A New Percutaneous Vascular Surgical Device After PTCA and Stenting

Winfried Haerer, Ralph Schulte, Bernd Ludwig. Dr Haerer Hospital, Ulm, Germany

The femoral artery is the most common vessel access for interventional procedures. Local problems are frequent by using larger devices or strong anticoagulation protocols after stenting. Therefore, we used the new single operator percutaneous vascular surgical device (Perclose) in a prospective trial. The new 9 F-device has a sheath which houses four sutured needles. The knots are made outside and are advanced by a special knot pusher to the artery surface.

A consecutive series of 350 patients (pts) was included. PTCA, athectomy or stent deployment was done by use of 8 or 9 F guiding catheters. A successful stent implantation was carried out in 320 pts of this series. In these cases the surgical device was employed immediately after the interventional procedure and there was no need for a withdrawal of heparin. The patients had to stay in bed for only 4 hours with a mild compression bandage and were allowed to walk after this period.

The results were as follows: Procedural success 322/320 pts (92%), false aneurysm 0/322 pts, haemotoma (over 5 cm) 0/322 pts, vessel thrombosis 0/322 pts. Compression of the artery (up to 1 hour) was done in the remaining 38 pts followed by a tight compression bandage (1 aneurysm). Before use of the new device our local complications were 1% false aneurysm or bleeding with surgical repair and 8% severe haematoma.

The new percutaneous vascular surgical device reduces local complications. Anticoagulation protocols can be administered unchanged and therefore the results of complex angioplasty can be optimized.

The "Dark Side" of Overly Aggressive Multidevice Athectomy


Adjuvant DCA following rotational athectomy (RA) results in a larger lumen than stand-alone RA or RA+adjunctive PTCA. To assess the long-term effects of this strategy, we followed 183 pts treated with RA+adjunctive DCA and managed using intravascular ultrasound. Pre- and post-intervention arterial, lumen, and plaque areas (mm²) were used to calculate plaque burden (plaque/arterial areas) and the contribution of tissue removal to lumen enlargement (athectomy index = plaques/arterial areas). After 11.8 ± 4.5 mos, 38 pts (23%) required target lesion revascularization (TLR).

Reference lumen area and total and superficial areas of lesion calcium were similar. Although post-intervention lumens were slightly smaller (6.0 vs 8.8 mm²), the athectomy index was strikingly greater (84 vs 59%) in pts requiring TLR. We conclude that the strategy of RA+DCA results in favorable long-term pt outcomes (TLR = 23%). However, pts requiring TLR had more aggressive tissue removal (higher athectomy index) suggesting that exaggerated deep vessel wall injury may be deleterious.

Reduced Anticoagulation Protocol for Stenting (RAPS) in Smaller Coronary Vessels Using Intravascular Ultrasound Guidance. 30 Day Procedural Results

David K. Roberts, Arvin Arthur, Rayo L. Bellinger, James E. Lewis, Sheryl L. Hass, Carol A. Parise, Sutter Institute for Medical Research, and U.C. Davis Affiliated Hospitals, Sacramento, CA

Although coronary stenting (S) of smaller < 3 mm vessels (SCV) was initially identified as a risk factor for stent thrombosis, STRESS enrolled a significant number of SCV with acceptable results. We report the initial 30 day results of RAPS in SCV. 43 patients with 57 lesions in 25-3.0 mm native coronary vessels underwent stenting using 79 Palmaz-Schatz coronary stents. Lesion indications were 51 (83.5%) de novo lesions, 1 (1.8%) restenotic, and 5 (8.7%) suboptimal results or threatened closures following balloon angioplasty or athectomy. Clinical presentation was 23 (53.4%) unstable angina, 14 (32.6%) stable angina, and 6 (14.0%) persons with clinical presentation was 72 hrs. S/P MI. Follow-

Intravascular ultrasound (IVUS), final stent deployment was performed at 16.0±0.5 atmospheres. All stents had a final cross-sectional area (CSA) > 80% of reference vessel CSA, and were treated with aspirin (325 mg and ticlopidine 200 mg daily for one month, and enoxaparin 60 mg daily for three weeks. IVUS Results

Reference Vessel CSA mm² 6.65±0.31
Reference Vessel MLD mm 2.5±0.07
Vessel Major/Minor Axis Ratio 0.78±0.02
Reference Stent CSA mm² 7.56±0.40
Reference Stent MLD mm 2.71±0.09
Stent Minor/Minor Axis 0.90±0.2
Mean % CSA Stent Stenosis -13.7±0.4%
Mean % MLD Stent Stenosis -8.4±0.03%

We have previously demonstrated the expression of inducible Nitric Oxide Synthase (iNOS) and peroxynitrite in impaired myocardial cells along with increased nitrate formation and O2: radical production in autoimmune myocarditis rats, suggesting that NO modulates myocardial damage in myocarditis. In the present study, we investigated whether aminoguanidine (AG), a selective inhibitor of iNOS, modulates the inflammatory disorder of myocarditis. Lewis rats were sensitized to develop myocarditis with an injection of porcine myosin. AG (400 mg/kg per day) was intraperitoneally administered to rats every day from the day prior to the first immunization with myosin. Hearts were removed for examination on day 21. Blood samples were simultaneously taken from the heart cavity. The severity of myocarditis was assessed by measuring the histopathologically affected area and serum CK-MB levels were also measured. Mean % area of inflammatory lesions and CK-MB levels were significantly decreased in the rats with myocarditis given AG.

Inhibition of Inducible Nitric Oxide Synthase Reduces Myocardial Injury in Autoimmune Myocarditis Rats

Michiaki Hiroe, Shigeru Ishiyama, Toshiro Nishikawa, Hiroko Nakazawa, Hiroshi Ito, Takashi Shinojo, Takeshi Kasajima, Fumaki Mano, Tokyo Medical and Dental University, Tokyo Women's Medical College, Tokai University, Japan

We have previously demonstrated the expression of inducible Nitric Oxide Synthase (iNOS) and peroxynitrite in impaired myocardial cells along with increased nitrate formation and O2: radical production in autoimmune myocarditis rats, suggesting that NO modulates myocardial damage in myocarditis. Lewis rats were sensitized to develop myocarditis with an injection of porcine myosin. AG (400 mg/kg per day) was intraperitoneally administered to rats every day from the day prior to the first immunization with myosin. Hearts were removed for examination on day 21. Blood samples were simultaneously taken from the heart cavity. The severity of myocarditis was assessed by measuring the histopathologically affected % area and serum CK-MB levels were also measured. Mean % area of inflammatory lesions and CK-MB levels were significantly decreased in the rats with myocarditis given AG.

Conclusions: AG inhibits the extension of myocardial injury in myocarditis by suppressing NO overproduction. AG may be an effective drug for the treatment of myocarditis.

Clinical Significance of Myocardial Inflammatory Cell Infiltration in the Subacute Phase of Myocarditis

Yasushi Abe, Jiyoung Kim, Masatake Fukunumi, Masaharu Ohmori, Tatsushi Shimomagata, Kazuaki Kumagai, Takahisa Yamada, Motoo Dad, Syoji Saita, Noritake Hokki. Division of Cardiology, Otsuka Prefectural Hospital, Osaka, Japan

The inflammatory cell infiltration in the subacute phase of acute myocarditis might deteriorate the recovery of left ventricular(LV) function through factors such as cytokines. To elucidate whether myocardial inflammatory cells infiltration in the subacute phase of myocarditis influences the prognosis of left ventricular function, we studied 16 pts with acute myocarditis diagnosed clinically. Endomyocardial biopsy specimens (3–4 pieces) were taken from...
LV in the subacute phase (1-6 months after the onset of myocarditis), and inflammatory cells (LCM cells) identified with immunoperoxidase staining using anti-leucocyte common antigen serum were counted in high power field of all samples. The LV end diastolic dimension (LVEDd) and ejection fraction (EF) were measured by the echocardiography at the biopsy (B) and one year later (F). There was a significant correlation between the mean number of LCA positive cells and the "change of LVDd (r = 0.551, p < 0.05). 16 pts were classified by the average number of LCA cells into Group-I (n = 7) with 1.0 or more (HF) LCA cells and Group-II (n = 9) with LCA cells less than 1.0 HF. The results were as shown below.

<table>
<thead>
<tr>
<th>Group</th>
<th>LCAcells/HF</th>
<th>LVEDd/mm(EF)</th>
<th>EF-LVEDd/mm(EF)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group-I</td>
<td>2.6 ± 0.8</td>
<td>57.5 ± 5.05</td>
<td>50.6 ± 6.05 ± 0.12</td>
</tr>
<tr>
<td>Group-II</td>
<td>6.6 ± 0.2</td>
<td>55.1 ± 5.05</td>
<td>50.6 ± 6.05 ± 0.09</td>
</tr>
</tbody>
</table>

*p < 0.05 vs Group-I

There was no significant difference in the period from the onset of myocarditis to the biopsy (Group-I vs Group-II: 3.0 ± 1.9 vs 1.9 ± 1.8 months). LVDd decreased in Group-I 1 year after biopsy more than in Group-I. Thus, these results indicate that the improvement of cardiac function in acute myocarditis might be predicted by the degree of residual LCA infiltration in subacute phase (over one month after the onset).

1033-71 Circulating Cardiac Autoantibodies as Autoimmune Markers in Clinical and Biopsy-Proven Myocarditis

Alida L.P. Caforo, Alan J. Haven, Jonathan H. Goldman, Luciano Della Libera, Kamran M. Baig, William J. McKenna. Deps of Cardiology, Exp Biomed Sci, University of Padua, Padua, Italy; Dept of Cardiological Sciences, St. George's Hospital, London, United Kingdom

Myocarditis and dilated cardiomyopathy (DCM) may be phases of an ongoing autoimmune disease of the myocardium. Cardiac autoantibodies (Abs) are found in 30-40% of DCM patients. Their detection in myocarditis could provide evidence for autoimmunity. Cardiac antibody status was assessed in 53 patients from the Myocardiitis Treatment Trial (35 male, aged 42 ± 15 years). The antibody specificities included organ-specific and skeletal muscle cross-reactive Abs by indirect immunofluorescence and anti-s myosin Abs by enzyme-linked immunosorbent assay (ELISA). All patients had clinical myocarditis and an unexplained heart failure, but only 24 were classified as histological myocarditis (Dallas criteria) on endomyocardial biopsy and randomised in the Trial. By immunofluorescence cardiac Abs were more common in myocarditis patients than in normals (13/53, 24% vs 18/200, 9%, p = 0.004); by ELISA normally raised anti-s myosin Abs were also more frequent than in normals (9/53, 17% vs 1/2, 2%, p = 0.005); 18 patients (34%) had a positive result with one or both tests. Similar proportions of patients with and without histological myocarditis contained Abs by immunofluorescence (9/24 vs 5/29 respectively, p = NS) and by ELISA (4/24 vs 5/29 respectively, p = NS). Cardiac Abs are found in 34% of patients with clinical myocarditis, providing evidence for myocardial involvement. The lack of correlation of antibody status with the histological diagnosis of myocarditis suggests that there may be inaccuracy when diagnosis is made on histology alone. Autoimmune markers may provide adjunct diagnostic tools and identify myocarditis patients in whom immunosuppression is of potential benefit.

1033-72 Preliminary Report of the Multicenter Giant Cell Myocarditis Study Group

Leslie T. Cooper, Gerald J. Berry, Ralph Shabetai. San Diego Veterans Affairs Medical Center, and University of California San Diego, San Diego, CA

Idiopathic Giant Cell Myocarditis is a rare and frequently fatal disease of unknown cause which has only been reported in isolated cases and two small series. Accordingly, research on this disease is hampered by a lack of data. In January 1995, we established a multicenter study group to better address the many unresolved questions about the natural history and treatment of idiopathic giant cell myocarditis. Direct mailing to cardiovascular centers worldwide and journal announcements produced 42 cases ranging in age from 39 days to 70 years. Most present with congestive heart failure or sudden, and 11 age-matched control hearts. These 53 patients from the Myocarditis Treatment Trial (35 male, aged 42 ± 15 years). Quantitative angiography was performed at baseline (B), after ischemia, and after administration of 5 mg of ISDN, in proximal (LAD) and distal (LAD) segments of the left anterior descending coronary artery and proximal segments of left circumflex artery (CXY). No patients had clinical or EKG ischemic changes during H, and never was local or diffuse spasm detected.

1033-73 Arrhythmogenic Right Ventricular Dysplasia—Cardiomyopathy: A Form of Healing Myocarditis?

Allen Burke, Andrew Farb, Renu Virmani. Armed Forces Institute of Pathology, Washington, D.C.

To determine the role of inflammation and fibrosis in right ventricular dysplasia (RVD), we histologically evaluated 15 hearts from patients with RVD dying suddenly, and 11 age-matched control hearts. RVD was defined as right ventricular dilatation with focal fibrosis or thinning < 0.5 mm. Eight histologic sections were taken from each autowipe, spot to base, stained for collagen, and the degree of fat infiltration, fibrosis, and number of inflammatory foci quantitated. The mean age of RVD was 31 ± 10 years. 4 cases (27%) were familial, 6 patients had a history of arrhythmias (40%), and 8 deaths occurred in the acute phase (53%). Foci of inflammation were present in 14/15 cases (93%)(mean number 15 ± 5.2), and microscopic foci of left ventricular fibrosis and/or inflammation were found in 12 cases (80%). Inflammation correlated negatively with age of the patient (p = 0.02, r = 0.4), and fibrosis showed a positive correlation with age (p = 0.008, r = 0.5). Left ventricular fibrosis was greatest in the base of the free wall (12 ± 3%) and least in the ventricular septum (6.6 ± 0.9%, p = 0.03). Fibrosis was diffuse in the right ventricle (mean 20 ± 5%) without predilection for site. No differences in the degree of fibrosis or inflammation were noted with respect to family history, exercise at death, or history of arrhythmias. Compared to controls, RVD was hearts had greater fat (p = 0.001) and inflammation (p = 0.0001), but there was no significant difference in degree of fat infiltration of the right ventricle (34 ± 4.8% RVD vs. 23 ± 5.6% control, p = 0.1). RVD is an inflammatory disease that progresses to fibrosis and often involves both ventricles; fat infiltration is a secondary phenomenon.

1033-74 Arrhythmogenic Right Ventricular Cardiomyopathy: Dysplasia, Dystrophy or Myocarditis?

Cristina Basso, Domenico Corrado, Marialuisa Valente, Annalisa Angelini, Andrea Nava, Gaetano Thiene. University of Padua, Italy

The aim of the investigation was to elucidate the nature of the pathobiological events underlying arrhythmogenic right ventricular cardiomyopathy (ARVC). 30 hearts with ARVC were studied (20 M, 10 F, aged 15–68 years, mean 28). Source of specimens was autopsy in 27 and cardiac transplantation in 3. Mode of death of autopsy cases was sudden in 24 and congestive heart failure in 3 (due to cerebral thromboembolism in 1). Previous symptoms in terms of syncope, palpitations or heart failure, were complained by 17 patients (57%). Basal ECG, available in 19, showed inverted T-waves in the right precordial leads in 15 (75%) and ventricular arrhythmias in 15 (75%). RV aneurysms were present in 15 hearts (50%), mostly located in the inferior wall. Involvement of the left ventricle was observed in 14 cases (47%) and in 6 of them was extended to the ventricular septum. Scattered foci of T-cell lymphocytes with myocyte necrosis were present in 20 cases (67%). On the basis of the histopathological substrate, we observed a fatty (40%) and a fibrous-fatty (60%) patterns. The fibrous-fatty pattern showed a higher incidence of ventricular arrhythmias (p = 0.05), a thinner RV wall (p < 0.0001), and a higher occurrence of focal myocarditis, left ventricular involvement and RV aneurysms (p = 0.001). In conclusion, myocardial atrophy observed in the fibrous-fatty variety of ARVC, appears to be the consequence of an acquired injury (necrosis) and repair (fibrous-fatty replacement) progressive process, mediated by patchy myocarditis; whether inflammation is a primary event or reactive to spontaneous necrosis remains obscure. A programmed cell death or apoptosis in the setting of postnatal involution of the RV might be considered.

1033-75 Chagas’ Heart Patients Without Cardiac Enlargement Have Impaired Epicardial Coronary Vasodilation but No Vasotonic Angina

Marcus V. Simões, Elias M. Ayms-Neto, J. L. Attab-Santos, B. C. Maclel, J. A. Marín-Neto. University of São Paulo, Ribeirão Preto, Brazil

Myocardial perfusion defects are detected in many Chagas' heart patients (CHP) and coronary spasms has been postulated to cause these disturbances. In pts with vasotonic angina (VA), hyperventilation (H) provokes vasoconstriction responsive to nitrates. Aim of this study was to measure vasomotor epicardial coronary responses to H and hypoxia to chagasic coronary (ISDN), in 24 CHP (age 56 ± 9, 14 M), 15 with (D-CHP) and 9 without (ND-CHP), and 9 VA pts with angiographically normal coronary arteries (age 45 ± 9, 3 M). Quantitative angiography was performed at baseline (B), after H, and after administration of 5 mg of ISDN, in proximal (LAD) and distal (LADD) segments of the left anterior descending coronary artery and proximal segments of left circumflex artery (CXY). No patients had clinical or EKG ischemic changes during H, and never was local or diffuse spasm detected.