CASE REPORT

Real-time Observation of Ruptured Right Sinus of Valsalva Aneurysm by High Speed Ultrasono-cardiotomography

Report of a Case

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SUMMARY A two-dimensional echocardiographic manifestation of a ruptured right sinus of Valsalva aneurysm is described in this case report. The ultrasono-cardiotomograms demonstrated the aneurysm cut longitudinally and protruding into the outflow tract of the right ventricle. The configuration of the aneurysm was consistent with the contrast medium-filled sinus demonstrated by angiocardiography. In systole, the aneurysmal sinus moved antero-inferiorly together with the aortic wall. The echocardiogram revealed an abnormal two-layered horizontal echo in the right ventricle which showed the aneurysm when the ultrasonic beam passed through the interventricular septum near the aortic root and mitral leaflets. During the operation, the protruding sinus and a small interventricular septal defect in the membranous septum under it were observed through the opened right ventricle. After the closure of the aneurysm, the two-dimensional echocardiogram no longer showed an abnormal configuration.

SINCE ULTRASOUND has been applied to the diagnosis of cardiac diseases, it has proved useful especially in anatomical and functional assessment of the structures of the left-sided heart, such as the mitral valve, aortic valve and left ventricle. Abnormal aortic wall echoes have been reported in such conditions as dissecting aneurysm and Marfan's syndrome. Some cases of right sinus of Valsalva aneurysm have been described, but they have not distinctly revealed the nature of the ruptured sinus itself. In the present report we describe a case in which the structure was demonstrated by high-speed ultrasono-cardiotomography and confirmed by operation.

Methods

Ultrasono-cardiotomograms and echocardiograms were obtained using a Sonolayergraph (Toshiba, Model SSL-51H) with a mechanical sector scanning system. A transducer, focused at 75 mm, which was made of lead zirconate-titanate, measuring 10 mm in diameter with a resonant frequency of 3 MHz, was used at the repetition rate of 3.6 KHz. It was mechanically oscillated at the rate of 15 or 18 cycles a second (30 or 36 cross-sections per second) by a scanner which was controlled to rotate alternately clockwise and counterclockwise. The oscillating angle of the transducer was detected by a potentiometer. The detected signals of rebounding echoes were displayed on oscilloscopes as B-mode so that the angle of an individual scanning line was equal to the angle of the transducer at the same instant. The oscillating angle of transducer was arbitrarily opened to the maximum of 90°, but the display angle on the oscilloscope was fixed at about 65°. When the oscillating angle of the transducer was smaller than the display angle on the oscilloscope, one image was composed of 120 or 100 scanning lines. When the former was greater than the latter, the scanning lines beyond the border were eliminated. To enable identification of the echo source, a particular scanning line was selected manually and B-mode was switched to M-mode to obtain the echocardiogram.

The patient was examined in the supine position. For coupling between the transducer and his chest, the so-called "proximity-immersed method" was employed and instead of degasified water, castor oil was utilized. The transducer was immersed in the oil bath placed on the chest surface and was brought close to the chest until multiple echoes from the bottom of the bath were eliminated.

Two methods were utilized to record the image on the oscilloscope screen: (1) a Polaroid camera or an ordinary 35 mm camera; and (2) an 8 mm movie camera. Two ultrasono-cardiotomograms in this case report were taken at a shutter speed of 1/15 second by the ordinary camera.

Case Report

The patient was a 44-year-old man. His infancy was uncomplicated and he had a normal development. At the age of three years, he was first noted to have a heart murmur on routine examination, but he showed no symptoms during childhood and adolescence, enjoying sports without any restrictions. However, beginning in May of 1974, he experienced palpitation on exertion. On the night of August 17, 1974, he suddenly complained of dyspnea and his case was diagnosed as acute heart failure. Admitted to the
Gamagori Municipal Hospital, he was given intensive care with oxygen, cardiotonics and diuretics, and showed improvement within a few days. He remained in the hospital until the 29th of October. After discharge, he was referred to the Nagoya University Hospital, where he was admitted for closer examination on November 5, 1974.

There was no history of rheumatic fever or syphilis. His father died from congestive heart failure.

Physical examination on admission revealed a small stature (159.2 cm, 52 kg) and pale skin color. Body temperature was 36.5°C. Blood pressure was 116/50 mm Hg and the pulse was 68 and regular. The neck veins were distended and the liver extended 2 cm below the right costal margin. There was no ankle edema and the lung fields were clear. A broad cardiac impulse was located on the precordium, and systolic and diastolic thrills were palpable. On auscultation, a grade VI/V1 continuous, harsh murmur, maximal in the 4th intercostal space along the left sternal border, was audible over a wide area. The first and second sounds were of normal intensity, whereas the third and fourth sounds were not audible.

Laboratory examination revealed no anemia. Electrolytes, serum transaminase, LDH, alkaline phosphatase, bilirubin and creatinine were within normal ranges. Tests for C reactive protein and rheumatoid arthritis factor were negative and the antistreptolysin-O titer was 100 Todd units.

Electrocardiogram showed left ventricular hypertrophy ($RV_1 = 4.1 \text{ mV}, SV_1 = 2.2 \text{ mV}$, ST depression in II, aV_{II}, V_6, V_3) and negative T in leads V_1 to V_4. Ventricular premature beats resembling left bundle branch block were occasionally recorded.

Chest X-ray demonstrated cardiomegaly; the left and right ventricular segments protruded markedly and the cardiothoracic ratio was 66.7%. Pulmonary vessels were engorged.

Ultrasoundograms in figures 1 and 2, obtained by scanning the intermediate plane between the cross-sections of the long axis of the left ventricle and the sagittal plane along the left parasternal line, revealed the ruptured right sinus of Valsalva aneurysm. In normal subjects, the anterior aortic wall is in continuity with the interventricular septum, whereas in this case, the lower portion of the anterior aortic wall adjacent to the septum, that is, the right sinus of Valsalva, protruded into the right ventricular outflow tract and the echo-interruption of its tip showed it to be ruptured. In systole, the ruptured aneurysm moved slightly downward and forward. The sinus in systole was less oblique than diastole. The right coronary cusp seemed to cover the sinus in systole. A ventricular septal defect was not recognized. The echocardiogram showed an abnormal double-layered echo in the right ventricle which was recorded only during the period from mid-systole to early diastole (fig. 3). This echo was obtained when the ultrasonic beam penetrated the protruding sinus (beam direction A shown in fig. 2). When the transducer was angled higher (beam direction B shown in fig. 2), the more complicated echoes indicating the aneurysm were recorded in front of the base of the anterior mitral leaflet (fig. 4). When the transducer was angled even higher, the echoes of the anterior and posterior aortic walls were clearly detected. The right coronary cusp showed a dip in early systole (fig. 5).

Right and left heart catheterization was performed and the hemodynamic and oxygen content data are summarized in table 1. Right atrial pressure (27/13, mean 21 mm Hg) and main pulmonary artery pressure (76/28, mean 48 mm Hg) were elevated. Dye dilution curve showed left-to-right shunt and a significant step-up of oxygen content was found at the right ventricle.

Angiocardiography showed the regurgitation of contrast medium from the aorta to the right ventricle through the ruptured right sinus of Valsalva aneurysm protruding anteriorly (fig. 6).
Marfan’s syndrome, ankylosing spondylitis and bacterial endocarditis. The patient under study showed no findings to suggest an acquired cause, and his condition ruled out Marfan’s syndrome and ankylosing spondylitis. For these reasons, his sinus of Valsalva aneurysm was considered a congenital anomaly.

Generally, in the congenital type, the basic defect is believed to be an absence of the media in the sinus wall, which appears to be due to a lack of continuity between the media of the aorta and the annulus of the aortic valve. When the defect of the aortic media occurs, the wall behind the right coronary cusp most commonly is involved. Moreover, an aneurysm of the right sinus usually protrudes into the right ventricle. The aneurysm is easily recognized clinically when it ruptures. It is associated with a new, continuous, loud murmur, progressive heart failure and pulmonary congestion. It resists any medical treatment except surgical closure. The most reliable diagnostic measure for this condition is angiography. However, ultrasono-cardiography can delineate the ruptured aneurysm protruding into the right ventricle. Moreover, the echographic configuration of the sinus is very similar to that seen by angiography.

In our experience, there are two other entities which must be differentiated from the sinus of Valsalva aneurysm. The first is the weak echo at the junctional area between the

![Figure 3](image3.png)

**Figure 3.** An echocardiogram from the same patient. Ultrasound beam penetrated the anterior wall of right ventricle, right ventricular cavity, the protruding right sinus, interventricular septum, anterior and posterior mitral leaflets, the junction between left atrium and left ventricle. An abnormal two-layered echo in the right ventricle appears at mid-systole and disappears at early diastole. It indicates the sinus protruding into the right ventricle.

At operation, which was performed on April 16, 1975, there was remarkable dilatation of both ventricles and little pericardial effusion. On the wall of the right ventricle, a strong thrill was palpated and observed. When the right ventricle was opened, a white protruding sinus was found. Moreover, a small ventricular septal defect (5 x 7 mm) was located in the membranous septum under the protruding aneurysm.

**Discussion**

There are many known causes of sinus of Valsalva aneurysm, such as congenital anomaly, atherosclerosis, syphilis,
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Figure 6. Lateral angiocardiogram shows the right sinus protruding anteriorly. The contrast medium regurgitates into the right ventricular cavity (between arrows).

Figure 6. Lateral angiocardiogram shows the right sinus protruding anteriorly. The contrast medium regurgitates into the right ventricular cavity (between arrows).

anterior aortic wall and the interventricular septum which occasionally is seen in the subjects with slightly dilated right aortic sinus. An echo defect is observed at the junctional area in some subjects. But these findings do not imply the presence of ruptured right sinus of Valsalva aneurysm. The major difference is that the right sinus in such subjects is not as large as the aneurysm. In addition, an echo defect is narrower than the echo-interruption from the ruptured aneurysm. The second entity is an interventricular septal defect including tetralogy of Fallot. This is more easily differentiated since the septal defect does not show a protrusion into the right ventricle. However, an aneurysm of the membranous ventricular septum with perforation and a pouch of tricuspid valve associated with ventricular septal defect may mimic the right sinus of Valsalva aneurysm very closely. The greatest difference is that the septal defect is due to the interruption of the septum below the aortic cusp echo and the ruptured right sinus of Valsalva aneurysm is due to interruption of the aortic wall over the aortic cusp echo.

The echocardiogram revealed an abnormal echo in the right ventricle, that is, a two-layered, canal-like pattern indicating the aneurysm itself, something which has never been described. It was recorded throughout mid-systole to early diastole because the protruding sinus had been moved into the path of the ultrasonic beam by cardiac contraction and then been moved out of its path by cardiac relaxation.

Cooperberg and coworkers reported that the cardinal echographic findings in their case were a defect in the anterior aortic root through which the right coronary cusp appeared to prolapse, and an abnormal echo in front of the defect. Matsuo and coworkers also reported a similar case. However, such findings differed from the present echographic patterns. In both cases, the posterior wall of the aneurysm was not detected, probably due to the positional gap between the direction of the ultrasonic beam and the posterior wall. In contrast, we could successfully record both anterior and posterior walls of the aneurysm, since the ultrasonic beam penetrated them. Moreover, Johnson and associates reported the abnormal systolic dip of the right coronary cusp, which is consistent with the present finding.

It is concluded that two-dimensional echocardiography and conventional echocardiography are useful means to diagnose ruptured right sinus of Valsalva aneurysm.

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