Uric Acid Metabolism in Hyperuricemic Hypertension

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The metabolism of uric acid in hypertensives with unexplained hyperuricemia was studied in comparison to that in gouty and/or normal subjects.

Materials and Methods: Materials were three hypertensives with hyperuricemia, four gouty and two normal men. The plasma (P_{ur}) and the urinary (U_{ur}) uric acid was assayed by a spectorphotometric method utilizing phosphotungstic acid. Renal clearance of creatinine (C_{cr}) and uric acid (C_{ur}) were measured at the same time. From the values thus obtained, the following parameters were calculated: glomerular filtered load of uric acid ($F_{ur}=P_{ur}\times C_{cr}$), and net tubular reasorption rate of uric acid ($T_{ur}=F_{ur}-U_{ur}\times V$). Ten μCi of uric acid-2-^{14}C was injected intravenously as lithium urate. For the following seven days, each 24-hour urine was collected. Radioassay of the uric acid isolated from the urine was performed in a liquid scintillator with the use of CO_2-oxidation method. The total amount of ^{14}C excreted in urinary uric acid was compared with the amount of ^{14}C injected dose as uric acid to determine the per cent of the dose recovered as urinary uric acid. The miscible pool and daily turnover of uric acid were calculated. The de novo synthetic rate of uric acid was studied with glycine-(U)-^{14}C administered orally.

Results: (1) Plasma uric acid level of gout ranged from 7.3 to 14.0 mg/dl., that of hypertension from 8.2 to 11.5 mg/dl., and that of normal subjets from 2.8 to 5.0 mg/dl. (2) There was large miscible pool in hypertension, the amount of which matched that of gout. (3) The synthetic rate of uric acid was not elevated in hypertension. (4) The cumulative urinary uric acid-^{14}C recovery was very low in hypertension, and in this respect, hypertension was different from gout. (5) Despite of normal range of C_{cr}, C_{ur} was distinguishably low in hypertension. The low C_{ru} in hypertension was attributable to elevated T_{ur}, as F_{ur} was larger in hypertension than in the two.

Conclusions: The results suggest that hyperuricemia in hypertension can not be attributed to oevrproduction of uric acid, and it is concerned with some specific type or renal tubular disturbances associated with hypertension. The relationship between hypertension and the tubular disturbance remains yet to be further elucidated.

VI. Liver, Biliary tract and Spleen

Scintigram of Liver Injury Localized in Irradiated Area (1)

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Six cases, which showed partial liver injuries on liverscintigram caused by radiation therpy to the neighbouring organs of the liver, were presented.

Each scintigram showed filling defects on the irradiated area. Scintigrams were taken 1 month to 2 years and 3 months interval after irradiation. Total dose was 5000-7000 R in each. Three cases were proved histologically. Pathologic specimen revealed the three

major finding, i.e. 1) congestion of liver acini around the central veins and dilatation of the sinusoids 2) fibrosis surrounding the central veins. On the other hand, fibrosis of Glisson's sheath was not remarkable, and liver cells of periacinal area were almost normal. The auther thought that so called "radiation hepatitis" (liver injuries following radiation therapy) might be caused by irradiation and some other factors. However, the irradiation must be the main factor of the above described findings of the liver.

Fibrosis surrounding central veins might be inhibited, followed by blood congestion in sinusoid. Blood congestion brought about a degeneration or disappearance of liver-parenchymal cells, or inhibition of regeneration of liver cell from the surrounding area of acini. Chronic partial congestion of the liver must be one of the most impportant factor of radiation injury of the liver.

We could not find any reactive proliferation of Kupper's star cells, and it was reason why filling defect appeared on liver scentigram.

Scintigram of Liver Injury Following Radiation Therapy (2)

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Purpose: We pursued process of an appearance of filling-defect of the scintigram of the liver following irradiation.

Method: 1000-4000 R does were given to the right lobe of each dog.

^{131}I-Rose-Bengal and ^{198}Au-colloid were injected intravenously, and uptake curve was recorded over the irradiated area and the other side. The scintigrams were analyzed as compared with hystological study.

Results: At the early stage after irradiation, scintigram showed increased up-take, and at the late stage decreased up-take and defect at the irradiated area. Maximum count ratio of the irradiated area and the other side showed similar changes. However, hepatic blood flow was similar changes at the irradiated area and also the other. Some cases, which irradiated small portion of liver by radiation therapy to the neighboring organs of the liver, were showed similar changes of scintigrams and up-take curve of dogs experiments. Although liver blood flow of the irradiated area was different from the other side.

Histologically, at the early stage after irradiation, hepatic and Kupper cells were enlarged and contracted sinusoides. At the late stage, fibrosis surrounding the central veins and liver acini congestion appeared. Characteristically same histologic appearance at the irradiated area and the other side.

Conclusions: Radiation to the liver showed hyperfunction of the liver and Kupper cells at the early stage of postradiation stadium.

But liver injury appeared at the late stage. Mechanism of liver injury following irradiation was briefly discussed.

Enlargement of liver and Kupper cells→ Stenosis of sinusoides→ Insufficiency of blood suply at the center of lobulus→ Fibrosis of the surrounding the central vein→ Congeston of sinusoides→ Decrease of hepatic blood flow→ Hypofunction and destruction of hepatic and Kupper cells.