Case Reports

Congenital Aneurysm of the Right Coronary Sinus of Valsalva with Rupture into the Left Ventricle

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ANEURYSM of the sinus of Valsalva is a rare abnormality. Since the first description by Thurmann in 1840, about 50 cases have been reported. Among these, a case of an aneurysm of the sinus of Valsalva which ruptured into the left ventricle was described by Warthen11 in 1949.

The purpose of this paper is to present a case of a congenital aneurysm of the right coronary sinus of Valsalva which ruptured into the left ventricle, and to discuss this abnormality.

CASE REPORT

A 52-year-old man was admitted to the Tokyo University Hospital on November 24, 1958, with complaints of “exertional dyspnea and palpitation” for 3 months.

Patient had been well till September 5, 1958, when he experienced severe dyspnea associated with precordial pain. At the time of this occurrence patient was running fast to get on a train. On September 8, 1958, he entered another hospital where a diagnosis of the combined valvular disease was made. He was treated with salt restriction and digitalization. However, against a doctor's advice he discharged himself from the hospital. After a short period of time, especially paroxysmal nocturnal dyspnea increasingly became worse. At the same time, he noted the shortness of breath on walking or climbing stairs and had a productive cough with a white foamy sputum. On November 24, 1958, he was admitted to the Tokyo University Hospital. He had no history of syphilis or rheumatic fever. Family history was noncontributory.

Physical Examination: No cyanosis present. Blood pressure was 150/0 mm. Hg; pulse regular and rate 100 per minute; respiration regular and 20 per minute; temperature 98°F. There was no periorbital edema and conjunctival pallor. Pupils reacted normally to light and the fundi appeared normal. Tonsils were small and normal in appearance. Neck veins were moderately distended and pulsating in sitting position. Venous pressure was 120 mm. in water. Carotid and sub-

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clavian arteries pulsed vigorously. No mass or node was palpable in the neck. The examination of the lung revealed moist rales and dullness at the right lung base. The cardiac apical beat was forceful and localized to an area in the left fifth intercostal space 2 cm. lateral to the midclavicular line. The right heart border was percussed 1 cm. lateral to the sternum. The left heart border was percussed 3 cm. lateral to the left midclavicular line. On auscultation there were a soft systolic and an early high-pitched diastolic murmur of maximum intensity in third left intercostal space in the parasternal line. These murmurs were transmitted along the left sternal border to the apex. At the apex, a weak systolic and a diastolic murmur were heard. The machinery continuous murmur was not heard. The liver was palpable two finger-breadths below the right costal margin and slightly tender. The spleen was not palpable. No abdominal masses were palpable. There was no ascites and no edema. Neurologic examination was normal.

Laboratory Data: Urinalysis; specific gravity 1.025, yellowish colored, albumin trace, sugar negative, bile and urobilinogen negative, sediment negative.

Hemogram: hemoglobin 70%, R.B.C. 385 millions, W.B.C. 5800 with an essentially normal differential, platelet 16.9 millions, sedimentation rate 60 mm. per hour. Wassermann reaction was negative.

Blood chemistry; blood non-protein nitrogen 26.5 mg./100 ml., total cholesterol 178 mg./100 ml. Electrolytes were all within normal range, total serum protein 8.0 Gm./100 ml. Serum albumin and globulin ratio was within normal limits. C-reactive protein was two plus.

Roentgenogram; P-A chest X-ray (Fig. 1) revealed cardiac enlargement with the prominence of the left ventricular contour. Cardiac fluoroscopy demonstrated enlargement of the left ventricle, left atrium and right ventricle.

Electrocardiogram (Fig. 2) was suggestive of left ventricular hypertrophy. There was slight depression of the ST segment in leads I, II, aV_F, V_5 and V_6. On the phonocardiogram (Fig. 3), a high-pitched decrescendo diastolic murmur immediately following the second sound and an early systolic murmur with a low amplitude
were recorded at the third left intercostal space. A low-pitched protodiastolic murmur simulating mitral diastolic rumble was recorded at the apex.

*Hospital Course*: Following admission, patient had moderate nocturnal paroxysmal dyspnea every night. He was treated with bed rest, low salt diet (NaCl 5 Gm. daily), digitalis (Digitoxin 0.15 mg. daily), chlorothiazide (0.5 to 1 Gm. daily), and aminophylline (250 mg. intravenously). There was significant response to the therapy and the patient became more comfortable. However, at the end of February, 1959, congestive heart failure became gradually manifest.
He complained of epigastric discomfort, anorexia and nausea. There was enlargement of liver, pretibial edema and a decrease in urine volume. At the beginning of April he had fever (102°F. to 104°F.) and hemoptysis. Ascites was also evident. He became progressively anemic and had hypoproteinemia (total serum protein 5.5 Gm./100ml.). In mid-May, he experienced episodes of acute pulmonary edema and severe pains over the right chest. On June 9, 1959, he died of congestive heart failure.
Autopsy: Pericardium revealed no abnormality. The heart weighed 580 Gm. and was greatly enlarged. The left ventricle was considerably and the right ventricle slightly hypertrophied. The right and left sinuses of Valsalva had undergone an aneurysmal enlargement, especially the former. The non-coronary sinus was normal. The aneurysm of the right coronary sinus ruptured into the left ventricle at the apex. The aortic valve was incompetent. The tricuspid, mitral and pulmonary valves were normal. Foramen ovale and ductus arteriosus were not patent. There was no septal defect. There was a thrombus in the right atrium. Histologic examination of wall of the sinus of Valsalva showed disappearances of elastic fibres, but it revealed no evidence suggesting a rheumatic or syphilitic endocarditis. At the aorta, there was no atheroma but mild mesoaortitis with a small aneurysm. There was a hemorrhagic pulmonary infarction with fibrinohemorrhagic pleurisy involving the middle lobe of the right lung. There were clear straw-colored pleural effusions. The right pleural cavity contained 1,200 ml. of fluid. At the left lower lobe, hypostatic pneumonia was observed. There were 400 ml. of clear yellowish ascites. The liver, spleen, kidneys and brain were congested. Marked peptic esophagitis was present and abscess of prostate and cystitis were also seen.

Discussion

Aneurysms of the sinuses of Valsalva were reviewed by Morgan Jones and Langley, Oram and East, Sawyers et al. and Dubost and his associates. It may be congenital or acquired. There is some difference of opinion as to the frequency of congenital aneurysm. Taussig stated that these aneurysms were most commonly due to lues. However, Oram and East in a recent review of 23 aneurysms of the aortic sinus, described that 4 were syphilitic, 3 were bacterial and the rest were congenital in origin.

Aneurysms may originate in one or more of the sinuses of Valsalva. An aneurysm of the right coronary sinus is most frequently encountered. An aneurysm of the non-coronary sinus is second in frequency. Even rarer are aneurysms of the left coronary sinus of Valsalva.

Depending upon the anatomic location of the aneurysm, it extends into the interventricular septum or the wall of the ventricles or atriums. Aneurysms of the right coronary sinus rupture usually into the right ventricle, occasionally into the right atrium and rarely into the left ventricle or the pulmonary artery. Aneurysms of the non-coronary sinus tend to perforate into the right atrium. Aneurysms of the left coronary sinus perforate into the left ventricle or the pericardial sac.

Rupture of the aneurysm can occur at any time of life and is usually provoked by physical strains. The most common site of rupture is into the right ventricle and right atrium, and less commonly left ventricle, pericardial sac and pulmonary artery. These aneurysms occur more frequently in men than in women. As an unruptured congenital aneurysm is usually asymptomatic, we have difficulty in diagnosing it during life. Rupture is occasionally accompanied by severe precordial or substernal pain and shortness of

breath. The most frequent clinical picture of aneurysm of the sinus of Valsalva is that of aortic insufficiency due to dilatation of the aortic ring. The commonest cause of death after rupture is the congestive heart failure. Rarely, death may result from subacute bacterial endocarditis and cardiac arrhythmia or heart block as a result of a disturbance of the conductive system. Sudden death has been also reported at the time of rupture of aneurysm. Mean survival time after the rupture was 3.9 years by Sawyers.

The case reported here represents aneurysms of the right coronary sinus and the left coronary sinus of Valsalva. The former bulged into the left ventricle and ruptured into the left ventricular cavity. A definite clinical diagnosis was not established during life in this patient.

As no pathologic evidence of syphilis or subacute bacterial endocarditis was found, a congenital aneurysm with spontaneous rupture is postulated, although aortitis (etiology unknown) can not be absolutely excluded. After the review of the patient’s clinical history, it seems probable that perforation occurred at the time, when the patient ran fast to get on a train on September 5, 1958. His death was no doubt the result of congestive heart failure with superimposed pulmonary infarction. Clinically, he represented the typical picture of aortic insufficiency.

**SUMMARY**

A patient is described in whom an aneurysm of the right coronary sinus of Valsalva had ruptured into the left ventricle. This is believed to be the second report of such a case as far as we know.
REFERENCES