4:30

ULTRASTRUCTURAL STUDY OF RESTENOSIS AFTER DIRECT PTCA

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Direct PTCA is more commonly chosen as a treatment for acute myocardial infarction(AMI) than thrombolysis but its mechanism of restenosis is unknown. In order to disclose the mechanism of restenosis after direct PTCA, we transmission-electron-microscopically investigated 125 areas taken from PTCA sites in 6 necropsy subjects who died immediately(2,4 and 6 days) and early(28, 36 and 68 days) after successful direct PTCA for AMI in comparison with 12 elective PTCA subjects. In the immediately subjects after direct PTCA, we found plaque rupture with haemorrhage and mural thrombosis without intimal proliferation of smooth muscle cells(SMCs). By transmission-electron-microscopy(TEM),there were many migrating SMCs from the media into the intima, based upon a morphological findings in which cells stretched into the intima through fenestrae of the intimal elastic lamella, and many activated macrophage cells were seen in the intima. In the early subjects after direct PTCA, we found more remarkable intimal proliferation of synthetic type cells of SMCs than in elective PTCA, and many macrophage cells modurated to the "form" cells.
We concluded that proliferation of migrating SMCs would be the cause of restenosis after direct PTCA and activity of SMCs after direct PTCA seemed to be higher than after elective PTCA.

5:00

EMERGENCY CORONARY STENTING FOR ACUTE DISSECTION DURING PTCA.

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In a series of 2100 patients (pts) undergoing PTCA from March 89 to August 90, 72 pts (3.4%) who experienced ischemic complications due to acute coronary dissection were treated with emergency implantation of a Palmaz-Schatz stent (S). Of 57 men and 15 women with a mean age of 58.7 years (range 35-76 yrs), 2 had silent ischemia, 25 had stable angina, 44 had unstable angina, 1 had an acute anterior myocardial infarction (MI) treated with thrombolysis 24 hours before PTCA. Mean ejection fraction was 57.7% (range 40-70%). 35 pts had multivessel disease. Dissection involved 76 vessels : left anterior descending artery in 35 pts, left circumflex artery in 14 pts, right coronary artery in 26 pts, saphenous vein graft in 1 pt. 101 S (1.4 S/pt) were successfully delivered in 72 pts. Major complications occurred in 4 pts (5.6%) : 3 thrombotic occlusion in the first 24 hours following implantation, one late closure 5 days after the procedure. 2/4 pts had repeat PTCA and coronary surgery and the other 2 were recanalized with PTCA. At follow-up (1-16 months, mean 5.3 months), 58 pts were symptom free, 6 pts had stable angina, and 4 pts unstable angina. Angiographic follow-up obtained in 27 pts revealed no late closure and restenosis in 9 pts (33%): 4/19 pts (21%) who received single stent and 5/8 pts (63%) who had multiple stents implanted. We conclude that implantation of balloon expandable stents is a safe and effective alternative to emergency bypass surgery for the treatment of acute dissection following PTCA.

4:45

HISTOLOGIC PREDICTORS OF RESTENOSIS AFTER DIRECTED CORONARY ATHERECTOMY

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The morphology of tissues excised from 127 arterial lesions by directed coronary atherectomy (DCA) was correlated with the presence or absence of restenosis on follow-up quantitative angiography. Angiography was performed at 6 months after DCA or when symptoms recurred. Stepwise logistic regression analysis was used to determine if intimal hyperplasia, atherosclerotic plaque, high lesion cellularity, thrombus, calcium, lipid, media, or adventitia in resected tissues were associated with subsequent restenosis. The measured aggregate lengths of media and adventitia present were also correlated with the likelihood of restenosis.
Thirty four of 90 (38%) restenotic sites (REST) had renarrowed at follow-up angiography as compared to 12 of 37 (32%) primary (PRIM) stenoses treated by DCA (p>.05). For the REST group only, the presence of media in excised tissues was associated with a higher likelihood of restenosis after DCA (odds ratio 1.22, p=.02), and each 1 mm increase in aggregate length of media increased the odds of restenosis by 22%. Calcium in REST lesions reduced the odds of restenosis by a factor of three (odds ratio 0.31, p=.02). For the PRIM group, only lesion smooth muscle cell density was associated with greater odds of restenosis, with moderately cellular lesions being 8 times more likely to renarrow than minimally cellular lesions (odds ratio 8.21, p=.02). The presence or absence of atherosclerotic plaque, intimal hyperplasia, thrombus, lipid, and adventitia had no correlation with outcome.
Conclusions
Excising media, especially long segments, from REST sites during DCA may increase the risk of restenosis. Calcified REST lesions are less likely to renarrow than are noncalcified lesions. PRIM stenoses with moderate cellularity are at greater risk for restenosis than are minimally cellular lesions.

5:15

HISTOLOGICAL FEATURES 3 TO 320 DAYS AFTER STENTING OF HUMAN SAPHENOUS VEIN BYPASS GRAFTS.

H.M.M. van Beusekom, P.W. Serruys, W.J. van der Giessen, B.H. Strauss, P.J. de Feyter, H. van Loon, R.J. van Suylen, H. Suryapranata, M.J. van den Brand. Dept. of Cardiology, Thoraxcenter, Erasmus University Rotterdam, The Netherlands

The histopathological changes after stent implantation in human saphenous vein bypass grafts (SVBG) have not ye. been reported. We studied 10 SVBG's, of which 7 were obtained surgically in addition to atherectomy biopsies performed in 3 separate stents. The surgical specimens were resected at 3 days to 10 months following stent placement. Thrombosis within the stents or necessity to interrupt the aggressive anticoagulant treatment were the indications for early surgery. In these specimens the underlying graft tissue protruded through the stent mesh. The stent wires were covered by massive amounts of platelets, fibrin and numerous white blood cells, and particularly at branching points. At 3 months the stents were found incorporated within a neointima consisting of thrombus remnants, a disorganized fibrocellular layer and at the luminal side an endothelium covered organized layer of smooth muscle cells. Morphology of the endothelial lining varied greatly and there was a variable amount of neointimal hyperplasia, mainly caused by differences in the amount of matrix material. The 3 atherectomy procedures were for symptomatic restenosis within the stent at 4 and 5 months. In these specimens smooth muscle cells, confirmed by immunological staining, were present in several areas and associated with an extracellular collagenous matrix. In conclusion: histopathological observations in 10 SVBG's showed that the endothelial lining resembled that on atherosclerotic plaque. A significant amount of fresh or old thrombus was associated with the stent wires. These data however do not discriminate between thrombus organization or primary smooth muscle cell proliferation as the cause for excessive neointimal hyperplasia.