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Surgical Correction of Ruptured Aneurysm of Aortic Sinus of Valsalva

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This is a report of 13 cases of surgically corrected ruptured aneurysm of the aortic sinus of Valsalva. The simultaneous trans-venous and trans-arterial catheterization and cineangiogram are the best diagnostic procedures. In 12 cases, the aneurysm originated from the right coronary sinus and only one from the noncoronary sinus. Eleven aneurysms ruptured into the right ventricle and 2 into the right atrium. Eight cases were associated with ventricular septal defect and 5 with aortic regurgitation. In three cases the Hancock porcine valve replacement was done. There was no surgical mortality.

Ruptured aneurysm of the aortic sinus of Valsalva has became a surgically correctable cardiac anomaly with the help of cardiopulmonary bypass, improved surgical technique and post-operative patient care. In 1831, Janes Hope first described an aneurysm of the aortic sinus of Valsalva originating from the right coronary cusp and rupturing into the right ventricle. Lillehei *et al.* (1957) reported the first sucessful surgical correction using cardiopulmonary bypass.

At Yonsei University College of Medicine, Severance Hospital during the period from 1964 to 1979, we have experienced 13 cases of this lesion and have done corrective surgery using extracorporeal cardiopulmnary bypass.

CASE MATERIALS

Ten adult males, one adult female and 2 pediatric females (M:F=10;3) have undergone corrective surgery for ruptured auerysm of aortic sinus of Valsalva. Seven patients (54%) were diagnosed as having ruptured aneurysm by aortogram preoperatively and three were suspected by cardiac catheterization. These 13 cases were studied and analysed in terms of clinical symptoms and signs, possible etiology, hemodynamics, surgical correction and prognosis.

Clinical Symptoms and Signs (Table I)

Dyspnea on exertion was the chief complaint in 11 patients and its duration ranged from one month to 25 years. One case (case no 11) had sudden onset of dyspnea with chest pain, dizziness and syncope. Four patients came in with congestive heart failure and case no. 11 was operated on because of intractable heart failure under emergency cardiopulmonary bypass. One patient experi-

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Table I. Clinical symptoms and signs

Case No.	Age	Sex	Chief Complain	Duration	B.P.	Type Murmur	Grade
1	M	20	Palpitation D.O.E.	(Childhood)	125/80	Systolic	III/IV
2	F	12	Chest discomfort, D.O.E.	(2 months)	115/90	Systolic	III/IV
3	M	25	Generalized edema D.O.E.	(3 years)	150/30	To & Fro.	III/IV
4	M	41	D.O.E.	(25 years)	140/40	To & Fro.	III/IV
5	M	27	D.O.E.	($1^1/_2$ years)	145/0	Continuous	III/IV
6	M	24	D.O.E.	(1 months)	140/130	Continuous	III/IV
7	M	30	Indigestion, D.O.E.	(4 months)	150/60	Continuous	III/IV
8	M	26	Generalized weakness	(1 year)	200/100	Continuous	III/IV
9	F	18	D.O.E.	(3 years)	110/90	To & Fro.	II/IV
10	M	25	Chest discomfort, D.O.E.	(5 years)	140/0	Continuous	IV/IV
11	M	17	Indigestion	(10 days)	150/40	End diastolic & ejection systolic	IV/IV
12	F	13	D.O.E. & Palpitation	(Childhood)	110/0	Systolic & diastolic To & Fro.	IV/IV
13	M	33	D.O.E.&Chest discomfort	(23 years) (10 days)	130/60	To & Fro.	III/IV

D.O.E.: Dyspnea On Exertion

Table II. Electrocardiogram, pre-operative and post-operative findings

Case No.	E.K.G.	Preop. Dx.	Operative Findings
1	Sinus tachy	P.S.	RCS to RV
2	LVH	VSD	RCS to RA
3	BRH. LAE, 1 HB	RAASV, ASD 2 AI, Rt coronary cusp Prolapse, VSD	RCS to RV, ASD, VSD(I),AI
4	LVH & PVC	RAASV	RCS to RV
5	LVH	RAASV, AI	RCS to RV, VSD(I)
6	LVH	VSD, AI	RCS to RV, VSD(I), AI
7	LAE	RAASV	PCS to RA
8	BAE & Bigeminy	RAASV	RCS to RV, VSD(I)
9	BVH	RAASV, VSD	RCS to RV, VSD(I)
10	LVH	RAASV	RCS to RV, VSD(I)
11	LVH & Myocardial ischemic change	VSD, AI	RCS to RV, VSD(I), AI
12	LVH	VSD, PS, AI	RCS to RV, VSD(I), AI
13	LVH with Strain	RAASV, VSD, AI	RAASV, VSD, AI

LVH : left ventricular hypertrophy, BVH : biventricular hypertrophy,
LAE : left atrial enlargement, BAE : biatrial enlargement, PVC : premature ventricular contraction,
P.S : pulmonic stenosis, VSD : ventricular septal defect, AI : aortic insufficiency,
ASD : atrial septal defect, RAASV : ruptured aneurysm of aortic sinus of Valsalva,
RCS : right coronary sinus, PCS : posterior coronary sinus, RV : right ventricle, RA : right atrium

Table III. Cardiac catheterization & retrograde aortogram (1)

Case No.	1	2	3	4	5	6	7
Sex/Age	M. 20	F. 12	M. 25	M. 41	M. 27	M. 23	M. 30
	02 Cont. Pr. (Vol.%) (mmHg)	02 Cont. Pr. (Vol.%) (mmHg)	02 Cont. Pr. (Vol.%) (mmHg)	02 Cont. Pr. (Vol.%) (mmHg)	02 Cont. Pr. (Vol.%) (mmHg)	02 Cont. Pr. (Vol.%) (mmHg)	02 Cont. Pr. (Vol.%) (mmHg)
SVC	13.55	14.00	10.25	12.00	10.56	12.48	14.70
RA (H)	14.26	13.70 2.3/−3	13.85	11.76	10.56	12.48	17.30
(M)	13.55 4/0		14.56 30/20(26)	22/8(14)	10.56	12.48	17.80
(L)	15.05	13.60	14.33	11.88	10.56		16.80
RV (I)	15.50 46/0(17)	14.40 22/−5	16.71	13.92	12.48	15.36	17.10
(M)		14.50	18.16	68/0/23	14.64 61/0/13	61/0/13	17.70 43/0/13
(O)	15.54 21/0(7)	14.20	17.44 112/0/29	16.32	15.84	15.36	
PA (Main)	15.54 26/7(11)	14.10 22/2.3		15.60 68/25(41)	15.60 61/22(40)	15.12 61/20(34)	17.80 43/18(27)
(Rt.)		14.50 14/2.3	17.91 112/41(58)	16.32 58/25(37)	15.36	15.60	17.80
(Lt.)							
Aorta	19.40	17.40	16.48 114/66(83)	18.24 130/60(95)	16.80 120/57(89)	18.36 116/59(92)	180/63(85)
Qp : Qs	1.16 : 1	1.16 : 1		2.78 : 1	4.30 : 1	1.92 : 1	3.40 : 1
Rp : Rs		79 : 1,240	473 : 1,382	245 : 2,240	150 : 1,820		85 : 1,226
Aortogram				Dye Pass AO-RV	Dye Pass AO-RV Cath. Passage	Dye Pass AO-RV, LV	Dye Pass AO-RA-RV

AO: Aorta, RA: Right Atrium, RV: Right Ventricle, LV: Left Ventricle

Table IV. Cardiac catheterization & retrograde aortogram (2)

Case No.	8		9	10	11	12	13
Sex/Age	M. 28	Postop. 5 mon.	F. 16	M. 24	M. 17	F. 13	M. 34
	02 Cont. Pr. (Vol.%) (mmHg)	02 Cont. Pr. (Vol.%) (mmHg)	02 Cont. Pr. (Vol.%) (mmHg)	02 Cont. Pr. (Vol.%) (mmHg)	02 Cont. Pr. (Vol.%) (mmHg)	02 Cont. Pr. (Vol.%) (mmHg)	02 Cont. Pr. (Vol.%) (mmHg)
SVC	13.69	13.55	14.21	14.64	6.63	9.85	13.41
RA (H)	13.32	14.18	14.61	14.12	6.04	10.05	14.34
(M)	13.51	14.30	14.58		7.37	10.59	13.31
(L)	13.41	14.12	14.94		7.89		13.14
RV (I)	15.38	13.77	15.49		7.22	9.51	13.78 92/4/12
(M)	16.79 64/0/15	13.77 41/0/5	15.30 31/0/2	18.90 57/0/13		10.96 87/17/28	
(O)		13.67	15.40	19.73		10.05	14.71
PA (Main)	64/19(26)	13.56 40/8(20)	15.40 19/5(10)	57/24(38)	11.42 87/44(62)	10.25 29/17(23)	15.04 39/11(24)
(Rt.)							
(Lt.)				18.90			
Aorta	17.92 156/54	17.18 162/75(101)	17.49 103/61(83)	19.94 101/56(78)	12.46 104/54(80)		125/56(88)
QP : QS	4 : 1				3 : 1		1.8 : 1
Rp : Rs		98 : 1228		99 : 1346	150 : 868	146 : 930	32 : 937
Aortogram	Ao-RV	Ao-LV (Grade II)	Ao-RV Aneurysmal Dilatation of RCS (Grade II-III AI)	Ao-RV	Unable to obtain because of ventricular tachycardia	AI (Grade IV)	Unable to obtain because of patient's poor condition

Ao : Aorta, RA : Right Atrium, RV : Right Ventricle, LV : Left Ventricle

enced acute chest pain one year prior to admission.

Eight patients had pulse pressure widening and one patient had a water hammer pulse. A continuous murmur was noticed in 9 patients. Radiologic examination showed cardiomegaly especially in cases with ventricular septal defect and increased pulmonary vasculatures in the plain chest film. In 8 patients the electrocardiogram showed left ventricular hypertrophy and one with strain pattern. Among these 8 patients with ventricular septal defect(Type I), 4 had associated aortic insufficiency. Biventricular hypertrophy was noticed in two patients. These two had ventricular septal defect and one of these had first degree heart block and left atrial enlargement(Table II). Case no 11 showed left ventricular hypertrophy with myocardial ischemia which was presumably due to the absence of the right coronary ostia.

Cardiac Catheterization(Table III and IV)

Oxygen step-up, more than 2 volume percent was noticed in 8 cases(3 cases in the right atrium, 5 cases in the right ventricle when combined with ventricular septal defects). Pulmonary arterial blood flow was increased definitely in 11 cases and 6 patients showed marked elevation of the pulmonary arterial systolic pressure(more than 50% of the systemic systolic pressure). The Qp:Qs ratio was measured in nine of our cases which ranged from 1.16 to 4.3:1.

With increased experience, we were able to make improvements in order to obtain better preoperative diagnoses. The method was that of simultaneous trans-venous and trans-arterial catheterization. After identification of two catheter tips in the right ventricle, with simultaneous pull back tracing, we obtained the aortic pressure directly from the right ventricle and right atrial pressure from right ventricle by venous catheter which facilitated the confirmation of ruptured aneurysm of aortic sinus of Valsalva (Fig I). Correct diagnosis of ruptured aneurysm of aortic sinus of Valsalva was confirmed by retrograde aortography preoperatively in 7 cases(54 %), showing a jet stream of contrast material passing from the ruptured anuerysm into the right atrium or ventricle.

Operative Findings(Table II)

In 12 patients(92.3%),the aneurysm originated from the right coronary sinus and one from the noncoronary sinus. In 11(84.6%)the aneurysm ruptured into the right ventricle and two into the right atrium.

Associated anomalies were present in 9 cases(69.2%). Eight cases were combined with type I ventricular septal defect, 5 cases with aortic insufficiency and one case with secundum type atrial septal defect. Eight cases with ventricular septal defect had aneurysms originating from the right coronary sinus and which ruptured into the right ventricle. One aneurysm from the right coronary sinus ruptured into the right atrium and an opening at the junction of the medial cusp of the tricuspid valve. In case no 4, the aneurysm ruptured into the right ventricle through the pulmonic valvular leaflet resulted in multiple perferations and pulmonary insufficiency. Case no 13 had two aneurysms measuring 4×1.5 cm and 1×1.5 cm, originated from the right coronary sinus and both ruptured into the right ventricle.

These patients had surgery under emergency cardiopulmonary bypass with moderate hypothermia. Through either right atriotomy or

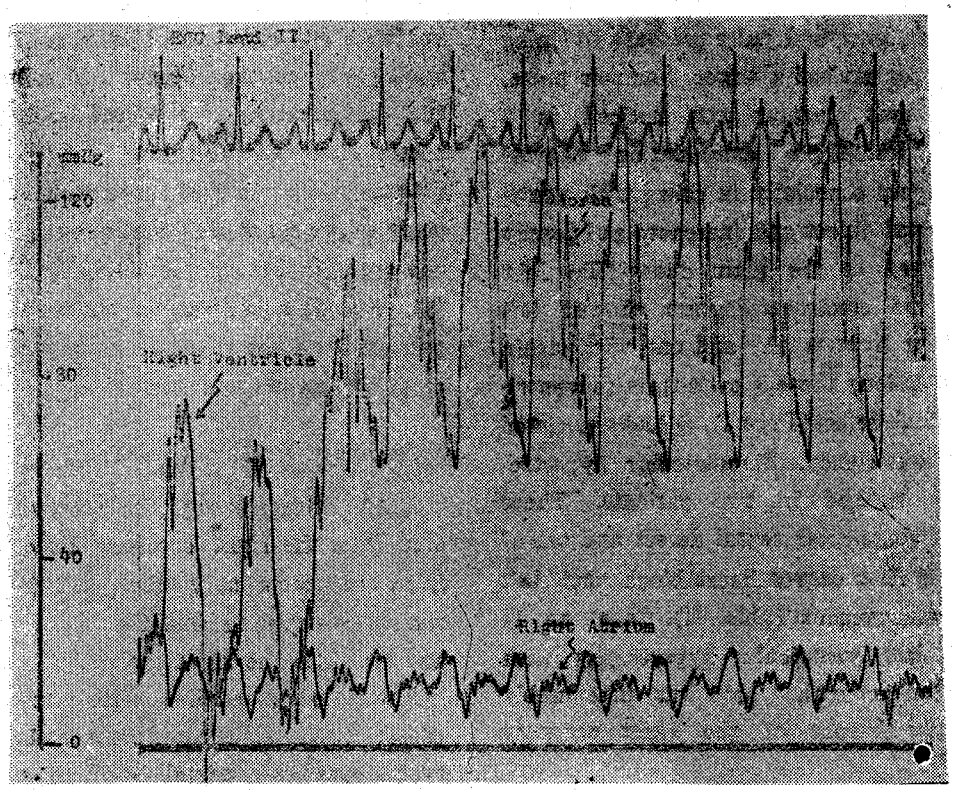

Fig. 1. Pressure Tracing.

ventriculotomy the aneurysm was resected at it's base. The free margin of the aneurysmal opening was closed directly using a Dacron pledget and interrupted mattress sutures. The ventricular septal defect was closed by direct suturing or a Teflon patch. In three cases with aortic insufficiency, the aneurysmal sac was resected first followed by aortic valvular replacement using the Hancock porcine valve.

Histologic examination failed to show any evidence of inflammation, such as bacterial endocarditis or syphilis.

RESULTS

There was no surgical mortality. One patient developed left hemiplegia probably due to cerebral embolism and was fully recovered by the 12th post-operative day. During the 4 months to 15 years follow-up, all patients have been in good condition except 3 with aortic regurgitation preoperatively and who still have a murmur (case no 5,6, and 8), and one has post-operative aortic insufficiency (Grade II).

DISCUSSION

Taguchi *et al*(1969) reported the incidence of aneurysm of the aortic sinus of valsalva is about 3.5% of the surgical cases of congenital disease. In our department it was 4.3%. Possible etiologic factors of this disease

are congenital, acquired(bacterial and fungal endocarditis, arteriosclerosis, syphilis, and trauma), collagen disease producing cystic medial necrosis, and osteogenesis imperfecta (Heppner *et al.*,1973). Many believe that the majority of these aortic aneurysms of the sinus Valsalva are probably due to either congenital or acquired anatomic defects in the elastic tissue between the base of the aorta and annulus fibrosus. With constant stress from the high pressure aortic pulsations the weakened aortic wall push into the low pressure cardiac chambers or adjacens structures(Howard *et al.*, 1973). No evidence of bacterial endocarditis, syphilis and collagen disease was observable in our series.

In the sex ratio of aneurysm of sinus of Valsalva males predominate by 1.98:1 according to Nowicki *et al.*(1977). The mean age of patients without symptoms is 20 years and with symptoms is 30. In our series, males were more than 70% and the mean age with symptoms was 23.9 years.

The clinical symptoms and signs depend on the existance of rupture, type of combined cardiac anomaly, and the site of aneurysm. Before rupture, most patients didn't have any symptoms other than that due to the combined cardiac anomaly if any. However, in some patients this aneurysm, especially when it originated from non-coronary sinus or the posterior part of the right coronary sinus, statistically produce atrioventricular conduction disturbances. It also can block the right ventricular outflow tract, as in our series case no 3, (Hong *et al.*,1966) and produce sysptoms which mimic pulmonary stenosis. After it ruptures, clinical symptoms depend on the time of rupture, the size of the opening and the presence of an associated cardiac anomaly. The patient may complain of acute onset of dyspnea, chest pain, dizziness, syncope and possibly rapid congestive heart failure. In our series, 77% of the patient had gradual onset of symptoms over months or years and 23% had a sudden onset. Dyspnea was the most common chief complaint(85%) and chest pain occured in 22% of the patients.

Most patients with aortic aneurysm of the sinus of Valsalva have the findings of collapse or bounding water hammer pulse, pulse pressure widening, precordial diastolic or systolic thrill and continuous murmur which can be best heard in the third or fourth intercostal space along the left sternal border. Nowicki *et al*(1977) observed, in review of 175 surgical cases, collapsing or bounding pulse in 82.55%, precordial thrill in 78.7% and continuous murmur in 93.4%. In terms of continuous murmur, it is loudest in midsystole in ruptured aneurysm of the sinus of Valsalva, instead of at the time of the second heart sound in patent ductus arteriosus. In our series, 9 patients(69%) had either a to and fro murmur or continuous murmur, 2 patients had only systolic murmur and two patients had different types of systolic and diastolic murmurs. We also observed in three patient with continuous murmur that there was more systolic than diastolic intensity and three that had equal intensity.

The systolic shunt flow being impaired by a vetnri effect in the aortic root and by a mechanical occlusive or kinking effect of the ventricular systol upon the fistula tract. An associated lesion may change the intensity of the systolic or diastolic component of the continuous murmur in aortic aneurysm rupture of sinus of Valsalva.

Electrocardiogram is not specific in diagnosis of ruptured aortic aneurysm of the sinus

of Valsalva. But in 80% of cases one may observe abnormalities such as left ventricular hypertrophy, biventricular hypertrophy, conduction distrubances and arrhythmia,

Chest X-ray findings are also not significant in diagnosis. Increased pulmonary vascular marking and cardiomegally can be observed.

By cardiac catheterization, significant oxygen step up(more than 2 vol%) and elevated pressure may be observed in the cardiac chamber which has been ruptured into. In some cases there is no pressure elevation even after aneurysm rupture, especially into right atrium. It may be related to increased compliance permitting this chamber to dilate and still maintain normal pressure(Minkoff *et al.*,1967). Oxygen step up also can be misleading because of stream effects or tricuspid regurgitation.

Correct diagnosis of ruptured aortic aneurysm of the sinus of Valsalva was confirmed by aortogram in 87.5% and in one case we were able to demonstrate by passage of the catheter the abnormal connection through the ruptured sinus of Valsalva.

The possible developmental explanation of this sinus Valsalva aneurysm is the anatomic or embryologic failure of fusion between the aortic wall media and the annulus fibrosus of the aortic value which perhaps occurs soon after fusion of the folds of the primitive bulbocordis and at which time the aortic root trunk begins to differentiate(Edwards and Burchell, 1957). This embryologic defect possibly explains the frequent association with ventricular septal defect. Taguchi *et al.* (1969) divided this congenital anomaly in to 6 main types and 16 subtypes depending on the origin and exact anatomic rupture site of the aneurysm, combination of ventricular septal defects and congenital aotric valvular anomaly.

In general, most of the aneurysms originated from the right coronary sinus and ruptured into either the right ventricle or atrium. We found an interesting difference between the Oriental and Caucasian. In Oriental reports, the aneurysm originated from the right coronary sinus in 85-95%, noncoronary sinus in 5-15% and the left in 1% while the European and North American report it from the right coronary sinus 25-28%, left in 7%.

The principles of surgical correction are 1) resection of aneurysm at its base 2) anatomic closure of the aneurysmal defect of the aortic sinus of Valsalva 3) correction of any associated cardiac anomalies such as ventricular septal defect or aortic regurgitation. Neither the atrial or ventricular approach revealed advantage because of the possible injury to coronary artery or aortic valvular cusp and failure placement of each suture to reunite the aortic wall media to the valve annulus(Nowicki *et al.*, 1977).

It appears that repair of the fistula itself is best carried out through an aortotomy and the cardiac chamber where the aneurysm extended after cross clamping with either topical hypothermia or coronary perfusion for protection of the myocardium. Nowicki *et al*(1977) reported surgical mortality of 12.7% and failure to close the fistula in 1.6%.

CONCLUSION

During the past 15 years at Yonsei University College of Medicine, Thoracic and Cardiovascular Division, we experienced 13 cases of ruptured aneurysm of the aortic sinus of Valsalva. In 12 cases, the anerysm originated from the right coronary sinus and only one

originated from the noncoronary sinus and ruptured into the right atrium. Eight cases were associated with ventricular septal defect and 5 cases with aortic regurgitation. Surgical correction of the ruptured aneurysm and associated anomaly were done under cardiopulmonary bypass. Three aortic valve replacements were done with porcine valve. There was no surgical mortality.

Long term follow up of 9 months to 14 years revealed all patients to be in good clinical condition.

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