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The Four Seasons of Ruptured Sinus of Valsalva Aneurysms: Case Presentations and Review

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ABSTRACT

The sinuses of Valsalva are 3 distinct outpouchings of the aortic wall associated with the 3 cusps of the aortic valve that may develop aneurysmal dilation because of weakness of or injury to the sinus wall. Rupture of a sinus aneurysm can create an aortocardiac fistula. Ruptured sinuses of Valsalva aneurysms (RSVAs) may present a diagnostic dilemma because of their varied clinical presentations. However, if included on a differential, they are easily diagnosed and surgically treated. In our article we detail 4 RSVA cases, each demonstrating a manner in which an RSVA may present clinically. Our first case involves a 68-year-old patient with an RSVA diagnosis after presenting with cardiac arrest and congestive heart failure. Our second case involves a 42-year-old patient with an RSVA diagnosis in the context of acute chest pain, ischemic electrocardiographic changes, and hypotension. Our third RSVA case involves a 60-year-old patient who presented solely with a sudden onset of lower-extremity edema. Our fourth case involves a 46-year-old asymptomatic patient with RSVA diagnosed during a routine physical exam. Comparisons of reported case series from around the world illustrate RSVA epidemiology, concomitant lesions, clinical presentations, and repair techniques. Comparisons of Eastern and Western series reveal that the incidence of RSVAs is higher in Eastern than in Western countries, with a 4:1 male preponderance across ethnic lines. Among the Eastern series reporting RSVAs, ventricular septal defects and aortic valve incompetence were the only frequently associated concomitant lesions. In contrast, Western series of RSVAs showed a wide range of concomitant lesions. The difficulty in diagnosing RSVAs is mainly due to the variability of their clinical impact and presentation. These factors largely depend on the cardiac chamber into which the aortocardiac fistula forms. However, once RSVA is on a differential, the advent of transesophageal and transthoracic radiography has made RSVA diagnosis relatively easy. Surgical repairs of RSVAs are of low risk and generally have an excellent long-term prognosis. As a result, many authors believe that early surgical intervention in patients with an RSVA is justified. Among the series studied, there is evidence that the patch technique is the safest approach because of its lower association with fistula recurrence. This article highlights for the clinician the diversity of clinical presentations of this often overlooked disorder.

INTRODUCTION

The sinuses of Valsalva are situated above and associated with each of the 3 cusps of the aortic valve. These sinuses are symmetrical and distinct outpouchings of the aortic wall and are most prominent just above the aortic valve annulus. As they rise above the level of the aortic valve leaflet commissures, the 3 sinuses taper and join the tubular ascending aorta at the sinotubular junction. Leonardo da Vinci first deciphered the role of these subtle outpouchings. Through detailed anatomical observations of human dissections, the 15th century artist and scientist deduced the role of the sinus of Valsalva in the closure of the aortic valve. He proposed that the shape of the sinus of Valsalva creates a vortex within the sinuses that separates the aortic wall from the edges of the aortic valve leaflets during systole [Gharib 2002]. The valve leaflets can then close during diastole without the impedance of surface tension. Leonardo da Vinci created a glass model to visualize flow through the aorta, and his observations supported this hypothesis [Gharib 2002]. Not until the advent of magnetic resonance imaging in the 20th century was in vivo evidence obtained that supported this earlier hypothesis. Magnetic resonance imaging has also defined the role of the sinus of Valsalva in aiding leaflet opening through the creation of a low-pressure system by means of the Venturi effect [Gharib 2002].

Sinuses of Valsalva can develop aneurysmal dilation, called a sinus of Valsalva aneurysm (SVA), when there are defects in the aortic media leading to a separation of the media from the aortic annulus fibrosus. The aneurysmal dilations can rupture, and the ruptured SVA (RSVA) forms a communication between the aortic sinus and a cardiac chamber, creating an aortocardiac fistula [Azakie 2000]. RSVAs are relatively rare cardiac anomalies and may present a diagnostic dilemma because of their varied clinical presentations. Below we detail 4 cases, each demonstrating an unusual way that an RSVA presented clinically. A clearer understanding of RSVA epidemiology, concomitant lesions, clinical presentations, and

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repair techniques may also be gained through the following comparisons of reported case series from around the world.

CASE PRESENTATIONS

Case 1: Sudden Death in a Patient with Coronary Artery Disease

A 68-year-old man from India had been in the United States for 2 months to visit his son. He had a medical history significant for diabetes mellitus (well controlled with oral hypoglycemic agents), chronic atrial fibrillation, and coronary artery disease and a history of a myocardial infarction more than 10 years prior. The patient had a sudden cardiac arrest while at home and was resuscitated by paramedics and taken to a local hospital emergency room, where he was found to be in congestive heart failure. A cardiac examination noted a loud holosystolic murmur. The electrocardiogram showed a wide-complex idioventricular tachycardia. The results of laboratory tests were remarkable for acidosis and severe hyperkalemia (serum potassium level of 7.4 mmol/L) and were negative for cardiac enzymes. After correction of the metabolic disturbances and arrhythmias, further evaluation revealed new diagnoses of hypertension, hypothyroidism, and diabetes. An echocardiogram demonstrated an SVA involving the sinus of the noncoronary cusp with fistulous communication to the right atrium, a normal-appearing and competent aortic valve, severe right atrial enlargement, right ventricular enlargement, and moderate tricuspid regurgitation. Cardiac catheterization confirmed the presence of a noncoronary SVA, with a 3:1 fistulous shunt to the low right atrium. Pulmonary artery and right atrial pressures were mildly elevated, and the pulmonary capillary wedge pressure was 19 mm Hg. There was no significant coronary disease. Because of the large shunt, surgical repair of the fistula was recommended. At the time of surgery, the right atrium and right ventricle were markedly distended. After circulatory support with cardiopulmonary bypass and arrest of the heart, the aorta was opened, and a 6-mm opening of the fistula tract was seen in the wall of the dilated noncoronary sinus. This rupture was repaired with a 10-mm prosthetic patch that was sewn over the opening from within the aorta by means of a running suture.

Case 2: Patient with Acute Chest Pain, Ischemic Electrocardiographic Changes, and Hypotension

A 42-year-old male smoker with no significant past medical history (except occasional alcohol use and a remote history of drug abuse) presented to the emergency room with the complaint of substernal chest pain, nausea, and vomiting for 8 to 10 hours. While being examined, the patient became progressively hypotensive (systolic blood pressure dropping from approximately 90 mm Hg to approximately 80 mm Hg) and continued to complain of chest pain. The patient was treated with intravenous fluids, sublingual nitroglycerin, intravenous heparin infusion, and intravenous dopamine infusion for pressure support. An electrocardiogram showed a sinus rhythm with an ST-segment depression of 1 to 2 mm in the lateral leads. The patient's serum creatine kinase level was 500 IU/L with an 11% MB fraction. Because of continuing chest pain and hypotension, the patient underwent urgent cardiac catheterization. The left and right coronary arteries were free of significant disease, but the examination revealed moderate aortic insufficiency, elevated right heart pressures (pulmonary artery pressure of 54/25 mm Hg, and a thermodilution cardiac index of 5.5 L/min per m^2), and a large SVA fistula from the aortic root to the right atrium. Because of his precarious hemodynamic status, the patient was taken directly to the operating room. An aneurysm of the noncoronary sinus of Valsalva was confirmed, with a fistulous windsock extending into the right atrium. The windsock was everted onto the aorta and resected, and the sinus wall defect was closed with a prosthetic patch.

Case 3: Patient with Onset of Lower-Extremity Edema

A 60-year-old developmentally delayed institutionalized man with a history of non–insulin-dependent diabetes, hypothyroidism, and hypertension was referred for significant bilateral lower-extremity edema. The results of venous Doppler studies of his legs were negative for deep venous thrombosis. The patient was treated with diuretics, which resolved his edema. The detection of a heart murmur in the physical examination prompted an echocardiographic examination, which demonstrated an SVA of the noncoronary sinus with a fistulous communication from the sinus into the right ventricle. Cardiac catheterization revealed diffuse, nonsignificant coronary artery disease and a noncoronary sinus aneurysm with a fistula stream transversing the right atrium and ending in the right ventricle. At the time of surgery, an SVA of the noncoronary sinus with a 10-mm circular wall defect was detected with a fistula into the right atrium. A thickened calcified fistula windsock extended across the tricuspid valve into the right ventricle. These defects were repaired by patch closure of the defect on the aortic sinus wall and by resection of the windsock and oversewing of the fistula opening in the upper atrial wall.

Case 4: Asymptomatic Patient with a 20-Year History of a Ventricular Septal Defect

A 46-year-old man with a 20-year-history of a heart murmur attributed to a ventricular septal defect (VSD) presented to a new physician for an initial routine examination. Although he was asymptomatic, the patient was referred to a cardiologist for evaluation and follow-up of his VSD. An echocardiogram demonstrated a normal-sized left ventricular cavity with an ejection fraction of 65%, a nonstenotic aortic valve with trivial insufficiency, and what appeared to be a membranous VSD high on the interventricular septum with a significant left-to-right shunt. Cardiac catheterization revealed a communication between the aorta and the right ventricle suggestive of a sinus of Valsalva fistula and no evidence of a VSD. A subsequent transesophageal echocardiogram showed an SVA of the noncoronary sinus with a communication from above the aortic valve to the right atrium, with the fistula jet directed into the right ventricle. Because of the large left-to-right intracardiac shunt, surgical repair of this defect was recommended. At the time of surgery, an opening of 8 to 10 mm was found in the base of the noncoronary sinus, with a fistula tract extending into the right atrium

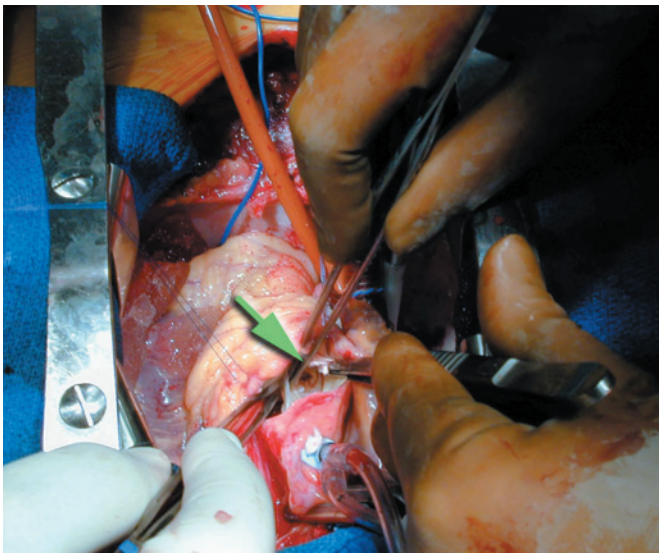

Figure 1. Fistula opening (dark circle at tip of green arrow) in noncoronary sinus of Valsalva seen through the transverse opening in the aortic root. The patient's head is at the bottom; forceps are retracting the aorta wall.

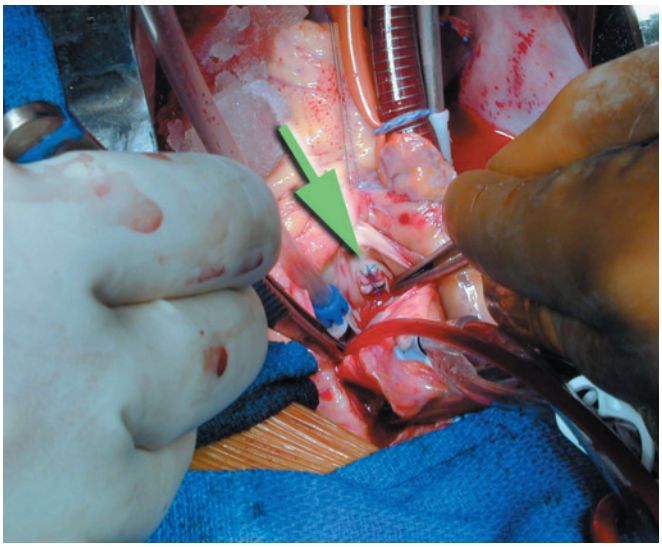

Figure 3. The fistula opening in the aortic root is closed with a polyethylene terephthalate (Dacron) prosthetic patch (green arrow). The head of the patient is at the bottom; forceps are retracting the aorta wall.

(Figure 1). A large windsock in the right atrium extended down through the tricuspid valve and directed the fistula flow into the right ventricle, simulating a VSD (Figure 2). The defect in the aortic sinus wall was closed with a prosthetic patch (Figure 3). The right atrial windsock was resected, and the fistula opening was sutured closed (Figure 4).

TYPES OF SVAS

The growing application of echocardiography for diagnostic testing has increased the rate of SVA diagnosis and has aided in the better understanding of the causes [Tak 2002]. SVAs can be acquired, congenital, or associated with cystic medial necrosis. Congenital SVAs are the most common type of SVAs, with an incidence rate ranging from 0.1% to 3.5% of all congenital heart defects [Goldberg 1990]. They are commonly attributed to muscular or elastic tissue deficiencies in the aortic wall behind the sinus of Valsalva [Lijoi 2002]. Acquired SVAs are usually secondary to conditions that weaken the aortic wall. These conditions include, but are not limited to, bacterial endocarditis, tuberculosis, syphilis, blunt or penetrating trauma [Goldberg 1990], atheromatous

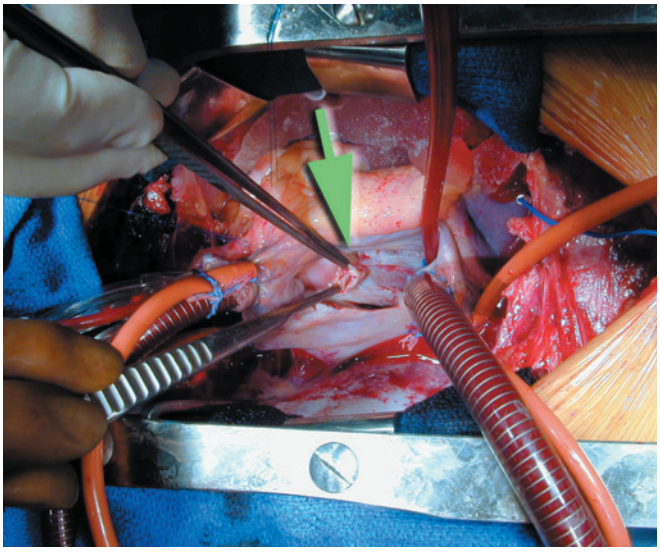

Figure 2. Seen through an opening in the lateral wall of the right atrium, the fistula windsock (in forceps tips, green arrow) is emanating from the lower right atrial septum. The tip of the windsock is extended through the tricuspid valve, seen to the right of the windsock. View is from the right side of the patient; the patient's head is to the left.

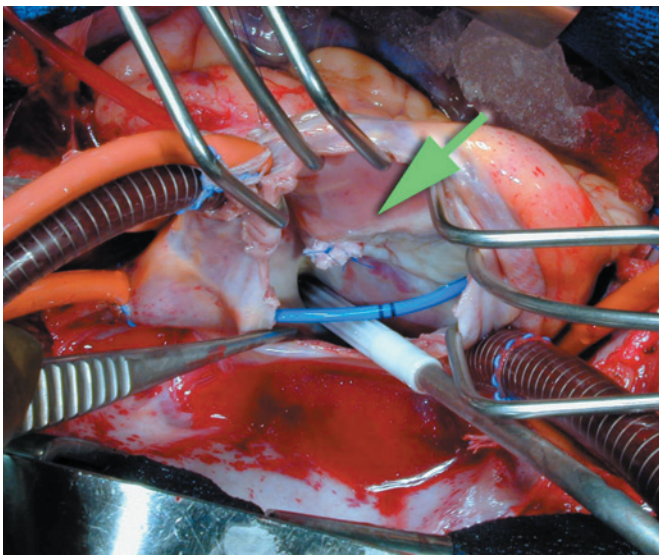

Figure 4. The fistula windsock has been resected and oversewn (green arrow). The tricuspid valve is seen to the right of the resected windsock. View is from the right of the patient; the head of the patient is to the left. Retractors are exposing the right atrium. The blue pulmonary artery–monitoring catheter is seen transversing the right atrium.

Concomitant Congenital Heart Lesions Associated with Sinus of Valsalva Aneurysms

Concomitant Lesion	References
Ventricular septal defect	[Azakie 2000]
Aortic valve incompetence	[Henze 1983, Mayer 1986, Abe 1988, Barragry 1988, Burakovsky 1988, Chu 1990, Isomura 1994, van Son 1994, Pannu 1995, van Son 1995, Choudhary 1997, Azakie 2000, Vural 2001, Dong 2002]
Atrial septal defect	[Mayer 1986, Burakovsky 1988, Zikri 1999]
Bicuspid aortic valve	[Henze 1983, Burakovsky 1988, Azakie 2000]
Left ventricular outlet obstruction	[Ates 2002]
Patent ductus arteriosus	[Burakovsky 1988, Vural 2001, Ates 2002]
Aortic stenosis	[Mattila 1987, Vural 2001]
Tetralogy of Fallot	[Mattila 1987, Barragry 1988, Hamid 1994]
Pulmonary stenosis	[Henze 1983, Barragry 1988, Burakovsky 1988, Chu 1990, Hamid 1994, Isomura 1994]
Coarctation of the aorta	[Mattila 1987, Chu 1990]
Mitral valve regurgitation	[Mayer 1986, Mattila 1987]
Left superior vena cava septal defect	[Pannu 1995]
Patent foramen ovale	[Mayer 1986]

degeneration [Tak 2002], Marfan syndrome, and senile dilation [Amoah 2000]. SVAs associated with cystic medial necrosis are a result of mucoid degeneration and fragmentation within the media of the aortic root walls [Tak 2002]. All forms of SVAs can rupture and result in an RSVA.

DEMOGRAPHICS

The published literature describes demographic differences for RSVAs with respect to incidence and age. The reported incidence of RSVAs is higher in Eastern countries than in Western countries, with 1 series finding a 5-times more likely rate of RSVAs among East Asians compared with Western populations [Chu 1990]. In addition, the age of rupture onset is more evenly distributed across a wider age range in Western countries, compared with the relatively younger age distribution and the rarer reported incidence of rupture at either end of the age range seen in Eastern countries [Chu 1990]. More recent series support the prior observations of different age ranges for presentation of RSVAs in Eastern versus Western populations. In Eastern countries, a series from China had a mean (± SD) age of 32 ± 10 years (n = 67) [Dong 2002], and a series from Japan reported a mean age of 27.5 ± 14.4 years (n = 35) [Murashita 2002]. By comparison, a contemporary US series had a mean age of 42.2 ± 16.7 years (n = 24) [Zikri 1999]. Recent series published from groups in India (mean age, 27.51 ± 3.21 years [n = 104] [Choudhary 1997]) and Turkey (mean age, 24 ± 12 years [n = 53] [Vural 2001]; 28.3 ± 10.7 years [n = 20] [Kirali 1999]) indicate that these populations more closely resemble Eastern populations in their incidence of RSVAs.

Gender variation in incidence, however, seems to be similar across ethnic lines with a 4:1 male preponderance [Goldberg 1990]. Overall, young East Asian males are the highest risk group for RSVAs [Karavidas 2000]. The high magnitude of the male-female ratio may be multifactorial and may reflect different pathologies, gender differences in health care use, or both.

CONCOMITANT LESIONS

There is a wide variety of concomitant lesions among patients with RSVAs. The most commonly associated concomitant lesion is a VSD [Henze 1983, Mayer 1986, Mattila 1987, Abe 1988, Burakovsky 1988, Hamid 1994, Isomura 1994, Pannu 1995, Choudhary 1997, Kirali 1999, Azakie 2000, Vural 2001, Dong 2002, Murashita 2002]. Other concomitant lesions seen frequently with RSVAs are summarized in the Table.

Among the reports of 5 Eastern series that described RSVAs in 206 patients, VSD and aortic valve incompetence were the most frequently associated concomitant lesions; only 2 cases of patients with pulmonic stenoses and 1 case of coarctation of the aorta have been reported [Abe 1988, Chu 1990, Isomura 1994, Dong 2002, Murashita 2002]. In contrast, RSVAs reported from Western series show more variation in the concomitant lesions, which ranged from VSDs and aortic regurgitation to bicuspid aortic valves, pulmonic stenosis, atrial septal defects, tetralogy of Fallot, patent ductus arteriosus, coarctation of the aorta, and subaortic stenosis [Azakie 2000]. An Eastern-Western comparative series also found such variation to be the case, supporting the conclusion that the clinical lesions associated with RSVAs are more uniform among Eastern patients [Chu 1990].

CLINICAL PRESENTATIONS

The clinical presentations of ruptured and unruptured SVAs vary dramatically. Almost 20% of SVAs are unruptured and are diagnosed only once the patient has undergone surgery or autopsy [Murray 1993]. Unruptured SVAs are usually clinically silent and therefore may be underreported in the literature [Goldberg 1990]. Symptomatic unruptured aneurysms can present, however, as sudden death [Engel 1981], coronary ostial obstruction resulting from thrombus [Kwittken 1965], pulmonary outflow obstruction, tricuspid regurgitation, conduction disturbances, severe aortic insufficiency as a result of compression/distortion of the surrounding structures [Vural 2001, Ates 2002], cardiomegaly [Ates 2002], or an aneurysm burrowed into the interventricular septum [Choudhary 1997].

Most SVAs, however, are clinically undetected until rupture [Goldberg 1990]. Ruptured SVAs can present suddenly, and approximately 25% of RSVAs result in the sudden death of the patient [Turgeon 2003]. Approximately 80% of surviving patients with RSVAs are symptomatic [Hamid 1994, Tur-

geon 2003]. The most common clinical presenting symptoms include dyspnea, paroxysmal nocturnal dyspnea, fatigue, palpitations, syncope, angina, endocarditis, edema, arterial hypertension, atrial fibrillation, or supraventricular tachycardia [Goldberg 1990, Azakie 2000, Dong 2002, Murashita 2002, Tak 2002, Turgeon 2003]. More than 50% of patients reportedly have a gradual onset of symptoms [Mayer 1986]. The progressive manifestation of symptoms can result from the gradual enlargement of a small rupture [Goldberg 1990]. The severity of a patient's symptoms may also vary because of the presence of a VSD or aortic insufficiency [Hamid 1994]. Symptomatic patients frequently present with cardiac enlargement, and pulmonary congestion is often present in those with severe enlargement [Chu 1990]. RSVAs are also commonly associated with the finding of a harsh systolic murmur at the left sternal border [Hamid 1994].

The clinical impact of an RSVA is largely determined by the cardiac chamber into which the aortocardiac fistula forms, creating a volume overload for either the left or right heart. RSVAs most commonly rupture into the right ventricle, followed by the right atrium and then the left ventricle [Kirali 1999]. Ruptures of the right and noncoronary sinuses of Valsalva often create fistulas into right-sided chambers [Goldberg 1990], resulting in left-to-right shunts. Tricuspid insufficiency may result from ruptures into the right ventricle just below the tricuspid valve. Aortic insufficiency can also result if the RSVAs deform the aortic cusps or interfere with normal aortic leaflet motion or coaptation [Tanabe 1979]. Ruptures of the left sinus of Valsalva create fistulas into left-sided chambers. In addition to rupture and fistula tract formation, aneurysmal enlargement of the SVA may protrude into the ventricular outflow tract [Sakakibara 1962, Bulkley 1975]. Although rare, ruptures of noncoronary SVAs can also result in fistulas into the pericardium and cause sudden death [Perloff 1987].

DIAGNOSIS, SURGERY TREATMENTS, AND OUTCOMES

Hope was the first to describe an SVA after he discovered it during an autopsy in 1835 [Hope 1985]. Before the advent of noninvasive imaging techniques, cardiac catheterization was the only means by which to diagnose RSVAs. Less invasive imaging studies can now be used to diagnose and evaluate RSVAs. Transthoracic and transesophageal echocardiography can reveal the location and magnitude of the shunt as well its physiologic and anatomical orientation. Magnetic resonance imaging may also facilitate the identification of RSVAs, but it has not yet been widely used [Goldberg 1990]. Other diagnostic studies are also important in the evaluation of the pathophysiologic effects of RSVAs on the heart. Radiography can demonstrate cardiomegaly, and electrocardiography can detect conduction system impairment or arrhythmias.

Lillehei and his colleagues first successfully repaired an RSVA in 1956 with techniques acquired in the development of cardiopulmonary bypass surgery [Lillehei 1957]. The following year Morrow used a hypothermic inflow occlusion technique to repair an RSVA [Morrow 1957]. Since then, significant advances have been made in the surgical treatment of RSVAs.

Surgical repairs of RSVAs are of low risk and generally have an excellent long-term prognosis [Zikri 1999]. As a result, many authors believe that early surgical intervention in patients with an RSVA is justified [Henze 1983, Mayer 1986, Abe 1988, van Son 1995, Choudhary 1997, Zikri 1999, Azakie 2000]. The most routine form of repair entails an approach through the aorta. An aortic approach is widely considered the safest approach to obtaining closure at the base of the aneurysms because it provides excellent visibility and access to a firm base for stitches while at the same time minimizing the risk of injury to the right coronary artery or the aortic valve [Henze 1983]. The advantages of this approach are further supported by a series study that found that the risk of reoperation in the repair of right ventricle fistulas was higher when a right ventriculotomy was used alone than when it was combined with an aortotomy [van Son 1994].

Patch and primary-suture techniques are the 2 principal means by which RSVAs are closed. There is evidence that the patch technique may be the more effective repair. In 1 series, recurrence of the fistula was correlated with primary repair but not with patch repair [Azakie 2000]. Another series found that 30% of the patients who had their RSVA closed by suture had to have reoperations for recurrent fistulas and or recurrent VSDs; only 13% of the patients who had their RSVA closed with a patch had to have a reoperation for these recurrent problems [Abe 1988]. Dramatically, another series found that all patients with patch closure had a 100% actuarial freedom of recurrence of fistulas at 12.35 years [Kirali 1999].

Perioperative mortality rates associated with RSVA repair are relatively low, ranging from 1.9% to 11.8% [Pan 1981, Vural 2001]. Among the series we studied, mortality was attributed to a variety of causes, including the development of a low cardiac output after surgery [Vural 2001], congestive heart failure immediately after surgery [Abe 1988], complete heart block intraoperatively [Barragry 1988], intraoperative cardiac tamponade [Barragry 1988], septicemia and multiorgan failure [Hamid 1994], generalized septic peritonitis [Burakovsky 1988], acute heart failure during surgery [Burakovsky 1988], recurrent ventricular arrhythmias resistant to treatment [Choudhary 1997], toxic epidermonecrosis followed by disseminated intravascular coagulation [Choudhary 1997], and death unrelated to cardiac causes [Zikri 1999].

Similarly, perioperative morbidity, although infrequent, has presented in a variety of complications, including a case of acute renal failure [Zikri 1999], 2 cases requiring permanent pacemaker insertions [Zikri 1999], an anticoagulation complication [Dong 2002], 2 cases of residual shunts [Dong 2002], a case of aortic valve incompetence [Dong 2002], a case of endocarditis [Barragry 1988], a case of recurrent subacute endocarditis [Hamid 1994], a case of recurrent fistulation with small shunt [Mattila 1987], a case of atrial fibrillation [Mattila 1987], and a case of wound infection [Burakovsky 1988]. A few series have examined reoperative risk. The evaluation of 1 series found that the risk of reoperation is higher in right ventricular fistulas than with right atrial fistulas [van Son 1994]. Another study found that late aortic regurgitation is significantly associated with preoperative or early postoperative aortic regurgitation [Murashita 2002]. Nevertheless, the analysis of a long-term case series demon-

strated a clear majority of the postoperative patients in New York Heart Association class 1 or 2 at long-term follow-up periods ranging from 8.2 to 23.6 years [Zikri 1999].

CONCLUSION

We have described 4 RSVA cases, each demonstrating a different manner in which an RSVA may present clinically. RSVA epidemiology, concomitant lesions, clinical presentations, and repair techniques have also been presented through comparisons of reported case series from around the world. Comparisons of the results from Eastern and Western series reveal that the incidence of RSVAs is higher in Eastern countries than in Western countries, with a 4:1 male preponderance occurring across ethnic lines. Among the Eastern series reporting RSVAs, VSDs and aortic valve incompetence were the only frequently associated concomitant lesions. In contrast, Western series of RSVAs showed a wide range of concomitant lesions. The difficulty in diagnosing RSVAs is mainly due to the variability in their clinical impact and presentation. These factors largely depend on the cardiac chamber into which the aortocardiac fistula forms. The advent and availability of transesophageal and transthoracic echocardiography enable RSVA diagnosis with greater precision. Surgical repairs of RSVAs are of low risk and generally have an excellent long-term prognosis. As a result, many authors believe that early surgical intervention in patients with an RSVA is justified. Among the series we studied there is evidence that the patch technique is the safest approach because of its lower association with the recurrence of fistulas. RSVAs may present a diagnostic dilemma because of their varied clinical presentations. However, if included on a differential, they are easily diagnosed and surgically treated.

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