Frequency of Angina Pectoris and Coronary Artery Disease in Severe Isolated Valvular Aortic Stenosis†

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Abstract

Angina pectoris is a frequent symptom of severe valvular aortic stenosis (AS), even in the presence of normal coronary arteries.

To determine the prevalence of angiographically significant coronary artery disease (CAD) and its relation to angina pectoris and coronary risk factors in severe isolated valvular AS patients.

All cases of symptomatic AS patients who underwent aortic valve replacement and pre-operative cardiac catheterization at the Central Chest Hospital between January 1,1986 and December 31, 1996 were retrospectively analyzed. Excluded were those with multiple valvular disease, aortic regurgitation of grade 2 or more, and prior coronary or valve surgery.

A total of ninety consecutive patients with severe AS (64 men and 26 women, mean age 58.94 years, range 38 to 71) were studied. Significant CAD (coronary diameter stenoses $\geq$ 50%) was found in 15 patients (16.7%). Typical angina was present in 66.7 per cent of them but it was also found in 46.7 per cent of the non-CAD patients. This symptom had low positive predictive value (22%). Of the patients without angina (n=45) 11.1 per cent had significant CAD, The negative predictive value of angina alone was thus 89 per cent. By univariate logistic regression, the statistically significant variables to discriminate those with or without significant CAD were age, history of hypertension, positive familial history of premature CAD, and cholesterol level. However, only age and hypertension were statistically significant by multivariate logistic regression analysis.

Coronary arteriography can probably be omitted in severe valvular AS, especially those without a history of hypertension and < 40 years of age in men and <50 years in women. For all other cases, coronary arteriography is recommended. In our study, angina pectoris is not a significant predictor for associated CAD.

Key word : Angina Pectoris, Coronary Artery Disease, Severe Isolated Valvular Aortic Stenosis

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Angina pectoris is a common symptom of both severe valvular aortic stenosis (AS) and coronary artery disease (CAD)[1-4]. In both conditions the angina is due to an imbalance between oxygen supply and demand[5-7]. The necessity of defining coronary artery anatomy before performing aortic valve replacement and planning for coronary artery bypass graftings has been recommended in most centers for patients with arteriographic evidence of significant obstruction in one or more graftable coronary arteries[8-13]. This recommendation is based on the observation that coronary artery disease may be an appreciable cause of morbidity and mortality after valve replacement[8-12]. Although such an approach may seem reasonable, no controlled studies clearly document the validity of this strategy, and the topic is still a source of debate[8-28]. Because of the small, but definite, hazard of coronary angiography, the risk to the patient of delaying an urgently needed operation and in the interest of cost containment we reviewed our experience at the Central Chest Hospital to determine whether the absence of a history of angina chest pain in patients with severe AS precluded significant coronary artery disease, thereby, obviating the need for coronary arteriography in such patients. A secondary purpose was to find out whether other atherosclerotic risk factors could be used as the statistically significant predicting factors for the presence of associated coronary artery disease.

METHOD

Selection of patients

All symptomatic aortic stenosis patients who underwent aortic valve replacement and preoperative cardiac catheterization at our institution from January 1, 1986 through January 31, 1997 were retrospectively reviewed. We excluded patients who had one or more of the following factors ; 1) multiple valvular disease; 2) peak systolic aortic valve gradient less than 50 mmHg by cardiac catheterization or Doppler echocardiographic study except for those who had severely impaired left ventricular systolic function (left ventricular ejection fraction less than or equal to 0.30); 3) aortic regurgitation defined by aortic root angiogram of grade 2 or more ; 4) other congenital heart disease ; 5) prior coronary or valve surgery. Ninety consecutive patients were finally selected for the study.

Clinical data

The medical records of all patients with severe AS were retrospectively reviewed to determine the presence and characteristics of angina pectoris, history of orthopnea, paroxysmal nocturnal dyspnea (PND), dizziness, syncopal attacks, palpitations, and peripheral vascular disease (PVD). The other associated coronary risk factors were also evaluated : smoking habits, personal history of hypertension of diabetes mellitus, prior myocardial infarction, familial history of CAD, hyperlipidemia, and the use of diuretics or digitalis. Typical angina pectoris was defined as a classic history of chest pain or tightness lasting for 1 to 15 minutes precipitated by effort, emotion, or exposure to cold, which was promptly relieved by rest or nitrates. Cases with nocturnal angina and typical angina at rest were also included. Atypical or undefined chest pain was characterized by a history of chest pain not fulfilling the requirements for a diagnosis of angina pectoris because of location, quality, or precipitating and relieving events. The preoperative severity of angina pectoris was graded according to the Canadian classification and the preoperative functional status was classified symptomatically according to the New York Heart Association (NYHA) functional class [29]. The history of prior myocardial infarction was diagnosed in patients who had a history of chest pain associated with electrocardiographic (ECG) or cardiac enzyme changes that were consistent with this diagnosis. The history of hypertension was diagnosed in patients who had had a medical documentation of either systolic blood pressure (SBP) $\geq$140 mmHg or diastolic blood pressure (DBP) $\geq$90 mmHg for at least 3 years perior study Preoperative chest radiographs were available for all patients. Cardiac enlargement was defined as a cardiothoracic ratio greater than 50 per cent in the standard posteroanterior projection. Preoperative electrocardiograms were also studied in all patients. Included in the data base were preoperative presence of pathological Q-waves, evidence of left ventricular hypertrophy (Estes' Scoring system)[30], ST-T changes that were consistent with classic systolic strain pattern (without digitalis), and the presence of atrial fibrillation.

Cardiac catheterization

Right and left heart catheterization, left ventriculography, aortography and selective coronary angiography were performed in all 90 patients.

During selective coronary angiography, multiple left and right anterior oblique projections were obtained in an attempt to provide optimal display of the arteries. The degree of coronary artery narrowing was expressed as the percent reduction of the internal diameter of the artery under study. Coronary artery disease was considered hemodynamically significant if one or more coronary arteries had a 50 per cent or greater estimated narrowing of lumen diameter. Stenoses of branch vessels supplying more than 50 per cent of the distribution of a major vessel were classified as stenosis of that major vessel.

Left ventricular contraction was evaluated by analysis of an angiogram of the left ventricle performed in the 30 degree right anterior oblique (31). Cardiac output was determined by the dye dilution technique and by the Fick method.

The pressures were recorded with a fluid-filled manometric system, the zero reference point being the mid-chest level. A pullback pressure tracing was obtained to confirm the location of the obstruction at the level of the aortic valve and to determine the gradient across the aortic valve. The diagnosis of severe aortic stenosis was based on the measurement of peak systolic aortic valve gradient of 50 mmHg or greater in 57 patients and on angiographic, echocardiographic and clinical criteria in 33. The degree of aortic regurgitation was assessed by root aortography in the left anterior oblique projection(32). Aortic regurgitation was graded as trivial (1+), mild (2+), moderate (3+), and severe (4+). Trivial regurgitation was defined as a small amount of dye entering the left ventricle during diastole and washing out during systole. Only those with trivial regurgition were included in the study. Valve calcifications were evaluated by fluoroscopic examinations.

The aortic valve area was calculated by means of the Gorlin's formula(33). Aortic stenosis was considered hemodynamically significant if the calculated aortic valve area was less than 0.8 cm^2 per square meter of body surface area.

During the period under review, selective coronary arteriography was routinely performed before aortic valve replacement. Coronary bypass graftings were undertaken if significant stenosis presented angiographically and when, in the surgeon's opinion, revascularization was technically possible.

Statistical methods and analysis

Preoperative clinical and differences between patients with and without coexistent coronary artery disease were analyzed by using chi-square analysis (with Yates correction) for frequency distribution and Fisher exact tests for categorical variables and with student *t* tests for quantitative variables(34). Multivariate analysis using a stepwise logistic regression procedure was then applied to further test varibles significantly related to CAD to derive a prediction model. A multivariate risk score was also calculated to stratify patients into groups with or without associated CAD. Standard methods were used to derive sensitivity, specificity and the predictive values of a positive and negative test result(35).

Prediction of coronary artery disease

The sensitivity of angina pectoris to indicate the presence of CAD in the current study was 66.7 per cent (10 of 15), but its specificity was only 53.3 per cent (35 of 40). The positive predictive value of this symptom alone was 22 per cent (10 of 45) and its negative predictive value 88.9 per cent (40 of 45).

By univariate logistic regression analysis, the statistically significant variables to discriminate those with or without significant CAD were age, history of hypertension, positive familial history of premature CAD, and cholesterol level. But only age and hypertension were of statistical significance by multivariate logistic regession analysis.

RESULTS

Clinical characteristics

A total of 90 consecutive patients with severe isolated valvular aortic stenosis was studied. Forty-five patients (50%) had angina pectoris. Of these, 35 had no significant CAD. In the CAD group, 1 had atypical chest pain and 4 did not have any chest pain. Similar grades of angina were observed in both groups, with 17 per cent of patients having grade 1, 56 per cent grade 2, 21 per cent grade 3, and 6 per cent grade 4. CAD patients were significantly older (64.7 *vs* 57.8 years; $p < 0.02$), and had somewhat greater frequency in the male sex but no statistical significance. They also had a higher total serum cholesterol level (Table 1). Of the patients with CAD, all men were $\geq$ 45 years of age and all women were $\geq$ 55. Of the non-CAD group,

Table 1. Clinical characteristics of 90 patients with severe AS.

Variables	No. patients with CAD (n = 15)	No. patients without CAD (n = 75)	p - value
Age(years)	64.7 ± 6.6	57.8 ± 10.6	< 0.02
Sex (M/F)	10/5	54/21	NS
Mean duration (mths)	32.4 ± 35.3	30.2 ± 29.0	NS
Typical angina(%)	10 (66.7)	35 (46.7)	NS
Atypical chest pain (%)	1 (6.7)	13 (17.3)	NS
Orthopnea (%)	5 (33.3)	33 (44.0)	NS
PND (%)	3 (20.0)	36 (48.0)	< 0.05
Syncope (%)	2 (13.3)	19 (25.3)	NS
Dizziness (%)	6 (40.0)	22 (29.3)	NS
Cerebrovascular disease (%)	1 (6.7)	0 (0)	0.02
Peripheral vascular disease (%)	1 (6.7)	0 (0)	0.02
Palpitation (%)	0 (0)	11 (14.7)	NS
Diabetes mellitus (%)	2 (13.3)	4 (5.3)	NS
Hypertension (%)	10 (66.7)	6 (8.0)	< 0.00001
Smokers (%)	8 (53.3)	37 (49.3)	NS
Ex-smokers >5 years (%)	1 (6.7)	6 (8.0)	NS
History of MI (%)	1 (6.7)	1 (1.3)	NS
Familial history (%)	5 (33.3)	9 (12.0)	< 0.04
Dyspnea on exertion			
NYHA I, II	9 (60.0)	34 (45.3)	NS
NYHA III, IV	4 (40.0)	41 (54.7)	NS
Cholesterol (mg%)	236.4 ± 36.0	211.8 ± 36.9	0.02
Use of diuretics (%)	11 (73.3)	60 (80.0)	NS

(*Abbreviations : CAD = coronary artery disease; MI = myocardial infarction;
NYHA = New York Heart Association classification (Classes I-IV);
± = one standard deviation ; n = number of patients)

21 were < 45 years of age, with 6 men and 5 women being < 40 and 50 years old, respectively. Paroxysmal nocturnal dyspnea (PND) occurred more in non-CAD (48%) than CAD patients (20%) ($p < 0.05$). The prevalence of peripheral vascular disease (PVD), and cerebrovascular disease(CVD) were significantly more common in the CAD group ($p < 0.02$). The CAD patients also had a more frequent history of hypertension ($p < 0.00001$) and positive familial history of premature CAD ($p < 0.04$). The distribution of other clinical findings is given in Table 1.

The electrocardiographic (ECG) analysis revealed the presence of left ventricular hypertrophy (LVH) and ST-T changes consistent with a systolic strain pattern with nearly the same frequency in both groups. The ECG evidence of pathological "Q" waves as noted at the time of preoperative angiography was present in 19 patients. Of these, 14 had normal coronary arteries. All of them had "QS" patterns in leads V_1, V_2 and not beyond Leads V_3. Infarction patterns represented by pathological "Q" waves in Leads V_4 - V_6 or II III, and aVF were, on the other hand, associated with CAD in 4 of 7 instances. A history of acute myocardial infarction was recorded in the past in 2 of them.

Complete right bundle branch block was present in one patient, aged 68 years with normal coronary arteries. The ECG evidence of atrial fibrillation was present in 8 patients. Of these, 1 had associated CAD which involved 3-vessel and left main stem. The distribution of electrocardiographic, radiographic and hemodynamic data is demonstrated in Table 2.

Catheterization and coronary angiographic findings

The distribution of coronary arteriographic findings in the CAD patients, with or without

Table 2. Electrocardiographic, radiographic and hemodynamic data in 90 patients with severe AS.

Variables	No. patients with CAD (n = 15)	No. patients without CAD (n = 75)	p - value
ECG			
LVH (%)	13 (86.7)	64 (85.3)	NS
ST-T strain (%)	12 (80.0)	62 (82.7)	NS
Pathological-Q (%)	5 (33.3)	14 (18.7)	NS
AF (%)	1 (6.7)	7 (9.3)	NS
CXR			
Redistribution (%)	6 (40.0)	24 (32.0)	NS
CT-ratio >- 0.5 (%)	4 (26.7)	29 (38.7)	NS
Calcified AV (%)	13 (86.7)	68 (90.7)	NS
Hemodynamic			
Mean LVEF	0.64 ± 0.08	0.59 ± 0.02	NS
Mean gradient (mmHg)	91.0 ± 20.2	95.2 ± 28.3	NS
AVA (cm^2/M^2)	0.59 ± 0.16	0.61 ± 0.29	NS

Table 3. The distribution of significant coronary occlusive lesions in 15 CAD patients and subclassification of those with or without angina.

CAD occlusive lesion	No. patients (n) overall (n = 15)	with angina (n = 10)	without angina (n = 5)
I Single lesion			
- LAD*	2	0	2
- LCX	0	0	0
- RCA	0	0	0
II Double lesions			
- LAD + LM	1	1	0
- LAD + LCX	1	1	0
- LCX + RCA	1	0	1
III Triple and quadruple lesions			
- LAD + LCX + RCA	2	2	0
- LAD + LCX + RCA + LM	8	6	2

* LAD = left anterior descending coronary artery :
LCX = left circumflex coronary artery;
RCA = right coronary artery;
LM = left main coronary artery

angina, is shown in Table 3. The left anterior descending artery was found to be the most frequently involved vessel. Combinations involving two-or three- major vessels were demonstrated more frequently than single- vessel involvement. Significant stenosis of the left main coronary artery occurred in 9 patients (60% of those with CAD and 10% of all cases) ; all but one had multivessels disease. Of these, 2 had no history of chest pain at all. Calcification of the left main coronary artery was demonstrated in 16 patients, of these,only 2 showed significant stenosis of the left main itself, but 3 had significant lesions elsewhere in the proximal coronary arteries. Minor disease (irregularity of vessel wall or luminal diameter reduction less than 50%) was also found more frequently in the left anterior descending artery than in the non-CAD group.

On fluoroscopic examination, calcification of the aortic valve was noted in 93 per cent and 89 per cent in the CAD and non-CAD patients, respectively. There were no significant differences in the peak systolic gradient across the aortic valve, aortic valve area, and cardiac output between the two groups (Tabel 2).

DISCUSSION

Angina pectoris has long been recognized as as one of the cardinal symptoms of severe AS even in patients without evidence of hemodynamically significant CAD. There has also been wide variation in the reported prevalence of this symptom in patients with significant AS, ranging from 49-80 per cent, with an average of 66 per cent in the studies listed in Table 4[8,11,12,14-26]. In this study, the prevalence of angina was 50 per cent. Similarly, the prevalence of associated CAD in AS patients has been reported to be within a wide range between 21.0 and 56.0 per cent, compared with 16.7 per cent in this data. Between 24.0 and 80.0 per cent (with an average of 51.3 per cent in Table 4) of the patients had angina pectoris despite normal or only slightly narrowed (diameter reduction less than 50%) coronary vessels, compared with 46.7 per cent in our study. This wide variation in the prevalence of angina and CAD in patients with severe AS may be a result of differences in the age of the patients studied, and the variable criteria used by different investigators in defining significant coronary artery stenosis.

Some investigators[11,13,15,17,18] have used a criterion for luminal diameter reduction of at least 70 to 75 per cent, whereas others[8,12,16] have used as significant a lumen narrowing of $\geq$ 50 per cent. It is generally accepted that a luminal diameter reduction of 70 to 75 per cent is probably a prerequisite to cause hemodynamically significant abnormalities in the coronary circulation. We, neverthe-

Table 4. Prevalence of angina pectoris and significant coronary artery disease in patients with severe aortic valve stenosis.

Study	Pts (n)	Age Range (yrs)	Series	Prevalence (%)		Angina (%)		CAD (%) Patients without AP
				CAD	AP	CAD	Non-CAD	
Basta et al (1975)	68	34-77	CS	24	60	-	-	0
Harris et al (1975)	69	38-77	CT	23	58	81	51	10
Mandal et al (1976)	60	45-66	CF	23	47	67	41	>6
Paquay et al (1976)	48	39-84	C	44	75	95	59	8
Moraski (1976)	65	31-81	CS	46	80	80	80	46
Hancock (1977)	173	40-83	NC	56	74	85	61	33
Swanton et al (1977)	140	18-75	CS	23	56	72	52	15
Graboys et al (1977)	19	47-76	CF	21	63	100	53	0
Hakki et al (1980)	39	43-79	CF	46	51	83	24	16
Exadactylos et al (1984)	88	38-77	CT	34	49	86	35	7
Monsuez et al (1984)	148	20-81	C	18	55	70	51	7
Green et al (1985)	103	44-87	CF	45	61	78	47	25
Abdulali et al (1985)	32	32-72	C	34	63	82	52	17
Ann Vandeplas (1987)	192	28-82	C	24	66	83	61	12
Present study (1996)	90	38-71	C	16.7	50	66.7	46.7	5.6

AP = angina pectoris ; CAD = coronary artery disease ; C = consecutive ;
CT = consecutive, age limit 35 years ; CF = age limit 40 ; CS = consecutive, surgical ; NC = not consecutive.

less, agree with previous authors[8,12,16,21] that in patients with severe AS a luminal narrowing even of $\geq$ 50 per cent can cause significant reduction of coronary blood flow with regards to increasing myocardial oxygen demand.

The mechanisms responsible for angina symptoms in patients with AS have been explained on the basis of an imbalance between myocardial oxygen supply and demand[1,5,36-39]. The apparent pathophysiological consequence of left ventricular outflow obstruction, such as severe AS, has been well known to increase intraventricular pressure, increase wall tension[7,37,38], and finally result in myocardial hypertrophy[39]. The thicker wall reduces its stress, but the greater muscle mass inevitably increases myocardial oxygen demand. Several investigators[35,38-53] have also suggested abnormal small vessel behavior associated with severe left ventricular hypertrophy, that of impairing coronary vasodilator reserve, or impairing ability to increase blood flow during maximal vasodilatation response to catecholamine administration stress. Severe AS itself also reduces myocardial oxygen supply. A fixed coronary flow and decreased myocardial oxygen supply may result from the fixed stenotic valve orifice and the low mean aortic pressure. The increased left ventricular end diastolic pressure (LVEDP), or left ventricular wall tension [36,37] and the abnormal flow patterns of a Venturi effect in the aortic root[52] may further reduce coronary perfusion in these patients.In addition, the prolonged ejection time, which, if combined with the tachycardia of exercise, may have a deteriorating effect by decreasing diastolic filling period and also oxygen supply to the myocardium.

These are among the main several factors accounting for angina in severe AS without associated CAD. The presence of significant coronary artery stenosis in these patients is an additional, critically important single factor, to lower still further the myocardial oxygen supply. A coronary embolism from calcified fragments of the aortic valve may, furthermore, be a possible cause of coronary obstruction.

The prevalence of coronary artery disease in our patients with typical angina pectoris was 66.7 per cent, in contrast to patients with atypical chest pain with only a 6.7 per cent rate of associated CAD. These findings agree with those of Paquay and associates[15] that in patients with aortic valve disease, the more typical the angina the more likely these patients are found to have associated CAD. In view of the relationship of angina and associated dyspnea, Hancock[8] commented that angina occur-

ring on exertion in the absence of dyspnea, or occurring at rest, was associated with coronary artery disease in 80 per cent of instances, whereas, angina occurring only in association with dyspnea on exertion was associated with CAD in 45.0 per cent of instances. In our study, however, we did not find this relationship probably because of the small number of patients.

On the other hand, another important group of severe AS patiens are those with significant CAD but without angina pectoris. The reported incidence of unsuspected CAD patients in the absence of angina symptoms has also varied widely between a low of zero up to 46.0 per cent[8,11,12,14-26] (with an average of 15 per cent from all the studies in Table 4), compared with 5.6 per cent (5 of 90) in the current investigation. Of these, only 1 had diabetes mellitus, so that the "silent ischemia" could not be simply explained by the process of diabetic autonomic neuropathy. It is of interest also to note that 60 per cent (3 of 5) had clinical evidence of congestive heart failure as the principal presenting problem, especially 2 with triple-vessel disease plus significant left main stenosis. This finding agrees with that of Hancock[8] that patients with severe valvular AS who presented with congestive heart failure appear to be particularly likely to show significant CAD without angina. This group of patients may be similar to those with ischemic cardiomyopathy in the absence of aortic valve disease, as described by Burch and associates[53]. This may simply reflect the general clinical observation that angina pectoris tends to be absent when congestive heart failure develops[54,55], perhaps because physical activity becomes limited by exertional dyspnea. Thus, the absence of angina alone cannot be used as a significant predictor to exclude associated CAD.

Similarly with previous investigators[5, 56-58], we found one CAD patient without angina in whom effort syncope was the only complaint and whose left ventricular systolic function was very well preserved. The prevalence of syncope tended to be lower in the CAD group but was not statistically significant. On the other hand, the congestive symptom of PND was found more frequently in the non-CAD group ($p < 0.05$), which has also been observed by previous authors[19,20]. A clear explanation for this difference could not be found because the cardiac output and the use of diuretics or digitalis were not statistically different between CAD and non-CAD patients.

Similarly like other investigators[15,17, 26], we found no significant differences in mean aortic valve area, systolic gradient or left ventricular ejection fraction between the two groups. There was no mean systolic gradient that excluded the presence of significant CAD. However, patients with angina and CAD tended to have a lower gradient than those with angina without CAD. This has also been found by other investigators[19,20,26].

The distribution of coronary occlusive lesions in our patient population is shown in Table 3. The CAD patients with angina tended to have a higher incidence of multivessel disease and more severe disease of involved coronary vessels than those with CAD in the absence of angina, but this difference was not statistically significant. It is of interest also to note that two of the 5 CAD patients but without angina had evidence of significant left main stenosis. Of these, one had a history of diabetes mellitus, and both had a history of long standing hypertension. Over all, the left anterior descending artery was the vessel most frequently involved, and combinations involving two or three major vessels were more frequent than single-vessel involvement, as was observed by Hancock[8]. However, the findings of a low incidence of multivessel disease were also reported by other investigators[11,21,26]. There was a significantly higher prevalence of both cerebrovascular disease and peripheral vascular disease in CAD patients than in those without CAD. This may probably be explained on the basis of an atherosclerotic role in both coronary and peripheral vessels. The familial history of premature CAD was also found more commonly in the CAD group ($p < 0.04$). Serum cholesterol level was , as could be expected, significantly higher in those with CAD. However, only older age and history of hypertension were statistically significant by multivariate logistic regression analysis.

Serious complications of cardiac catheterization and coronary angiography in our study included one death due to ventricular fibrillation, and another 3 who developed early morbidity including acute pulmonary edema, cerebral embolic stroke and acute renal failure post ventriculography.

Clinical implications :

Knowledge of the extent of CAD involvement in patients with severe AS is clearly of predictive value with regard to the operative morbidity, mortality, and prognostic standpoints. Different

guidelines are therefore advocated to delineate which patients with symptomatic AS should have coronary arteriography performed preoperatively. Some suggest this procedure for all cases prior to surgery[20,23,25], whereas, others advocate only for those over age 40[18,19,21] or even age 50 years[9]. Our data are in more agreement with the latter. Based on the findings of our study, coronary arteriography can probably be omitted in severe valvular AS, when they do not have history of hypertension and age < 40 years for men and < 50 years for women. Otherwise, this diagnostic procedure should be recommended. In our study, angina pectoris is not a significant predictor for associated CAD.

Limitations :

Our study is in large part retrospective and therefore, is affected by the problems inherent in that approach. It should also be pointed out that our study population is not a very large one, making it more difficult to draw accurate conclusions. However, to overcome this limitation, we should co-investigate with other institutions and re-analyse a larger number of the patient population.

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ความบ่อยของอาการเจ็บเค้นหน้าอกแบบแองไจนาและภาวะหลอดเลือดหัวใจตีบในผู้ป่วยลิ้นหัวใจเอออร์ติคตีบชนิดรุนแรง†

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อาการเจ็บเค้นหน้าอกแบบแองไจนา เป็นอาการที่พบได้บ่อยในผู้ป่วยลิ้นหัวใจเอออร์ติคตีบชนิดรุนแรง แม้ในผู้ป่วยที่ตรวจพบว่าหลอดเลือดหัวใจปกติ

เพื่อประเมินความชุกของภาวะหลอดเลือดหัวใจตีบ (วินิจฉัยจากการตรวจฉีดสีสวนหัวใจ) ในผู้ป่วยลิ้นหัวใจเอออร์ติคตีบชนิดรุนแรง พร้อมทั้งหาความสัมพันธ์ของภาวะหลอดเลือดหัวใจตีบนี้กับอาการเจ็บเค้นหน้าอกชนิดแองไจนาและปัจจัยเสี่ยงอื่นๆ ของการเกิดภาวะหลอดเลือดหัวใจตีบ

ได้ทำการศึกษาย้อนหลังผู้ป่วยลิ้นหัวใจเอออร์ติคตีบชนิดรุนแรงทุกราย ที่ได้รับการตรวจฉีดสีสวนหัวใจก่อนการผ่าตัดเปลี่ยนลิ้นเอออร์ติค ณ โรงพยาบาลโรคทรวงอก ระหว่างวันที่ 1 มกราคม 2529 ถึง วันที่ 31 ธันวาคม 2539 โดยได้คัดผู้ป่วยที่มีปัจจัยข้อใดข้อหนึ่งต่อไปนี้ออกจากการศึกษา ได้แก่ มีความพิการของลิ้นหัวใจอื่นๆ ร่วมด้วย ลิ้นเอออร์ติครั่วมากกว่าหรือเท่ากับเกรด 2 ขึ้นไป หรือเคยได้รับการผ่าตัดหลอดเลือดหัวใจหรือลิ้นหัวใจมาก่อน

ได้ทำการศึกษาผู้ป่วยลิ้นหัวใจเอออร์ติคตีบทั้งสิ้น 90 ราย (ชาย 64, หญิง 26 คน, อายุระหว่าง 38–71 ปี อายุเฉลี่ย 58.94 ปี) พบภาวะหลอดเลือดหัวใจตีบ (เส้นผ่าศูนย์กลางหลอดเลือดตีบ $\geq$ 50%) ในผู้ป่วย 15 ราย (16.7%) โดยพบอาการเจ็บเค้นหน้าอกแบบแองไจนา 66.7% ขณะที่อาการเจ็บเค้นหน้าอกนี้ยังสามารถพบได้ถึง 46.7% ของผู้ป่วยหลอดเลือดหัวใจปกติด้วยเช่นกัน ทำให้อาการนี้มีค่าบ่งชี้ในทางบวกต่ำ คือ เพียง 22% สำหรับกลุ่มผู้ป่วยที่ไม่มีอาการเจ็บเค้นหน้าอก (45 คน) พบว่า 11.1% มีภาวะหลอดเลือดหัวใจตีบ ดังนั้นอาการนี้จึงมีค่าบ่งชี้ในทางลบ 89% อาศัยวิธี Univariate analysis พบว่าตัวแปรสำคัญที่ใช้แยกกลุ่มผู้ป่วยที่มีหรือไม่มีภาวะหลอดเลือดหัวใจตีบ ได้แก่ อายุ ประวัติความดันโลหิตสูง ประวัติบุคคลในครอบครัวเป็นโรคหลอดเลือดหัวใจตีบก่อนวัยอันควร และระดับโคเลสเตอรอลในเลือดสูง อย่างไรก็ตาม เฉพาะอายุและประวัติความดันโลหิตสูง เท่านั้น ที่เป็นตัวแปรสำคัญเมื่อใช้วิธี Multivariate analysis

การตรวจฉีดสีสวนหัวใจในผู้ป่วยลิ้นเอออร์ติคตีบรุนแรงนั้น อาจจะไม่จำเป็นต้องทำในผู้ป่วยชายที่อายุน้อยกว่า 40 ปี หรือผู้ป่วยหญิงอายุน้อยกว่า 50 ปี และไม่มีประวัติของความดันโลหิตสูงมาก่อน นอกเหนือจากนี้แล้ว ควรจะต้องทำการศึกษาหลอดเลือดหัวใจด้วยการฉีดสีสวนหัวใจทุกราย จากการศึกษานี้ อาการเจ็บเค้นหน้าอกแบบแองไจนาไม่สามารถนำมาเป็นตัวแปรบอกถึงภาวะหลอดเลือดหัวใจตีบได้

คำสำคัญ : อาการเจ็บเค้นหน้าอกแบบแองไจนา, ภาวะหลอดเลือดหัวใจตีบ, ผู้ป่วยลิ้นหัวใจเอออร์ติคตีบชนิดรุนแรง

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