Journal of Clinical and Basic Cardiology

An Independent International Scientific Journal

Journal of Clinical and Basic Cardiology 2001; 4 (2), 165-167

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Indexed in
Chemical Abstracts
EMBASE/Excerpta Medica

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Early Post-Operative Haemodynamic and Neurohumoral Follow-Up After Endoaneurysmorrhaphy

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Results of endoaneurysmorrhaphy (EAR) have been followed haemodynamically and functionally. Since heart failure and myocardial infarctions with left ventricular (LV) dysfunction are associated with prognostically significant neurohumoral activation, we sought to study patients undergoing EAR haemodynamically and neurohumorally in the early post-operative period. Arterial plasma levels of N-terminal pro-atrial natriuretic peptide (Nt-proANP), N-terminal pro-brain natriuretic peptide, arginine vasopressin (AVP), endothelin, norepinephrine and epinephrine were measured before and early postoperatively (day 6–21, median 8.5) in 11 patients undergoing EAR. Pre- and postoperative haemodynamic and angiographic examination was carried out in all patients. Ejection fraction improved from 31.7 ± 4.3 to 48.2 ± 4.4 % ($p < 0.005$) after surgery. Left ventricular end-diastolic pressure (LVEDP) increased significantly from 11.3 ± 2.4 to 19.1 ± 3.5 mmHg ($p < 0.05$). The Nt-proANP levels also increased significantly from 1121 ± 219 to 1921 ± 190 fmol/ml ($p < 0.005$). Postoperatively, LVEDP showed a positive correlation with plasma Nt-proANP ($r = 0.73$, $p = 0.01$). There appears to be a discordance in LV pump functions early postoperatively. An improvement in systolic performance is accompanied by a worsening of diastolic function. This is further substantiated by an increase in Nt-proANP, a bad prognostic marker. The results are, however, preliminary. *J Clin Basic Cardiol 2001; 4: 165–167.*

Keywords: aneurysm, revascularisation, myocardial infarction, haemodynamics, hormones

Formation of left ventricular aneurysms is not uncommon after myocardial infarction [1]. Surgical excision is carried out to improve the clinical manifestations, most often heart failure, but sometimes also angina, embolization, and life threatening tachyarrhythmias [2, 3]. The operation has evolved from conventional aneurysmectomy, ie repair with linear plication of the aneurysm sac to the present day techniques of aneurysmorrhaphy, which emphasize the restoration of normal left ventricular geometry [1, 4]. It has been customary to follow-up these cases haemodynamically and functionally. Thus literature is replete with reports of improvement in left ventricular ejection fraction and New York Heart Association (NYHA) class. Since heart failure and large myocardial infarctions are associated with significant neurohumoral activation [5, 6], it would be of interest to have a neurohumoral follow-up of these patients. If the surgery improves left ventricular functions, it must have an effect on the neurohumoral profile. We sought to have a short term haemodynamic, angiographic and neurohumoral follow-up of patients undergoing endoaneurysmorrhaphy, and in doing so attempted to find out changes which could have a bearing on the ultimate outcome of the operation.

Methods

Eleven patients were studied early postoperatively (day 6–21; median 8.5). All had congestive heart failure and anterior left ventricular aneurysm. In every case the aneurysm resulted from myocardial infarction. Indications for operation were congestive heart failure and/or angina pectoris. Co-morbid risk factors like hypertension, diabetes mellitus, atrial fibrillation, mitral regurgitation, ventricular arrhythmias were present in some patients. All patients were receiving decongestive treatment in the form of diuretics, angiotensin-converting enzyme inhibitors, angiotensin receptor antagonists, low dose beta-blockers, etc. and all gave written informed consent. Cooley's surgical technique was followed, the details of which have been cited in [7]. Intracardiac pressures were obtained during cardiac catheterisation and quantitative left ventriculography (QLVA-CMS, Medis Medical Imaging Systems, Leiden, Holland) was employed to derive the angiographic data. Global left ventricular ejection fraction was calculated by the area-length method. Arterial blood was collected after coronary angiography. It was centrifuged within 20 minutes and the separated plasma was stored at –80 degree centigrade until assayed. A competitive enzyme immuno assay (EIA, Biomedica GmbH, Vienna, Austria) was used to measure the immunoreactive N-terminal proatrial natriuretic peptide (Nt-proANP) (1–30) and N-terminal pro-brain natriuretic peptide (Nt-proBNP) (8–29). Norepinephrine and epinephrine were quantified by high-pressure liquid chromatography (HPLC) and electrochemical detection. Commercially available enzyme linked immunosorbent assay (ELISA, Biomedica GmbH Vienna, Austria) was used to measure endothelin. Quantitative determination of argenine vasopressin was done by using a competitive enzyme immunoassay (Assay Designs, Inc., Ann Arbor, U.S.A.). Data are presented as mean ± S.E.M. Paired t-test was used for comparisons between pre- and postoperative parameters in patients. Bivariate Pearson analysis for correlations was done by SPSS software from SPSS Inc. A p-value of < 0.05 was used to indicate statistical significance.

Results

The results are summarized in Tables 1 and 2. Stroke volume showed an insignificant rise, though on subgroup analysis a differential pattern emerged, with a significant rise from 37 ± 8 to 61 ± 9 ml ($n = 6$, $p = 0.02$) in patients having low preoperative stroke volumes ≤ 70 ml), and a rather insignificant fall (93 ± 12 vs. 67 ± 8, $n = 5$; ns) in patients having higher (> 70 ml) preoperative stroke volumes. In the latter group, the postoperative rise of the ejection fraction may have re-

Received September 13th, 2000; accepted March 6th, 2001.
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Table 1. Haemodynamic Data

	Preoperative	Postoperative	p value
LVEF(%)	31.7 ± 4.3	48.2 ± 4.4	0.001*
ESV (ml)	130 ± 18	84 ± 16	0.02*
EDV (ml)	190 ± 22	154 ± 19	0.12
SV (Ml)	66 ± 11	67 ± 5	0.97
LVESP (mm Hg)	107 ± 9	109 ± 4	0.86
LVEDP (mm Hg)	11.3 ± 2.4	19.1 ± 3.5	0.03*
HR (beats/min)	76 ± 5	89 ± 7	0.06

LVEF = left ventricular ejection fraction; ESV = end systolic volume; EDV = end diastolic volume; SV = stroke volume; LVESP = left ventricular end systolic pressure; LVEDP = left ventricular end diastolic pressure; HR = heart rate; * = statistical significance

Table 2. Neurohumoral Data

	Preoperative	Postoperative	p value
NE (pg/ml)	544 ± 158	433 ± 77	0.40
EPI (pg/ml)	69 ± 5	104 ± 24	0.16
Nt-proANP (fmol/ml)	1121 ± 219	1921 ± 190	0.00 1
Nt-proBNP (fmol/ml)	1205 ± 199	1356 ± 152	0.36
AVP (pg/ml)	53 ± 7	67 ± 11	0.08
ET (fmol/ml)	0.4 ± 0.1	0.7 ± 0.2	0.15

NE = norepinephrine; EPI = epinephrine; Nt-proANP = N-terminal pro-atrial natriuretic peptide; Nt-proBNP = N-terminal pro-brain natriuretic peptide-, AVP = argenine vasopressin; ET = endothelin; * = statistical significance

sulted from a decrease in the left ventricular end-diastolic volume from 247 ± 32 ml to 137 ± 20 ml ($p < 0.05$).

Discussion

Cooley and co-workers described "ventricular endoaneurysmorrhaphy", a procedure that repairs the aneurysm by placing an intracavitary elliptical Dacron patch [7]. The restoration of left ventricular cavity theoretically accounts for improved myocardial performance seen with these repairs [1, 8]. Restoration of ventricular geometry reduced the paradoxical contractile forces. The end-diastolic volume is decreased, thereby diminishing wall tension, which in turn decreases myocardial oxygen demand.

The post-repair ventricle can function therefore on a more leftward point on the Starling curve [9]. In the present study we observed an increase in left ventricular ejection fraction in all patients. There was a significant decrease in left ventricular end-systolic volume and though there was a trend towards decrease in end-diastolic volume, this could not reach statistical significance, perhaps due to the small cohort size. Interestingly, the left ventricular end-diastolic pressure (LVEDP) increased significantly after surgery. To the best of our knowledge this is the first study suggesting worsening of diastolic performance after endoaneurysmorrhaphy during the early post-operative phase. Dor et al. in their series of 171 patients reported a significant decrease in pulmonary pressures and pulmonary capillary wedge pressures shortly after surgery. These parameters, however, were significantly higher at one year compared to both basal and early post-operative values [10]. In contrast, we observed a significant increase in LVEDP shortly after surgery (median 8.5 days). The post-operative ventricle presumably had an upward and steeper shift of the diastolic pressure volume curve, and the left ventricular distensibility was altered. The LVEDP is influenced by passive myocardial properties, left ventricular chamber volume, and relative left ventricular wall thickness [11]. The influence of the stiff [10] synthetic Dacron patch may be important in this context. This apparent dichotomy of improvement in ejection performance and worsening of diastolic function is the principal finding of our study.

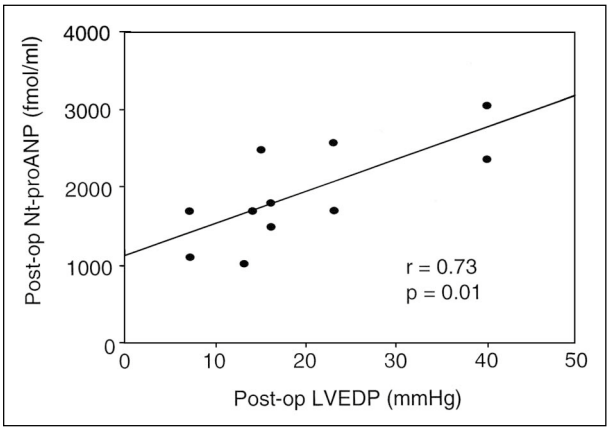

Figure 1. Scattergram illustrating the relationship between postoperative left ventricular end-diastolic pressure (LVEDP) and N-terminal proatrial natriuretic peptide (Nt-proANP) plasma concentration

The observation is further substantiated by a significant rise in Nt-proANP post-operatively. A significant correlation was found between LVEDP and Nt-proANP (figure 1), much in conformity with Takeichi et al., who found a linear correlation between plasma ANP levels, and pulmonary capillary wedge pressure and also between LVEDP and maximal left atrial volume [12]. The Nt-proANP has been shown to be an independent predictor of cardiovascular mortality and the development of heart failure following myocardial infarction [6, 13]. In a multivariate analysis Nt-proANP in contrast to ANP and other neurohormones was found to be a powerful and independent predictor when the model included age, gender, prior myocardial infarction, hypertension, diabetes, use of thrombolysis, Killip class, infarct location, and left ventricular ejection fraction. For the combined end-point of cardiovascular death or severe heart failure, Nt-proANP was the best clinical or laboratory predictor, even superior to age, left ventricular ejection fraction and prior myocardial infarction [6]. As increasing filling pressures are a major stimulus for eccentric ventricular hypertrophy [14] increased atrial pressure suggested by elevated ANP levels may be a marker of the risk of ventricular dilatation and thus increased mortality. Dor et al reported a significant increase in ventricular volumes in all subsets of patients one year after operation, compared to the early postoperative period [10].

Compared to Nt-proANP, the Nt-proBNP did not increase significantly. This was in consonance with other studies where LVEDP correlated significantly with ANP but not BNP levels [15]. Plasma concentration of BNP, however, rose in patients with symptomatic left ventricular systolic dysfunction [16, 17]. There was a strong correlation between BNP and left ventricular ejection fraction. It is possible that changes in left ventricular ejection fraction are better reflected by BNP, while LVEDP is better mirrored by ANP.

Other neurohormones did not change significantly. A positive correlation was found between the preoperative levels of circulating argenine vasopressin and the postoperative improvement of stroke volume ($r = 0.80$; $p = 0.02$). This could be a chance finding or it possibly could reflect the fact that neuroendocrine activation increases with the increase in left ventricular dysfunction [18], and that the patients who benefit most from endoaneurysmorrhaphy are those with more severe preoperative left ventricular compromise [10]. We also observed a significant rise in stroke volume in only those

patients who had a low pre-operative stroke volume (< 70 ml). It therefore transpires that if the observations are vindicated in a larger study, preoperative argenine vasopressin levels may be used as markers of post-operative improvement.

There are certain limitations of the study. Besides small cohort size and short follow up, there is absence of a control group with myocardial revascularisation but no EAR. Cardioactive drugs, which could have significant influence on neurohumoral status, were continued throughout the study period. Finally, follow up does not include clinical status.

In conclusion, there appears to be a discordance in left ventricular pump functions after endoaneurysmorrhaphy early postoperatively. An improvement in systolic performance is accompanied by a worsening of diastolic function. This is further demonstrated by an increase in Nt-proANP, a bad prognosticator. The observations are preliminary and need further validation.

Acknowledgement

We thank Mrs Anke Constantz, Mrs Cindy Krause and Mrs Dorit Kemken for excellent technical help. This work was supported by a grant of Deutsche Forschungsgemeinschaft (R/423/4-1).

References

1. Cooley DA, Frazier OH, Duncan JM, Reul JR, Krajcer Z. Intracavitary repair of ventricular aneurysm and regional dyskinesia. Ann Surg 1992; 215: 417–24.
2. Stephenson LW, Hargrove WC, Ratcliffe MB, Edmunds LH. Surgery for left ventricular aneurysm: Early survival with and without endocardial resection. Circulation 1989; 79 (Suppl X): 1108–11.
3. Couper GS, Bunton RW, Birjiniuk V, DiSesa VJ, Fallon MP, Collins JJ, Cohn LH. Relative risk of left ventricular aneurysmectomy in patients with akinetic scars versus true dyskinetic aneurysms. Circulation 1990; 82 (Suppl IV): 248–56.
4. Dor V, Montiglio F, Sabatier M, Coste P, Barletta G, Di Donato M, Toso A, Baroni M, Fantini F. Left ventricular shape changes induced by aneurysmectomy with endoventricular circular patch plasty reconstruction. Eur Heart J 1994; 15: 1063–9.
5. Francis GS, Goldsmith SR, Levine TB, Olivari MT, Cohn JN. The neurohumoral axis in congestive heart failure. Ann Intern Med 1984; 101: 370–7.
6. Hall C, Rouleau JL, Moye L, de Champlain J, Bichet D, Klein M, Sussex B, Packer M, Rouleau J, Arnold MO, Lamas GA, Sestier F, Gottlieb SS, Wun CC, Pfeffer MA. N-terminal proatrial natriuretic factor. An independent predictor of long-term prognosis after myocardial infarction. Circulation 1994; 89: 1934–42.
7. Cooley DA. Ventricular endoaneurysmorrhaphy: a simplified repair for extensive postinfarction aneurysm. J Card Surg 1989; 4: 200–5.
8. Mills NL, Everson CT, Hockmuth DR. Technical advances in the treatment of left ventricular aneurysm. Ann Thorac Surg 1993; 55: 792–800.
9. Grossi EA, Chinitz LA, Galloway AC, Delianides J, Schwartz DS, McLoughlin DE, Keller N, Kronzon Spencer FC, Colvin SB. Endoventricular remodeling of left ventricular aneurysm. Functional, clinical and electrophysiological results. Circulation 1995; 92 (Suppl 11): 98–100.
10. Dor V, Sabatier M, Di Donato M, Maioli M, Toso A, Montiglio F. Late hemodynamic results after left ventricular patch repair associated with coronary grafting in patients with postinfarction akinetic or dyskinetic aneurysm of the left ventricle. J Thorac Cardiovasc Surg 1995; 110: 1291–301.
11. Glantz SA, Parmley WW. Factors which affect the diastolic pressure volume curve. Circ Res 1978; 42: 171–80.
12. Takeichi N, Fukuda N, Tamura Y, Oki T, Ito S. Relationship between left atrial function and plasma level of atrial natriuretic peptide in patients with heart disease. Cardiology 1998; 90: 13–9.
13. Dickstein K, Aarsland T, Hall C J. Plasma N-terminal atrial natriuretic factor: a predictor of survival in patients with congestive heart failure. Card Fail 1997; 3: 83–9.
14. Grossman W, Jones D, McLaurin LP. Wall stress and patterns of hypertrophy in the human left ventricle. J Clin Invest. 1975; 56: 56–64.
15. Matsumoto N, Akalke M, Nishiuchi T, Kawai H, Saito S. Different secretion profiles of atrial and brain natriuretic peptides after acute volume loading in patients with ischemic heart disease. Acta Cardiol 1997; 52: 261–72.
16. Wei CM, Heublein DM, Perrella MA, Lerman A, Rodeheffer RJ, McGregor CG, Edwards WD, Schaff HV, Burnett JC Jr. Natriuretic peptide system in human heart failure. Circulation 1993; 88: 1004–9.
17. Motwani JG, McAlpine H, Kennedy N, Struthers AD. Plasma brain natriuretic peptide as an indicator of anglotensin converting enzyme inhibition after myocardial infarction. Lancet 1993; 341: 1109–13.
18. Francis GS, Benedict C, Johnstone DE, Kirlin PC, Nicklas J, Liang CS, Kubo SH, Rudin-Toretsky E, Yusuf S. Comparison of neuroendocrine activation in patients with left ventricular dysfunction with and without congestive heart failure. A substudy of the Studies of Left Ventricular Dysfunction (SOLVD). Circulation 1990; 82: 1724–9.

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