

following a gunshot wound to the abdomen 12 years earlier. Physical examination revealed low weight (67 kg), jugular venous distension, harsh vesicular breathing, audible abdominal bruit, and diastolic murmur over the aortic area. Laboratory results showed high B-type natriuretic peptide (BNP), 400 pg/ml (normal reference range for BNP, <100 pg/ml); hemoglobin, 11.8 g/dl; blood urea nitrogen, 27 mg/dl; creatinine, 1.3 mg/dl; potassium, 4.4 mEq/l. Electrocardiogram showed normal sinus rhythm; transthoracic echocardiography showed severe AR, aortic valve vegetation approximately 10 mm in size, pulmonary hypertension, and moderate tricuspid and mitral valve regurgitation with ejection fraction of 65%. Chest X-ray revealed cardiomegaly and multiple pulmonary nodules (Fig. 1). Blood cultures were positive for *Streptococcus* species. Cardiac catheterization confirmed the presence of severe AR, together with high cardiac output state, hyperdynamic left ventricular function, and disease-free coronary arteries. Abdominal magnetic resonance imaging (MRI) obtained about 1 week later showed an infrarenal aortocaval fistula below the inferior mesenteric artery and was suggestive for lumbar vertebral osteomyelitis (Fig. 2). The patient remained in a compensated physiological state and received a 1-week course of antibiotic therapy. We decided to treat endocarditis and osteomyelitis with a 6-week antibiotic course. Surveillance blood cultures would then be obtained to assure no growth had occurred in the blood. He would then undergo endovascular repair of the ACF under local anesthesia and sedation followed by AVR under general anesthesia (GA) in the same setting. On the day of surgery, patient vital signs were heart rate (HR) 109 beat/min, blood pressure (BP) 112/55 mmHg, and SaO_2 100%. Monitoring included pulse oximetry, electrocardiogram, Foley catheter, temperature, and a left radial arterial line for beat-to-beat monitoring of blood pressure. We secured peripheral venous access (18 G), and volume replacement was started with normal saline solution. Sedation was accomplished by intravenous fentanyl 2 μg/kg, ketamine 20 mg, and dexmedetomidine 0.7 μg/kg/min; oxygen was delivered via face mask. A pulmonary artery catheter (PAC) was inserted under real-time sonographic guidance via the right internal jugular vein. Hemodynamic variables recorded were pulmonary artery pressure (PAP), 54/25 mmHg; CVP, 29 mmHg; cardiac output (CO), 17.5 l/m; systemic vascular resistance (SVR), 219 dyne/s/cm; and mixed venous oxygen saturation (SvO_2), 91%. The high CVP tracing reflects the presence of a retrograde pulsatile waveform secondary to tricuspid regurgitation. Sodium nitroprusside infusion was started at 0.3 μg/kg/min and titrated before ACF closure, which was successful in preventing the anticipated rise in SVR. The ACF was repaired by placing an Endologix graft (3.5 cm) stent in the aorta, which was demonstrated by radiography to occlude flow into the vena cava. Vital data following ACF closure were as follows: HR, 71 bpm; CO, 5.1 l/m; SVR, 1,396 dyne/s/cm; SvO_2, 69%; PAP, 41/20 mmHg; BP, 142/89 mmHg; and CVP, 17 mmHg. At this point, we proceeded to GA by administration of IV etomidate 40 mg, rocuronium 50 mg, and fentanyl 50 μg. Sodium nitroprusside infusion was titrated to avoid excessive rise in SVR only in the pre- and post-induction periods. An endotracheal tube was then inserted and mechanical ventilation started. Anesthesia was maintained by fentanyl infusion 5 μ/kg/h together with sevoflurane 2%. Intraoperative transesophageal echocardiogram (TEE) showed dilated left ventricle (LV), elevated left ventricular diastolic pressures, and restrictive filling

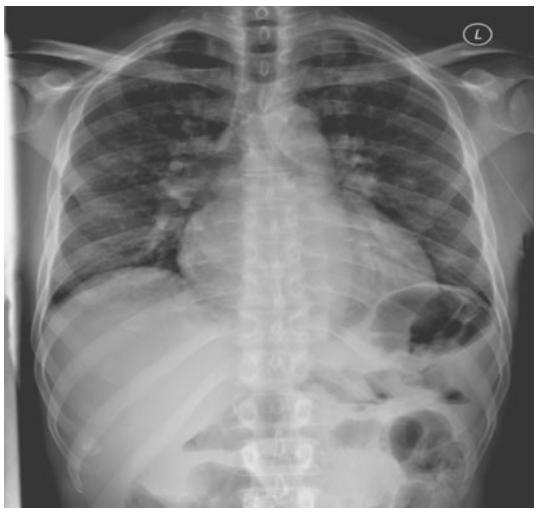

Fig. 1 Chest X-ray (CXR) shows pulmonary nodules and cardiomegaly

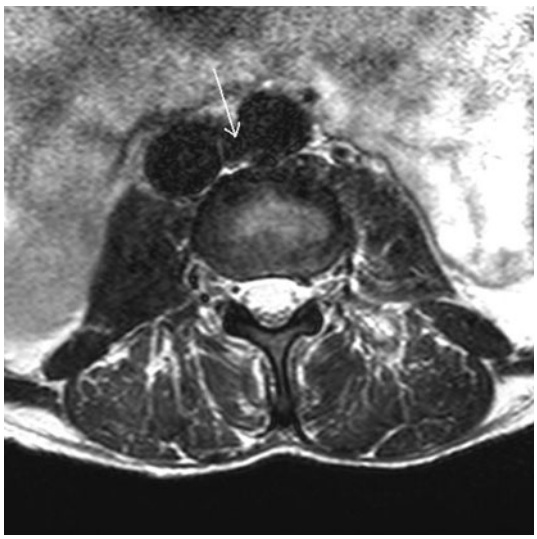

Fig. 2 Abdominal magnetic resonance imaging (MRI) at L3 shows the aortocaval fistula (ACF) as a communication (*arrow*) between the aorta (Ao) on the left and inferior vena cava (IVC) on the right

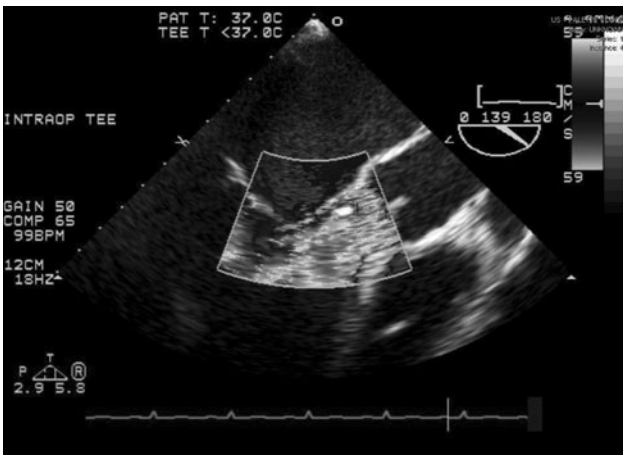

Fig. 3 Color flow Doppler demonstrates severe aortic regurgitation

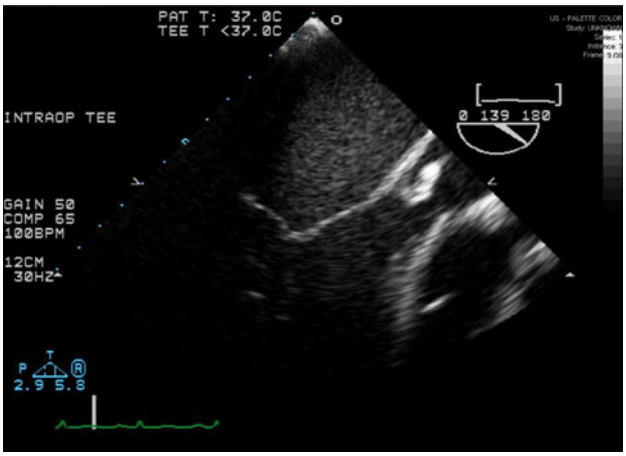

Fig. 4 Transesophageal echocardiograph shows septic vegetations over the aortic valve cusp

pattern attributed to severe AR (Fig. 3). It also showed organized vegetations on the left coronary cusp (Fig. 4). Vital signs after AVR repair were HR, 77 bpm; CO, 5.3 l/min; SVR, 1,262 dyne/s/cm; SvO_2, 72%; PAP, 40/23 mmHg; BP, 132/81 mmHg; and CVP, 15 mmHg. Milrinone was started immediately after cardiopulmonary bypass (CPB) at 0.2 μg/kg/h and continued postoperatively in the intensive care unit (ICU). AVR was completed using a bi-leaflet mechanical valve. The patient was transferred to the ICU intubated, with stable hemodynamics and with milrinone infusion.

Discussion

We first addressed medical treatment of the aortic valve endocarditis. Lumbar osteomyelitis was diagnosed a week earlier and may have contributed to aortic valve seeding via a hematogenous route. The blood culture revealed *Streptococcus viridians*, which was treated for 6 weeks with ceftriaxone 2 g/day for adequate osteomyelitis therapy. Postponing AVR was based on recommendations from the American College of Cardiology and the American Heart Association, which consider the hemodynamic profile, severity of CHF, the organism responsible, the site of infection, and the presence of septic emboli and recurrent fever [1–3]. Risk of an embolic phenomenon varies from 10% to 50% in different series [4]. Three quarters of embolisms occur before the beginning of antibiotic treatment [5]. Embolic episodes are common when the vegetation is mobile and its size is more than 10 mm [4, 6]. We believed delaying surgery was acceptable because the risk of embolic events is lower after 7–10 days of antibiotic therapy [4].

For management of ACF and AVR repair, careful attention was given to hemodynamic assessment of these lesions. ACF is an uncommon late presentation of penetrating injuries to the aorta and vena cava [7]. It rarely closes spontaneously and usually increases gradually in size [8]. ACF manifests locally at the site of injury by a continuous bruit or thrill and systemically as volume overload [9]. Factors that influence the severity of the clinical manifestations include size of the fistula, proximity to the heart, and cardiac reserve of the patient [10]. Clinically, traumatic fistula tends to be asymptomatic or present insidiously. These fistulas are usually smaller and occur in younger individuals who have good cardiac reserve [11]. When large enough, ACF leads to decreased tissue perfusion, a markedly increased venous return, and increased cardiac output. Hemodynamic manifestations include significant reduction in systemic vascular resistance, tachycardia, remarkable increase in cardiac output, and a significant increase in cardiac filling pressures [10]. Concurrently, arterial flow and perfusion pressure distal to the ACF decrease, leading to compensatory distal vasoconstriction and reduced capillary perfusion [12]. Possible complications include high-output CHF, often with tricuspid regurgitation. Renal dysfunction is present in 53% of cases, attributable to chronic venous congestion and decreased perfusion pressure [13]. Acute decompensation is reported less often and usually involves rupture of an associated aneurysmal sac [10]. Effective treatment includes open or endovascular repair. In our patient with high-output heart failure, the heart was compensated by the well-perfused functionally contractile myocardium. ACF decreased his SVR, and the AR was less severe. Therefore, we decided to proceed with endovascular repair of the ACF under local anesthesia with sedation after completion of antibiotic therapy. Anesthetic goals include avoiding disturbances in preload, heart rate, afterload, and oxygenation. Inotropes and vasopressors were ready for use when needed, keeping in mind that

elevation of SVR would worsen the AR [14]. We decided to proceed with a small dose of ketamine for its potent analgesic properties and its ability to maintain spontaneous respiration. Dexmedetomidine infusion was selected for its sedative effect, because its impact on hemodynamics is minimal and respiration is also well maintained. Fentanyl at low doses was used to supplement analgesia for the procedure. Our goal was to avoid general anesthesia and possible hemodynamic decompensation, which has been reported in previous studies [8, 11]. We also monitored fluid and electrolyte balance, as diuresis is expected after closure consequent to mobilization of edema fluid [9]. The PAC provided PAP, PA occlusive pressure, SvO_2, SVR, and CO [12] before and after ACF closure. Following closure of ACF, SVR increased and CVP decreased, the latter being maintained with additional boluses of crystalloids solution. Excessive rise in SVR was avoided with infusion of nitroprusside. For AVR, our goal was to minimize regurgitation through maintaining SVR at low normal values and heart rate at upper normal values and avoiding myocardial depressant drugs. We induced general anesthesia with etomidate because it would preserve or increase sympathetic tone and prevent vasodilation, which could decrease CVP and venous return; however, there was a risk of worsening the AR because of increased SVR. We believed nitroprusside infusion had a shorter duration of action and was easier to titrate. Anesthesia was maintained with sevoflurane and fentanyl infusion, and post-AVR vital signs remained stable intraoperatively and postoperatively. For the post-CPB period we used milrinone to maintain cardiac output. Milrinone is an inodilator that will maintain low SVR while supporting myocardial contractibility. Post-AVR TEE showed good aortic valve seating without perivalvular leakage, reduced LV size, and improved biventricular function with ejection fraction of 50%.

In summary, anesthetic challenges for aortic valve endocarditis and ACF repair are twofold: timing for aortic valve surgery in the setting of acute bacterial endocarditis and the management of two hemodynamically consequential lesions. Closing the shunt before AVR proved to be the better approach in terms of hemodynamic stability.

Acknowledgments The authors do not have any financial and personal relationships with other persons or organizations, such as employment, consultancies, stock ownership, honoraria, paid expert testimony, patent applications/registrations, and grants or other funding, that could inappropriately influence (bias) their work.

References

1. Weinberg AN, Gerald WA, William BS, Austen WG. Cardiac surgery in bacterial endocarditis. Circulation. 1968;38:514–23.
2. Dreyfus G. Surgery in acute bacterial endocarditis: restraints and possibilities. Arch Mal Coeur Vaiss. 1993;86:1869–75.
3. Griffin FM, Jones G, Cobbs GC. Aortic insufficiency in bacterial endocarditis. Ann Intern Med. 1972;76:23–8.
4. Di Salvo G, Habib G, Pergola V, Avierinos JF, Philip E, Casalta JP, Vailloud JM, Derumeaux G, Gouvernet J, Ambrosi P, Lambert M, Ferraci A, Raoult D, Luccioni R. Echocardiography predicts embolic events in infective endocarditis. J Am Coll Cardiol. 2001;37:1069–76.
5. Heiro M, Nikoskelainen J, Engblom E, Kotilainen E, Marttila R, Kotilainen P. Neurologic manifestations of infective endocarditis: a 17-year experience in a teaching hospital in Finland. Arch Intern Med. 2000;160:2781–7.
6. Olaison L, Pettersson G. Current best practices and guidelines indications for surgical intervention in infective endocarditis. Infect Dis Clin N Am. 2002;16:453–75.
7. Sharma ML, George KA, Gamble JA. Anesthetic implications of endovascular repair of aorto-caval fistula. Anaesthesia. 2000; 55:697.
8. Marikina K, Setoguchi H, Yoshino J, Motoyama M, Makizono R, Yokoo T, Suemori Y, Tanaka H, Takahashi S. Anesthetic management of a patient with aorto-caval fistula. J Anesth. 2009;23:111–4.
9. Spencer TA, Smyth SH, Wittich G, Hunter GC. Delayed presentation of traumatic aorto-caval fistula: a report of two cases and a review of the associated compensatory hemodynamic and structural changes. J Vasc Surg. 2006;43:836–40.
10. Neema PK, Ramakrishnan S, Sinha PK, Rathod RC. Anesthetic implications of surgical repair of an aorto-caval fistula. J Cardiothorac Vasc Anesth. 2003;17:236–9.
11. Bodenham A. Anesthetic hazards of aorto-caval fistula. Br J Anaesth. 1990;65:723–5.
12. Salo JA, Verkkala K, Ketonen P. Diagnosis and treatment of spontaneous aorto-caval fistula. J Cardiovasc Surg. 1987; 28:180–3.
13. Brewster DC, Cambria RP, Moncure AC. Aorto-caval and iliac arteriovenous fistulas: recognition and treatment. J Vasc Surg. 1991;13:253–65.
14. Stone ME, Fischer GW. New approaches for the surgical treatment of end stage heart failure. In: Kaplan JA, editor. Kaplan's cardiac anesthesia. 5th edn. Philadelphia: Elsevier Saunders; 2006. p. 884–5.