Clinical Medicine and Health Research Journal (CMHRJ)
Volume 04, Issue 02, March - April, 2024
Page No. 887-892,

Research Article

Operative Morbidity and Mortality of Aortic Stenosis with Left Ventricular Dysfunction

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Received: 10 January, 2023 Accepted: 10 April, 2024 Published: 28 April 2024

Abstract:

Objectives: Study of the operative morbidity and mortality of aortic stenosis with left ventricular systolic dysfunction (LVEF < or = 35%).

Method: 73 patients with a mean age of 55+/-8 years, 59 of whom were men, underwent surgery between January 2014 and december 2019; 14% (10/73) of these patients had a pre-operative low trans-valvular aortic gradient (<30mmHg).

We determined the factors predictive of operative mortality using univariate and multivariate analyses, analysed changes in functional status and left ventricular ejection fraction, and compared operative mortality and late survival in these 73 patients with those in a control group with no left ventricular dysfunction.

Results: The mean NYHA stage, before and after aortic valve replacement, fell from 3.8 to 1.7, and the mean improvement in LVEF was 28+/-13.

In univariate analysis, the parameters significantly associated with operative mortality were arterial hypertension (p=0.039), congestive heart failure (p=0.030), a smaller indexed left ventricular mass (p=0.039), a mean gradient of less than 30mmHg (at the limit of significance (p=0.053)), a low cardiac index (p=0.0045), and mono-truncular coronary involvement (p=0.042).

In multivariate analysis, no variable was identified as an independent predictor of operative mortality.

Conclusion: Operative mortality was significantly higher 11.7%vs 2.9% (p=0.048) in the group with left ventricular dysfunction, although late survival rates were similar in the 2 groups (86.8% vs 93%).

Given the spontaneous prognosis and the increased operative risk, these patients should be operated on.

Keywords: **aortic stenosis, LV dysfunction, surgery, mortality.**

Introduction:

Left ventricular diastolic dysfunction appears early in the natural history of aortic stenosis; according to Hess et al (1), it affects 50% of patients with aortic stenosis with normal systolic left ventricular function, and 100% of patients with impaired systolic left ventricular function. This diastolic dysfunction alone may be responsible for the signs of heart failure (2,3); its pathogenesis is multiple: hypertrophy, increased stiffness and impaired relaxation of the left ventricle.

Left ventricular systolic dysfunction occurs late in the course of aortic stenosis; different mechanisms may be responsible for this impairment of systolic function: after-load mismatch, intrinsic impairment of left ventricular contractility, and coronary insufficiency. The aim of this prospective study was to evaluate the operative morbidity and mortality of aortic stenosis with left ventricular dysfunction, in order to determine whether our surgical indications were valid.

We identified factors predictive of operative mortality, analysed changes in functional status and left ventricular ejection frac-tion, and compared operative mortality and late survival of patients with left ventricular dysfunction with those of a control group without left ventricular dysfunction.

Material and Methods:

Between January 2014 and december 2019, 73 consecutive patients (group 1) operated for narrow aortic stenosis (S<0.5cm²/m²) complicated by left ventricular systolic dysfunction (LVEF<or=35%) were included in our study.

This group was compared with a population of 73 patients (group 2) operated on for narrow aortic stenosis without left ventricular systolic dysfunction (LVEF>or=50%) matched for age, sex, type of prosthesis, size of prosthesis, year of operation and coronary involvement.

In terms of aetiology, our study identified 79% valvular sequelae of rheumatic fever, 12% bicuspidia, 09% degenerative aortic stenosis and 06.45% bacterial endocarditis (01.45% acute, 5% sub-acute).

Exclusion criteria were severe aortic insufficiency grade [III or IV], significant mitral insufficiency grade III or IV with intrinsic mitral valve damage, and associated mitral surgery excluding mitral annuloplasty for functional mitral insufficiency. Neither coronary disease nor age were exclusion criteria.

With regard to statistical analysis, the results are expressed as mean +/- standard deviation; each pre- and post-operative variable was tested in univariate analysis using either a non-paired Student's test for quantitative variables, or a << chi² >>

test for qualitative variables; significant variables (p<or=0.05) were included in a multivariate analysis using the Cox model for survival (total and late mortality), and a logistic regression model for operative mortality. Survival curves were established using the Kaplan-Meyer method, and were compared using the log-rank test; the significance threshold was set at p<0.05.

I/ Operative mortality:

10/146(7%) of the operated patients died during the operation: 08 (11%) in group 1 and 02(3%) in group 2 (p=0.049). Analysis of the causes of death reveals that:

- **in group 1:** 02 patients who had pre-operative cardiac insufficiency <DTDVG>58mm,DTSVG>48mm) with rhythm disorders died of refractory low flow without being able to be weaned from the CEC;04 died of cardiac failure immediately post-operatively (including 01 following a myocardial infarction); the last 02 patients died of septic shock due to bronchopulmonary infection.
- **in group 2:** the first patient died of refractory low flow at the end of bypass surgery, the second patient died in the intensive care unit of neurological complications (massive haemorrhagic stroke).

Operative mortality is significantly related to immediate peri-operative complications: the occurrence of haemodynamic instability at the end of extracorporeal circulation requiring the use of positive inotropic drugs, the impossibility of weaning the patient from the bypass, the occurrence of intra-operative refractory heart failure, myocardial infarction, complete atrioventricular block, and post-operative heart failure. On the other hand, the occurrence of cerebrovascular accident

In contrast, the occurrence of cerebrovascular accident, renal failure, and ventricular are not statistically related to supraventricular rhythm disorders, or to operative mortality.

Results:

Table N°I: postoperative complications (univariate analysis) in patients who died and those who did not.

Variables	deceased (n=10)	not deceased (n=136)	P
Circulatory support			
Essential (>3 mm)	6(60%)	8(6%)	**<0.0001**
Hemodynamic instability	7(70%)	19(14%)	<0.0001
Refractory low output	8(80%)	27(20%)	<0.0001
Heart failure	8(80%)	34(25%)	0.0005
Myocardial infarction	1(10%)	1(1%)	0.027
Cerebrovascular accident	1(10%)	4(3%)	0.27
Renal insufficiency	**4(40%)**	22(16%)	0.081
BAVIII degree	5(50%)	13(10%)	**0.0004**
Supraventricular arrhythmia	4(40%)	56(41%)	0.79
Ventricular arrhythmia	1(10%)	3(2%)	0.17

In univariate analysis, the clinical parameters significantly associated with operative mortality were: advanced age (p=0.038), atrial fibrillation (p=0.038), severe dyspnoea (p=0.020) and congestive heart failure (p=0.0016); among the echocardiographic parameters, the presence of a lowered shortening fraction (p=0.048) and a low mean transvalvular gradient (p=0.035) significantly increased the risk of operative mortality. We are approaching the significance threshold for the presence of a reduced ejection fraction (p=0.054). The existence of tritronvascular coronary disease (non-bypassable) worsens the operative prognosis.

In multivariate analysis, the presence of a mean transvalvular gradient of less than or equal to 30mm Hg was found to be the only independent predictive factor of operative mortality (p=0.047); the presence of advanced age was close to the significance threshold (p=0.071).

Table 2: Operative mortality (univariate analysis): clinical and biological parameters related to operative mortality in our 146 patients.

Variables	Deceased (n=10)	Not deceased (n-136)	P
Age (year)	50+/-8	55+/-7	**0.043**
Sex M (% F (%)	7(70%)/3(30%)	109(80%)/27(20%)	0.53
Arterial hypertension, n(%)	7(70%)	59(43%)	0.16
Hypercholesterolemia, n(%)	6(60%)	38(28%)	0.052
Diabetes, n (%)	2(20%)	15(11%)	0.96
Smoking, n (%)	3(30%)	66(49%)	0.17
Body surface area (m^2)	1.72+/-0.09	1.72+/-0.16	0.96
Atrial fibrillation, n(%)	6(60%)	35(26%)	**0.033**
History of MI, n (%)	1(10%)	10(7%)	0.82
Charlson index	5.40+/-1.43	4.58+/-1.74	0.15
Stress angina, n (%)	2(20%)	51(37.5%)	0.20
Stress syncope, n (%)	3(30%)	17(12.5%)	0.16
NYHA functional stage	3.8	3.0	**0.020**
Left sided heart failure, n (%)	7(70%)	30(22%)	**0.0016**
acute lung oedema, n (%)	5(50%)	40(30%)	0.24
Natremia (mmol/l)	138+/-4	139+/-3	0.25
Creatininemia (umol/l)	155+/-79	119+/-81	0.17

Table 3: Operative mortality (univariate analysis): radiological, electrocardiographic, coronary angiographic and echocardiographic parameters related to operative mortality in our 146 patients.

Variables	Deceased (n=10)	Not deceased (n=136)	P
Cardiothoracic index (%)	57+/-6	54+/-5	0.083
Complete LBBB, n (%)	3(30%)	14(10%)	0.082
Sokolow index (mm)	28+/-12	35+/-12	0.11
Coronary artery disease, n (%)	5(50%)	**26(19%)**	**0.14**
- Mono-truncular, n (%)	2(20%)	13(10%)	0.35
- Bi-truncular, n (%)	1(10%)	7(5%)	0.8
- Tri-truncular, n (%)	2(20%)	6(4%)	**0.049**
Aortic surface (cm^2)	0.54+/-0.18	0.61+/-0.15	0.20
Indexed aortic surface (cm^2/m^2)	0.29+/-0.09	0.33+/-0.08	0.23
Average gradient (mmhg)	43+/-22	56+/-18	**0.035**
Average gradient <or=30mmhg, n(%)	4(40%)	9(7%)	**0.0057**
DTDVG (mm)	56+/-8	54+/-8	0.83
DTDVG/SG (mm/m^2)	31+/-5	31+/-5	0.87
DTDVG (mm)	43+/-8	40+/-10	0.26
DTSVG/SC (mm/m^2)	24+/-5	22+/-6	0.28
MVG/SC (g/m^2)	154+/-44	176+/-10	0.18
Shortening fraction (%)	22+/-7	29+/-10	**0.048**
Ejection fraction (%)	36+/-17	49+/-20	0.054
Cardiac index (l/mn/m^2)	2.17+/-0.63	**2.84+/-0.55**	**0.0004**
systolic pulmonary artery pressure (mmhg)	47+/-19	43+/-15	0.41

Table 4: Operative mortality (univariate analysis): operative parameters related to operative mortality in our 146 patients

Variables	Deceased (n=10)	Not deceased (n=136)	P
Type of prosthesis M(%) B(%)	7(70%) 3(30%)	129 (94%)/7(6%)	0056
Prosthesis size (mm)	22+/-3	23+/-2	0.36
Coronary artery bypass grafting, n (%)	00	02(1%)	0.22

Table 5: Multivariate logistic regression analysis of parameters predictive of operative mortality.

Variables	Odds ratio	IC 95%	P
Age	1.13	0.98-1.30	0.071
Atrial fibrillation	2.41	0.43-13.44	0.31
NYHA functional stage	1.83	0.28-11.82	0.52
Congestive heart failure	2.90	0.33-25.25	0.33
Tri-truncular coronary disease	7.49	0.58-95.93	0.12
Shortening fraction	1.00	0.88-1.15	0.91
Mean gradient	1.03	0.96-1.10	0.31
Mean gradient<or=30mmhg	16.84	1.04-272.13	**0.047**
Cardiac index	0.18	0.24-1.40	0.10

Late mortality refers to patients who died after the hospital stay (>30 days); all patients lost to follow-up were excluded from the study. in our series have been followed up to date. Mean follow-up was 37+/-29 months for group 1 and 42+/-30 months for group 2. After the operation, 23/136 patients died (13/65 or 20% in group 1 and 10/71 or 14% in group 2)

Table 6: Causes of late mortality in the 2 groups

Causes de mortalité tardive	Groupe 1	Groupe2
Mort subite	03	02
Insuffisance cardiaque	05	01
AVC	03	02
Infection	02	01
Autres	00	02
Indéterminées	00	02
Total	**13**	**10**

Late survival does not include operative mortality; thus, the late survival curves for group 1 and group 2 are not statistically different (p=0.43) with a similar actuarial survival rate at 42 months, but lower in group 1 than in group 2 86.8% vs 93%.

In univariate analysis, the parameters significantly associated with late mortality were hypertension

hypertension (p=0.050) and a high Sokolow index (p=0.0086).

In multivariate analysis, however, no variable was identified as an independent predictor of late mortality.

III / Total mortality:

Total mortality takes into account operative mortality and late mortality at follow-up; 33/146(23%) died, 21/73(29%) in group 1 and 12/73(16%) in group 2.

The total survival curves of group 1 and group 2 were statistically different (p=0.04) with a better actuarial survival rate at 42 months in group 2: 87.2% vs 76.4%.

In univariate analysis, the presence of arterial hypertension (p=0.017) and atrial fibrillation (p=0.0083) were the two clinical variables that significantly increased the risk of total mortality.

Multi-variate analysis identified atrial fibrillation (p=0.047) and a low cardiac index (p=0.0074) as independent predictive factors of total mortality.

Table 7: Cox multivariate analysis of parameters predictive of total mortality.

Variables	Odds ratio	CI 95%	P
Arterial hypertension	2.03	0.96-4.30	0.062
Atrial fibrilation	2.04	1.01-4.13	**0.047**
Cardiac index	0.40	0.20-0.78	**0.0074**

We also determined the factors predictive of mortality by univariate and multivariate analyses, but this time only in group 1 (n=73).

We found the same significant parameters and the same independent predictive factors for operative, late and to-tal mortality as in the analysis of the total population.

I / Operative mortality:

In univariate analysis, the parameters significantly associated with operative mortality were: arterial hypertension (p=0.039), global heart failure (p=0.030), smaller indexed left ventricular mass (p=0.039), mean gradient less than or equal to 30mmhg (at the limit of significance (p=0.053), low cardiac index (p=0.004) and single-truncated coronary disease (0-0.042).

In multivariate analysis, no variable was identified as an independent predictor of operative mortality; however, the presence of a low cardiac index was close to the significance threshold with p=0.056.

II/Late mortality:

In univariate analysis, the parameters significantly associated with late mortality were: arterial hypertension (p=0.036), atrial fibrillation (p=0.0089) and a higher Sokolow index (p=0.0094).

In multivariate analysis, no variable was identified as an independent predictor of late mortality.

III/ Total mortality:

In univariate analysis, the parameters significantly associated with total mortality were: arterial hypertension (p=0.0043), atrial fibrillation (p=0.0021) and complete left bundle branch block (p=0.023). In multivariate analysis, no variable was identified as an independent predictor of operative mortality. We approached the significance threshold for the presence of complete left bundle branch block with p=0.066.

Finally, we analysed changes in functional status and left ventricular ejection fraction for patients in group 1 :

1/ NYHA functional status:

In group 1, the 8/73 patients who died during the operation were NYHA stage IV before the operation.

We collected the functional status of the 65 patients who survived before and after aortic valve replacement with a mean follow-up time of 37 months +/-30 (min: 8 months, max: 105months). Of these, 94% (61/65) were stage III-IV pre-operatively compared with only 11% (7/65) post-operatively; 91% (59/65) had functional improvement of at least one NYHA class and 83% (54/65) of at least two NYHA classes. 8 to 1.7.

In univariate analysis, the parameters significantly associated with improvement in the functional stage of at least one NYHA

class were: the absence of arterial hypertension (p=0.01), the absence of diabetes (p=0.01), the absence of renal insufficiency (p=0.0085) and a larger preoperative indexed aortic valve area (p=0.022).

In other words, patients with hypertension, diabetes, functional renal insufficiency and/or a small preoperative indexed aortic valve area were less likely to improve their functional status after surgery.

Multivariate analysis identified the absence of arterial hypertension as an independent predictor of functional improvement (p=0.04).

2/ Evolution of LVEF:

In group 1, after aortic valve replacement, we were able to collect the ejection fraction in all survivors within a mean time of 27 months+/-23(min: 04 months, max: 83 months). The 8 patients (out of 73) who died during the operation had a preoperative LVEF of 22, 27, 34, 35, 19, 31, 33 and 28%. The ejection fraction of the 65 surviving patients was on average 30%+/-4.5 preoperatively (min 19%, max: 35%) and 58%+/-12 postoperatively (min: 28%, max: 76%). 85% (55/65) recovered at least 10 LVEF points compared with the preoperative ejection fraction.

The average improvement in ejection fraction was 28+/-13% after valve replacement.

In univariate analysis, the only parameter significantly associated with an improvement in ejection fraction of at least 10 points was the existence of a higher mean preoperative gradient (p=0.0089).

In other words, patients with a low mean gradient are less likely to improve their ejection fraction after surgery.

There was a statistically significant positive linear relationship between the recovery of ejection frac-tion before and after surgery and the preoperative mean gradient (p=0.0079 and r= 0.34).

Discussion:

The operative mortality of aortic valve replacement with left ventricular systolic dysfunction (LVEF<or=35%) and/or mean gradient <or=30mmhg varies in the literature from 9% to 33%.

In our series, operative mortality was significantly higher (p=0.049) in group 1 (with LV systolic dysfunction) compared with group 2 (without LV systolic dysfunction): 11.7% versus 2.9%.

Authors	Headcount n	Inclusion criterias	Operative mortality	Predictive factors
Brogan (1), 1993	18	AG-30mmhg	33%	/
Connolly (4), 1997	154	EF<35%	9%	CAD
Blitz (3), 1998	52	AG<=40mmhg	11%	/
Powel (16), 2000	55	EF<=30 %	18%	History of MI
Connolly (5), 2000	52	EF<=35% AG<=30mmhg	21%	Small size of prothesis
Pereira (17), 2002	39	EF <35% AG<30mmhg	8%	/
Notre etude, 2004	68	EF <=35%	11.7%	EF<=30mmhg

EF: ejection fraction; AG: trans-aortic average gradient; CAD: coronary artery desease

Operative mortality for AS in the presence of a mean gradient of less than 30mmhg is also higher in more recent series of the literature (with the exception of Pereira et al.): 21% for Connolly et al(5), 33% for Brogan et al(1), Pereira et al (17), in a recent series, compared two groups of non-randomised patients with tight aortic stenosis with LVEF<or= 35% and mean gradient <or= 30mmhg. Patients in the first group underwent surgery and patients in the second group were treated medically.

Survival was significantly better in the surgical group: 82% at one year and 78% at 4 years, compared with 41% at one year and 15% at 4 years in the "medical" group (p<0.0001).

The spontaneous prognosis of patients with EF<or =35% and GM < or = 30mmhg is therefore particularly poor.

Thus, the operative mortality of AS with LV systolic dysfunction is higher but ultimately acceptable, given its very unfavourable spontaneous prognosis.

Various factors influencing operative mortality have been identified by authors: the mean trans-valvular gradient, the size of the prosthesis and coronary disease.

In 1980, Carabello et al (7) studied 14 patients with tight aortic stenosis, LVEF less than 45% and no significant coronary disease.

10 patients out of 14 had a mean gradient >30mmhg and 4 patients out of 14 had a mean gradient <30mmhg.

Among patients with GM>30mmhg, all were improved by surgery: among patients with GM<30mmhg, 3 died and 1 was not improved by surgery.

For these authors, a low trans-valvular gradient is an independent factor of poor operative prognosis, also linked to an intrinsic impairment of myocardial contractility.

In our study, if we isolate the subgroup of patients with GM<or=30mmhg, 4 patients out of 13 died.

In the study by Connolly et al (4) of 154 patients with LVEF<or=35%, the preoperative mean trans-valvular aortic gradient was lower in patients who died during the operative period (35+/-18mmhg versus 45+/-18mmhg, p=0.009) in univariate analysis, but this variable was not identified as an independent preoperative predictive factor in multivariate analysis.

In our study, the presence of a mean gradient of less than or equal to 30 mmhg was found to be the only independent predictive factor of operative mortality (p=0.047). A low preoperative mean trans-valvular aortic gradient is a factor of poor operative prognosis in patients with tight aortic stenosis and low ejection fraction.

A recent study by Connolly et al (5) included 52 patients with narrow aortic stenosis with a low mean trans-valvular gradient of less than 30mmhg associated with an ejection fraction of 35% or less. The operative mortality rate in this series was 21%.

In addition to advanced age in univariate analysis, this study identified small implant size in both univariate and multivariate analysis as a predictive factor for operative mortality in these patients.

The hypothesis put forward, which remains to be confirmed, to explain these results would be the persistence of the consequent increase in afterload linked to this small size of the prosthesis.

For this reason, Rahimtoola et al (18) have suggested that in this type of patient requiring the use of
prosthesis smaller than 21mm, to use so-called stentless or homograft prostheses.

Collinson et al(6) emphasised the benefits of stentless prostheses, which would enable a reduction in systolic stress to be achieved early on and better recovery of ventricular function.

In the series by Powell et al (16) of 55 patients operated on for AR with LVEF<or=30%, 10 patients (18%) died during the operation, 9 of whom had a history of MI. In univariate analysis, the presence of severe NYHA dyspnoea, a low mean gradient and a history of MI were associated with operative mortality. In multivariate analysis, the presence of a history of MI was the only independent predictor of operative mortality.

In the first study by Connolly et al (4), which looked at 154 AR patients with LVEF <or=35%, univariate analysis identified a decrease in mean gradient and a history of myocardial infarction as poor prognostic factors.

The existence of coronary disease (70% or more stenosis of 2 epicardial vessels or 50% or more stenosis of the common trunk of the left coronary artery) was the only factor in multivariate analysis predictive of a poor operative prognosis.

In our study, the percentage of coronary artery disease was higher in deceased patients than in survivors, 50% (5/10) versus 20% (25/126), although the difference was not significant (p=0.14). In univariate analysis, the presence of tri-truncutary coronary disease was significantly associated (p=0.049) with operative mortality.

However, in multivariate analysis, coronary disease was not found to be an independent factor in operative mortality (p=0.12).

It should be noted that the rate of patients with MI is very high in the populations of previous series: 36% (20/55) for Powell et al (16), 25% (39/154) for Connolly et al (4) compared with 8% in our study.

The rates of coronary artery disease and associated coronary artery bypass grafting were also very high in the Connolly et al cohort: 55% (85/154) and 51% (78/154) respectively, compared with 22% (30/136) and 2% in our study.

Furthermore, in the Connolly et al study, the patients were those with severe coronary artery disease defined either by bi- or tri-truncular involvement, or by involvement of the common trunk. In the latter two cases, we cannot speak of left ventricular
systolic dysfunction secondary to aortic stenosis, but rather of the intertwining of two distinct diseases, with mortality aggravated by coronary disease in its own right (rhythm disorders, etc.).

Finally, if we compare medium-term survival, the survival rates of patients operated on for AR with left ventricular systolic dysfunction are lower than those of "normal" patients operated on for AR. In the study by Connoliy et al (4) of 154 patients with tight aortic stenosis and LVEF<or=35%, the actuarial survival rate including operative mortality was 58% at 5 years.

One of the advantages of our study is that we have matched the two groups of patients, enabling us to compare the medium-term survival of patients operated on for AR with or without preoperative left ventricular systolic dysfunction.

The total survival curves (hospital phase and follow-up) are significantly different (p=0.04) for patients in the group with left ventricular dysfunction compared with patients in the group without left ventricular systolic dysfunction, with an actuarial survival rate of 76.4% and 87.2% respectively at 42 months.

It is to be expected that these results show that life expectancy is better for AR patients without left ventricular systolic dysfunction. However, in our study, if we look at the outcome after the hospital phase
hospital phase, the late survival curves (excluding hospital mortality) of group 1 and group 2 are not statistically different (p=0.43) with a similar actuarial survival rate at 42 months in group 1 and group 2: 86.8% versus 93%.

The difference in total survival (hospital phase and follow-up) between the two groups is therefore essentially linked to operative mortality.

Given the very poor prognosis of this disease in the absence of surgical intervention (74), our results clearly show that, despite a higher operative mortality which is acceptable in this context, patients with tight AS complicated by LV dysfunction benefit overall in the medium term from aortic valve replacement.

Various factors influencing medium-term survival were identified: the existence of atrial fibrillation and a low cardiac index were found to be independent predictors of total mortality (p=0.0466 and p=0.0074 respectively). We know that atrial fibrillation is a poor prognostic factor in heart failure. The presence of a low cardiac index is also one of the two parameters identified as independent predictors of total mortality by Connolly et al (4) in their study including 154 AR patients with LVEF<or=35%.

The second parameter identified by Connolly et al (4) is coronary disease. Its presence affects the survival rate: 39% at 5 years for coronary patients versus 69% for non-coronary patients (p=0.02).

However, in our study, coronary disease was not found to be an independent prognostic factor.

This may be explained by the high rate of coronary heart disease and history of MI in the North American cohort of Connolly et al. compared with our North African population.

We know that mortality is high in coronary patients, particularly those with
severe LV dysfunction (ventricular rhythm disorders due to sequelae of MI, etc.). Thus, the association of ischaemic heart disease with severe ARF logically worsens the prognosis.

It should be noted that there is a clear and close correlation between the two variables cardiac index and mean gradient; the gradient is independent of cardiac output. Connolly et al highlight the impact of a low mean gradient on mortality.

Indeed, in their second study(5), où mean gradient less than 30mmhg was an inclusion criterion, the authors report a high total mortality rate: 40.4% of patients (21/52, 2 lost to follow-up) died during follow-up (18 months on average), 3-year survival in this series was 62%.

The life expectancy of these patients with a mean gradient less than 30 mmhg is also statistically lower (p=0.04) than that of patients with a preoperative trans-valvular gradient greater than 30 mmhg.

A small prosthesis, hymodynamically less efficient and reflecting a small left ventricular outflow chamber, remains a factor of poor prognosis after surgery regardless of the existence of left ventricular dysfunction (14, 15).

Conclusion:

In our series, the statistical analysis of the results allowed us to demonstrate that the operative mortality of aortic valve replacement for tight AR was significantly higher (p=0.048) in the group with left ventricular systolic dysfunction compared to the group without ventricular systolic dysfunction. left: 11.7% versus 2.9%.

However, once the surgical milestone has passed, the late survival curves (operative mortality excluded) of group I and group 2 are not statistically different (p=0.43) with a similar actuarial survival rate at 42 months in group 1 and in group 2: 86.8% vs 93%.

Thus, the difference in total survival (hospital phase and follow-up) between the two groups is essentially linked to operative mortality.

The increased operative risk, in the event of impairment of left ventricular function, remains acceptable given the very poor spontaneous prognosis of this disease in the absence of surgery. In addition, surgical intervention usually allows a spectacular improvement in LVEF and symptomatology in the majority of cases.

Thus, despite a higher operative risk, patients with severe aortic stenosis complicated by left ventricular systolic dysfunction should, in our opinion, in the majority of cases, benefit from aortic valve replacement, especially in young subjects (age <65 years) and without defect. obvious associate. The intervention should only be postponed in the face of a collapse of left ventricular function with aggravating multiple visceral lesions.

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