

muscle alone, without the muscle fascia, the initial hemostatic closure is difficult and the skeletal muscle exposed to circulation is highly thrombogenic [5]. We determined that hemostatic closure of the full-thickness defect was easy to obtain with de-epithelized skin. During 17 months of follow-up there has been no thrombus formation in the right ventricular cavity.

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Traumatic Aorto-Right Ventricular Fistula With Aortic Insufficiency

Abdullah Kaya, MD, Paul Dekkers, MD,
Antonino Loforte, MD, Wybren Jaarsma, MD, PhD, and
Wim J. Morshuis, MD, PhD

Departments of Cardiothoracic Surgery and Cardiology, St. Antonius Hospital, Nieuwegein, the Netherlands

We present a case of a traumatic aorto-right ventricular fistula coexistent with aortic insufficiency due to perforation of the left coronary leaflet, which is a lesion rarely described in the literature. We compare our experience with reports from the literature.

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Since the first case report of traumatic aorto-right ventricular fistula in 1958, there have been 42 case reports in literature [1-3]. Only 17 of these describe the combination of traumatic aorto-right ventricular fistula

with aortic insufficiency [2, 3]. We present a case of an aorta to the right ventricular fistula combined with aortic insufficiency after a penetrating trauma. The aim is to compare our experience with the various approaches described in the literature.

A 19-year-old man was stabbed with a small bladed stiletto knife in the left third intercostal space adjacent to the sternum. On admission, the patient was alert and responsive with stable vital signs. There was no significant past medical history or medication. There was no thrill or murmur heard on examination. Chest roentgenogram showed fluid in the left hemithorax. A mild to moderate amount of pericardial effusion associated with left-sided pleural fluid was detected by transthoracic echocardiography. Mild aortic valve insufficiency and a small ventricular septal defect was also seen (Fig 1). Unfortunately the patient refused transesophageal echocardiography, which could have given more detailed information about the pathology. A left-sided chest drain produced 1,270 mL of serosanguineous fluid over a 2-day period. At reevaluation by transthoracic echocardiography the findings were unchanged. The patient was hemodynamically stable and maintained adequate oxygen saturation. He had no complaints and was optimally mobilized. He made an uneventful recovery and insisted on being discharged against medical advice. He did not report for follow-up at the outpatient clinic. A month later he was urgently readmitted due to severe dyspnea. A continuous pre-cordial murmur was heard on auscultation and congestion of his jugular veins was evident. Pericardial tamponade was confirmed by transthoracic echocardiography and a subxiphoid pericardiocentesis was performed with 1,060 mL of blood evacuated. Reevaluation with transthoracic echocardiography confirmed moderate aortic valve regurgitation, an increased left to right shunt between the aortic root and the right ventricular outflow tract, and a moderately dilated right ventricle (Fig 1). At surgery, a median sternotomy was performed, the pericardium was opened, and the epicardial adhesions were released. On cardiopulmonary bypass with double venous cannulation, cold crystalloid cardioplegia was selectively infused through the coronary ostia until a septal temperature of 10°C was achieved. The aorto-right ventricular communication was exposed through the transverse aortotomy. An imaginary line could be drawn from the lacerated left coronary cusp, crossing the interleaflet triangle between the left and right coronary cusp, penetrating the right ventricular outflow tract (Fig 2). A small opening was also noticed in the pericardium covering the right ventricle, thus confirming the trajectory of the penetrating injury. No superficial entry wound was found on the right ventricle, probably due to the adhesions. The septal communication was closed through the aortotomy with continuous 5-0 polypropylene suture. A small (5 mm) clean cut longitudinal laceration at the base of the left coronary cusp of the aortic valve could be repaired primarily with a double layer continuous 7-0 polypro-

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Address correspondence to Dr Kaya, Department of Cardiothoracic Surgery, St. Antonius Hospital, Koekoekslaan 1, 3435 CM Nieuwegein, the Netherlands; e-mail: a_kaya33@hotmail.com.

pylene suture (Fig 2). Postoperatively to the repair, an intraoperative transesophageal echocardiographic evaluation showed no evidence of aortic valve insufficiency or left to right shunt. The postoperative course was uneventful and the patient was discharged on postoperative day 5. To our disappointment, thus far the patient has continued to abstain from following up at the outpatient clinic.

Comment

Traumatic aorto-right ventricular fistulas with aortic insufficiency are rare lesions after penetrating thoracic injuries. According to the literature this specific lesion has been reported 17 times previously [2, 3].

The time interval between injury and surgical intervention is variable. Some patients require immediate surgical management due to instable hemodynamics, but others may have a delayed clinical presentation and therefore a delayed repair [3-5]. The interval until definitive repair could be as long as 17 years, as reported by Ehrenstein and colleagues [6]. In this case, the time interval between injury and repair was 56 days. The propensity for shunts in aorto-right ventricular fistulas to

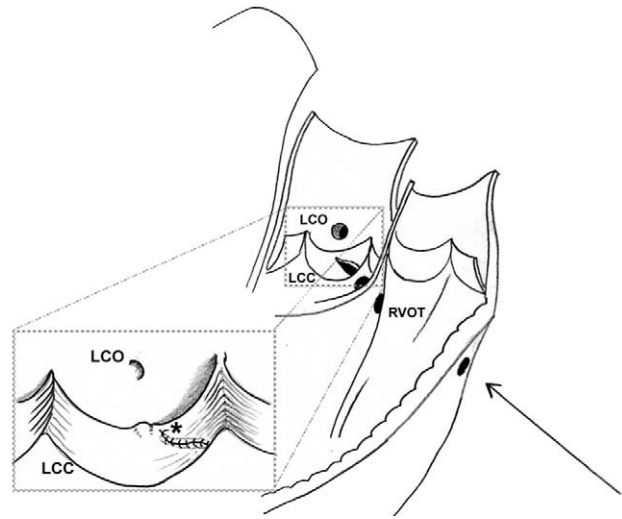


Fig 2. Artist impression of an intracardiac direction of penetrating injury (arrow). Inset: postoperative repair diagram of left coronary cusp (asterisk). (LCC = left coronary cusp; LCO = left coronary ostium; RVOT = right ventricular outflow tract.)

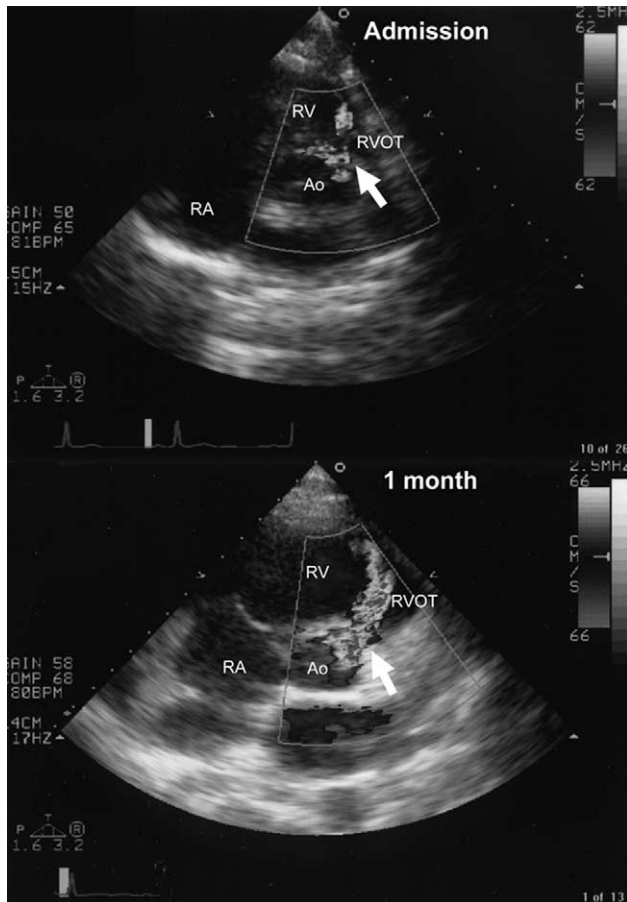


Fig 1. Parasternal short axis view showing increased left to right shunt (arrow) after 1 month. (Ao = aorta; RA = right atrium; RV = right ventricle; RVOT = right ventricular outflow tract.)

increase in size with time may explain the delayed time interval to definitive repair as reported by some authors [3-5, 7]. All patients with a traumatic aorta to right ventricular fistula combined with aortic insufficiency (except for one patient) were operated on sooner or later, as reported in the review by Samuels and colleagues [2]. Our experience confirms that a traumatic aorto-right ventricular shunt with aortic insufficiency has a tendency to increase in size with time. Therefore it is advisable that these patients be operated on at an early stage.

Although patients with aorto-right ventricular fistula combined with aortic insufficiency after a penetrating trauma may have no cardiac symptoms, they should be thoroughly evaluated, preferably by transesophageal echocardiography, and operated on during the same admission. If left untreated, congestive heart failure will invariably develop.

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Saccular Aneurysm of the Aortic Cusp Associated With Discrete Subaortic Stenosis

Seiji Matsukuma, MD, Kiyoyuki Eishi, MD, Shiro Yamachika, MD, Tomayoshi Hayashi, MD, PhD, Shiro Hazama, MD, Hiroichiro Yamaguchi, MD, Tsuneo Ariyoshi, MD, Hideaki Takai, MD, Tomohiro Odate, MD, Daisuke Onohara, MD, and Makoto Yanatori, MD

Department of Cardiovascular Surgery, Nagasaki University School of Medicine, Nagasaki, Japan

A 54-year-old male who experienced a syncopal episode underwent aortic valve replacement for aortic stenosis and regurgitation. The aortic valve was incompetent as a result of thickening of the left coronary cusp and non-coronary cusp. In addition a saccular aneurysm was indicated on the left coronary cusp. A shelf of tissue protruding at right angles from the ventricular septum was particularly prominent below the right coronary cusp, resulting in subvalvular stenosis. The cause of the saccular aneurysm was most likely caused by the long-term effects of the jet stream instigated by discrete subaortic stenosis.

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Saccular aneurysm of the aortic cusp is extremely rare and most valvular saccular aneurysms result from infective endocarditis. This report describes a saccular aneurysm of the left coronary cusp associated with discrete subaortic stenosis (DSS). Saccular aneurysms associated with DSS are not currently available, although DSS is itself associated with several congenital anomalies including aortic valve abnormalities.

A 54-year-old male who had experienced a syncopal episode and chest pain was referred for surgical treatment of aortic stenosis (AS) and aortic regurgitation (AR). The patient exhibited a history of heart murmur since childhood. In addition occasional dyspnea and palpitation developed. When the patient was 31 years of age, it was indicated that he possessed a double chamber right ventricle (DCRV) and underwent resection of the anomalous muscle bundle in the right ventricle. At that time enlargement of the ascending aorta and grade I AR were detected by aortography, but no remarkable pressure gradient was evident across the aortic valve. On admis-

sion a two-dimensional echocardiography revealed a 15-mm length of fixed subaortic obstruction on the thickened interventricular septum (Fig 1). Doppler echocardiography revealed grade III AR and a pressure gradient of 54 mm Hg across the subaortic obstruction. The coronary arteries and neck vessels were intact and there was no history of infection or endocarditis.

The operation performed was a median sternotomy using a standard cardiopulmonary bypass with cold crystalloid cardioplegia. The ascending aorta was opened through a horizontal incision. When the aortic cusps were retracted the subvalvular shelf and the saccular aneurysm of the left coronary cusp were exposed (Fig 2A). The shelf was located approximately 1 cm proximal to the base of the aortic cusps and was particularly prominent below the right coronary cusp. The aortic valve was tricuspid but was incompetent because of thickening of the left coronary cusp, whereas the right coronary cusp seemed to be relatively less degenerated than the other cusps. The aneurysm exhibited an orifice surrounded by a thickened portion on the aortic side of the cusp (Fig 2B). After the aortic valve and subvalvular shelf were resected, aortic valve replacement was performed using a mechanical valve (24 mm ATS AP).

Microscopic examination revealed that the bundle of elastic fibers of the left coronary cusp was curved toward the ventricular side at the orifice of the saccular aneurysm. At the top of the saccular aneurysm, the wall had become thinned and the bundle of elastic fibers disappeared (Fig 3). Furthermore, there was no evidence of infective endocarditis in the wall of the saccular aneurysm.

Comment

Four occurrences of saccular aneurysm of the aortic cusp have been reported [1-4], but literature concerning saccular aneurysm of the aortic cusp accompanied by DSS is not currently available. DSS was described in 1842 by

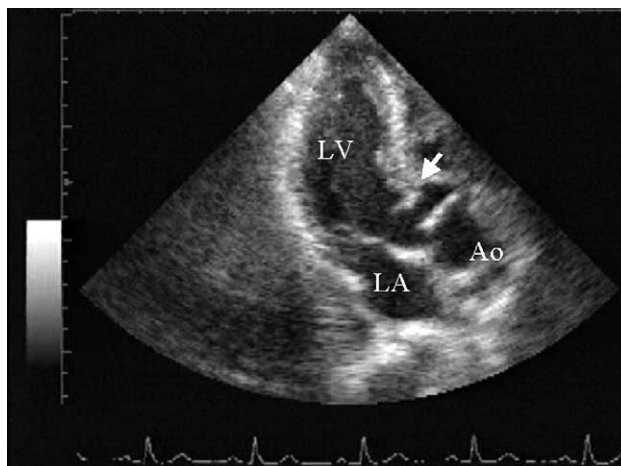


Fig 1. Transthoracic echocardiography revealed a 15-mm length of fixed subaortic obstruction (arrow) on the thickened interventricular septum at the left ventricular (LV) outflow tract. (Ao = aorta.)

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Address correspondence to Dr Matsukuma, Nagasaki University School of Medicine, Department of Cardiovascular Surgery, 1-7-1 Sakamoto, Nagasaki City, Nagasaki 852-8501, Japan; e-mail: seiji731@net.nagasaki-u.ac.jp.