Coagulation and Platelet Changes in Renal Diseases

Hungerford Room

Time
12.15

0991 NEPHROTIC SYNDROME (NS): EVIDENCE FOR INCREASED PLATELET PROSTAGLANDIN SYNTHESIS.
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NS is associated with an increased incidence of arterial and venous thrombosis. Hemostasis was evaluated in 11 children with NS (6 with active disease Grp I, and 5 in remission Grp II). Studies included assays for fibrinogen, FDP, Antithrombin III (AT III), platelet count and aggregations. Platelet malonyldialdehyde (MDA) in the presence of N-ethyl maleimide (NEM) or thrombin was used as an indicator of endoperoxide formation, and platelet survivals were performed in 3/11. Platelet hyperaggregability was present in Grp I and was associated with significantly increased platelet MDA in the presence of both NEM (4.0 ± 0.29), or thrombin (1.77 ± 0.32) compared to normal controls (3.20 ± 0.26; 1.26 ± 0.18). Other evidence for a "hypercoagulable" state included a marked reduction in plasma AT III levels to 9.4 ± 3.8 (controls 24 ± 3 mg/100 ml), and a reduction in platelet life-span in both children in Grp I in whom this study was performed ($T_{\frac{1}{2}}$ of 2.1 and 2.5 days). Grp II patients did not demonstrate platelet hyperaggregability and platelet MDA was normal (3.21 ± 0.4; 1.13 ± 0.19). AT III levels were normal at 26.5 ± 4.8 mg/100 ml, and platelet life-span was normal in 2/2 children ($T_{\frac{1}{2}}$ of 3.6 and 4.4 days). The normal half-life of 4.4 days was obtained in the same child in whom a $T_{\frac{1}{2}}$ of 2.5 days was present during active disease. Since a reduction in platelet survival is associated with an increased risk of thromboembolism in a number of pathological states, this finding is of clinical significance and may identify the patient with NS who is at risk. Platelet hyperaggregability in this syndrome is prostaglandin related, and appears to be due to an increase in platelet endoperoxide formation.

13.00

0992 PLATELET AND PLASMA SEROTONIN IN GLOMERULONEPHRITIS

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Intra-platelet and free plasma serotonin levels can be measured easily on casually collected samples of ACD-blood. Platelet serotonin levels were depressed and plasma serotonin levels were raised in progressive immune-complex mediated glomerulonephritis (i.e. patients with disease resulting in renal failure), but were normal in non-progressive forms of glomerulonephritis. Platelet serotonin levels correlated well with the immunological activity of the disease and to a lesser extent with renal function in the groups and in individual patients studied serially, particularly in SLE nephritis. Platelet serotonin levels also correlated with serum-platelet aggregating material (believed to be immune complexes) and with Raji cell-detected circulating immune complexes. Uptake of ^{14}C-serotonin by platelets in vitro did not correlate with platelet serotonin levels in progressive primary glomerulonephritis, but did so in SLE nephritis.

Platelet serotonin assay provides further evidence of platelet involvement in the pathogenesis of glomerular injury, suggests platelet activation in the circulation, and provides an easy measure of this involvement in relation to the clinical and the immunological disease activity.