0820
10:00 h

HAEMODIALYSIS WITHOUT HEPARIN, A POSSIBLE BENEFIT FROM THE USE OF TICLOPIDINE (TCP) IN END STAGE RENAL DISEASE HAEMODIALYSIS (HD) PATIENTS. C. Mion, G. Chong, Q.V.Nguyen. Service de Néphrologie, University Hospital, Montpellier, France.

In order to test the efficacy of TCP as an anti-thrombogenic agent during HD, we attempted to suppress heparin in 10 stable end stage renal disease patients, treated for 4 months or more, and taking a 500 mg daily dose of TCP and no other anticoagulant therapy. The dialysis schedule was: 3 weekly 3-5 hours HD; single use Cordis HFAK IV dialyser, 1.3 m^2 surface area; heparin dosage: 25-66 units per kg body weight per hour given 50% intravenously as a loading dose and 50% as a continuous infusion during dialysis. Two protocols were followed.

In protocol I, the maintenance dose was progressively reduced (10% of the initial dose every 3 HD) down to complete suppression; thereafter, the loading dose was progressively reduced and eventually suppressed. Reduction in dialyser fiber bundle volume (ΔFBV) was measured on each dialyser. When FBV was >15%, heparin dosage was maintained at the lowest level obtained. In 3/5 patients, total suppression of heparin was obtained and 25 dialyses without heparin were realised (6, 10 and 9 dialyses in each patient); dialyser efficiency was maintained despite formation of heparin deposit on the outlet blood chamber of the dialyser. In 3 other patients, total heparin dose was reduced by 75%.

In protocol II, progressive reduction of heparin dosage was done simultaneously on loading and maintenance doses; ΔFBV was also measured in each dialysis and heparin was maintained at a level compatible with ΔFBV <10%. In 5 patients, a 70-80% decrease in heparin dosage was obtained without reduction in dialyser performance.

In conclusion, TCP made possible HD without heparin and allowed a striking reduction in heparin dosage without loss of dialyser performance.

0821
10:15 h

EFFICACY OF DIFFERENT HEPARIN REGIMENS IN DIALYSIS AS ASSESSED BY RADIOIMMUNOASSAY FOR FIBRINOGEN/FIBRIN SPLIT PRODUCTS B. Kudryk, S. Wilhelmsson, C. Netré, D. Robinson and M. Blombäck. Karolinska Hospital, Stockholm Sweden and The New York Blood Center, New York, U.S.A.

Three different heparin regimens were employed to control clotting in uremic patients undergoing hemodialysis. The regimens differed with regard to dose prior to and/or during dialysis. The efficacy of heparin was checked with several parameters including blood levels of fibrinogen/fibrin split products. Blood was collected from each patient prior to and at three time points during dialysis. Samples were taken from both the blood inlet and outlet sides of the dialyser. Fibrinopeptide A (FPA) was determined by radioimmunoassay (RIA). Hi-2DSK (an Aα-chain derived split product) levels were measured by RIA using selected chromatography fractions of patient plasma and a new RIA method was used to measure plasma levels of split products which contain the Bβ 15-42 sequence.

FPA levels were lower during heparin infusion but rose after it was terminated. Blood from the outlet efferent side of the dialyser usually showed higher FPA levels. Hi-2DSK and Bβ 15-42 concentrations were always high before and during dialysis, sometimes 10-20 times or more that found in normal subjects. However, they never showed the variation observed for FPA. Also, in most cases, the levels were similar regardless from which side of the dialyser the samples were obtained.

All three regimens were equally as effective in preventing FPA generation in the dialyser. FPA estimation may be used to check adequacy of heparin dosage. Assays for the other two split products indicate no significant elevation of fibrino(geno)lysis during four hours of dialysis.

0822
10:30 h

PLATELET INHIBITORY DRUGS IMPROVE COLLATERAL BLOOD FLOW AFTER AORTIC THROMBOSIS. R. Schaub, C. Helenski, K. Gates, and R. Roberts. College of Veterinary Medicine, The University of Tennessee, Knoxville, TN, U.S.A. 37901.

Complete occlusion of the feline caudal aorta with a thrombus inhibits opening of collateral blood vessels to the hindlimbs. Our previous studies suggest this inhibition results from platelet activation. To test this hypothesis we evaluated the effect of indomethacin (I), aspirin (ASA), and prostacyclin (PGI) on collateral inhibition. Prothrombin times (OSPT), thromboplastin times (PTT), fibrin monomer (FM), and platelet counts were measured in 20 cats before the aortas were occluded by thrombosis. Five cats were untreated. Treatment of the others was started 1 hour prior to thombosis. Five cats received I (20 mg/kg i.v.), 6 cats received ASA (650 mg orally), and 4 cats received an i.v. infusion of PGI (50 ug/kg/hr). Cats were maintained 3 hours post-operatively. Coagulation parameters were measured after the 3 hour period and calculated as a percent of pre-thrombosis values. Collateral circulation was assessed as the time necessary for contrast media to appear in the iliac arteries caudal to the thrombus during aortography. All treated groups were statistically compared to the non-treated group. The OSPT, PTT, and FM values were not significantly changed in any group except for a prolonged PTT in the ASA group. Platelet counts were significantly reduced in non-treated cats (66 ± 4%) compared to treated cats I (96 ± 3%), ASA (97 ± 10), and PGI (97 ± 9%). Contrast media did not appear in the iliac arteries of non-treated cats until 8 seconds injection. In 4 I treated and all ASA treated cats contrast media appeared in the iliac arteries within 2 seconds after injection. The PGI treated cats had an intermediate response. Contrast media appeared in the iliac arteries 4 seconds after injection. These results suggest: (1) Some consequences of arterial thrombosis may be due to humoral inhibition of collateral blood flow. (2) This inhibition is related to platelet activation with release of vasoactive and aggregating agents and not to gross alteration of other clotting factors. (3) Platelet thromboxane A_2 may be the factor promoting collateral inhibition since prostaglandin synthesis inhibitors and antagonists are effective. (4) Inhibition of platelet function prior to thombosis could reduce ischemic damage by enhancing development of collateral blood vessels.

0823
10:45 h

INTERVENTIONAL RADIOGRAPHY IN RENAL ARTERY THROMBOSIS. A. Zajko, M.P. Solomon, E.J. Ring, R.A. Grossman, C.F. Barker, L.J. Perloff. Departments of Surgery & Medicine, University of Pennsylvania, Philadelphia, PA.

Recent reports of successful revascularization of kidneys sustaining prolonged warm ischemia secondary to acute renal artery occlusion prompted the following clinical trial in a renal allograft recipient: During treatment for a mild rejection reaction 19 days after receiving an HLA identical renal allograft, a 30-year old male suddenly developed oliguria with serum creatinine and BUN rising from 2.0-3.4 and 35-61 mg/dl respectively. DTPA technetium and I^{131} hippuran scans revealed functional activity and flow limited to the upper pole of the allograft. Arteriography showed multiple intrarenal emboli, a stenotic main renal artery distal to the internal iliac anastomosis, and complete occlusion of the lower pole artery. After transluminal dilatation of the stenosis with a 4 mm balloon catheter, the anastomosis was easily trasversed with coaxial catheters for infusion of streptokinase (5000U/hr) and heparin (800U/hr). Repeat arteriography 12 hours later showed a decrease in the amount of thrombosis. Streptokinase infusion rate was increased to 7500U/hr, and heparin was administered by continuous systemic infusion to maintain the PTT at 45-50. By manipulation of the catheters, thrombi in the major renal artery branches were maneuvered into the already occluded lower pole artery. At 36 hours arteriography revealed reopening of many of the distal occlusions. During this time fibrin degradation products were 10 μg/ml, thrombin time was 64 seconds and fibrinogen level was 235 mg/dl. Although the patient required four weeks of dialysis the urine output was maintained at >1000 ml/day, suggesting polyuric acute tubular necrosis. Five weeks after the occlusive episode, the main renal and upper pole arteries of the allograft were patent and serum creatinine was 3.5 without dialysis.

The previously poor results following acute renal artery occlusion in the renal allograft recipient may be improved with early interventional arteriography. This may provide marginal perfusion until surgical intervention ensues.