Poster Board P5-067

0719 FAILURE OF SUBCUTANEOUS LOW-DOSE HEPARIN TO PREVENT ARTERIAL EMBOLISM AFTER ACUTE MYOCARDIAL INFARCTION (MI)

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Low-dose s.c. heparin is recommended as alternative of oral anticoagulants during the hospital phase after MI. No figures are available on pulmonary embolism (PE) and arterial embolism originating from mural thrombosis is not mentioned. During 22 months 214 patients with MI were admitted to the CCU. Heparin was given in infusion (15-30000 U/24h) for 3-4 days, followed by twice daily 5000 U s.c. 5 patients had acute arterial occlusions, all while on s.c. heparin, 4 of them underwent embolectomy of the femoral artery, one had to be amputated. An additional patient had PE documented by lung scan. One patient had a drop of the platelets to 30000/mm3 at the time of femoral artery occlusion, the count normalizing after discontinuing heparin. In the Cooperative Clinical Trial of Ebert et al. (JAMA 225:724, 1973) arterial embolism into the lower extremities was found in 6 of 499 untreated patients with MI, in none of 500 on oral anticoagulants. Thus, 5 episodes of arterial occlusion in our 214 consecutive patients with MI indicate that s.c. heparin does not prevent arterial thromboembolic complications. Whether occlusions are due to embolism from mural thrombus or to local thrombosis in connection with heparin-induced thrombocytopenia as possibly in one of our cases, remains to be established.

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0720 VALUE OF LOW DOSE HEPARIN IN PREVENTION OF DEEP VENOUS THROMBOSIS AFTER ACUTE MYOCARDIAL INFARCTION

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Low-dose heparin has become widely used in the prophylaxis of venous thromboembolism in surgical patients but its use in medical patients is less well established. In particular, the reported incidence of deep venous thrombosis (DVT) after acute myocardial infarction (AMI) has varied considerably, as has its response to low-dose heparin prophylaxis. In 94 patients admitted with a provisional diagnosis of AMI, treatment was allocated randomly and blindly as either heparin 5,000 U subcutaneously twice daily or placebo. DVT was diagnosed by daily 125-I fibrinogen leg scanning. DVT occurred in 10% of control patients and in 3% of treated patients. All DVT's occurred in patients with subsequently proven AMI (68% of admissions) in whom the incidence was 19% in those untreated and 5% in those treated. DVT was more common in patients with cardiac failure (14%) than in those without (3%). There was no difference in incidence of DVT between patients with or without clinically significant arrhythmias or hypotension. Multivariate analysis combining these factors failed to improve the prediction of DVT. It is concluded that patients admitted for acute Coronary Care are a relatively low risk group for DVT. Minidose heparin appears to be warranted only in those with proven AMI and cardiac failure.

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0721 THE EFFECT OF CONTRAST MEDIA, ^{125}IODINE-LABELLED FIBRINOGEN, HEPARIN AND ASPIRIN ON PLASMA BTG CONCENTRATION

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Betathromboglobulin, a platelet-specific protein, released on activation of platelets is being evaluated as a marker of active thrombosis. Theoretically, several diagnostic and therapeutic procedures used in the thrombotic disorders might affect BTG by inducing platelet release or by interfering with the radioimmunoassay (RIA) method. This study determined the effect on plasma BTG concentration of contrast media used in venography, I^{125}-labelled fibrinogen for leg scanning and heparin or aspirin used in the management of venous thrombosis. Mean plasma BTG in normals was 28 ± 8 ng/ml. (n = 70), and do not fluctuate. Plasma BTG in 10 patients with normal venogram measured before and 30 minutes post-venography was 23 ± 10 ng/ml. and 26 ± 15 ng/ml. respectively ($p > 0.1$). ^{125}I fibrinogen in plasma by coprecipitating in the BTG/antibody complex, elevates bound radioactivity in the RIA and results in spuriously low levels of BTG and may preclude its use as a diagnostic test in patients having leg scanning. In-vitro, heparin (> 50 units/ml.) or aspirin (> 40 mg/100 ml.) affect the 1 hour incubation assay and give falsely high BTG levels but do not affect an optimized assay with 24 hours incubation. This study indicates that diagnostic and therapeutic measures in venous thrombosis patients may influence BTG measurement and caution is necessary to avoid confusing the results.