

G. Blood, Bone Marrow, Spleen and Reticuloendothelial System

Evidence for an Extravascular Catabolic Pathway of Human IgG

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IgE binds to basophilic leukocytes and mast cells and upon addition of antigen or anti-IgE results in the release of histamine or slow-reacting substance of anaphylaxis, which mediate immediate type of allergic reaction. IgE is catabolized at a greater rate than any of the immunoglobulins. To answer the question of whether IgE has an additional catabolic pathway, namely extravascular catabolism, metabolic studies of radioiodinated IgE along with the other four major immunoglobulin classes were performed in 26 patients with non-allergic diseases and two normal volunteers.

The data of IgE metabolism were analyzed first using computerfitting to compartmental models with and without an extravascular catabolic pathway. The IgE data was fit successfully to the former model. The data of IgE and other immunoglobulins were then analyzed with the method of Nosslin, which provides an intercept

value from plots of calculated values derived from the radioactivities in the serum and urine at different time points. If the intercept value is equal to one, there is no extravascular catabolic pathway, whereas if it is less than one, there is extravascular catabolism. The mean \pm SD of the intercept values of IgE was 0.68 ± 0.15 , which was significantly less than one ($P < 0.001$). The mean values of IgD was 0.80 ± 0.11 , which was also significantly less than one ($P < 0.01$). That of IgG was 0.92 ± 0.05 ($P < 0.02$). In contrast, the mean intercept values of IgA and IgM were 1.14 ± 0.12 and 1.20 ± 0.31 respectively, which were not significantly different from one ($P > 0.02$).

These results indicate that there is definite extravascular catabolism of IgE and possibly IgD. It is reasonable that the extravascular catabolic pathway of IgE is related to the unique biological as well as metabolic behaviors of IgE.

Localization of the Active Marrow Determined by Superimposing the Marrow Scintiphoto Image on the Bone X-Ray Picture in Reference to the Pattern of Periphery Extension

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Since the bone marrow image often exhibits a complex and ambiguous figure, the localization of the active marrow was exactly determined by superimposing the marrow scintiphoto image over the bone X-ray photofilm. The active marrow was delineated in the life-sized scintiphotograms using ^{99m}Tc sulfur colloid.

In those cases which displayed a pattern of periphery extension in the marrow distribution, difference was noticed in the radiocolloid distribu-

tion in the long bone between the epiphysis and metaphysis bounded by an epiphyseal fusion line.

In the infantile cases of hereditary spherocytosis, the active marrow was distinctly observed in the epiphyseal nuclei. When the cases were adult and/or developed mild hemolysis, the active marrow appeared to have diminished selectively in the epiphyseal part of both proximal and distal ends of the humerus and femur and that of proximal end of the tibia and fibula. The process was

evidently observed in the centralization phase of the marrow distribution after the splenectomy, in which the epiphyseal marrow disappeared prece-
dently to that in the neighbouring metaphysis.

The counter phenomenon was observed in chro-
nic myelogeneous leukemias in which the epiphyseal
marrow was more manifest than metaphyseal one
especially so in those in the terminal stage.

As we had previously reported, a periphery
extension factor and centralization one was deduc-
ed by principal component analysis on the 28
values of the local marrow activity. Intensity of
the epiphyseal active marrow was referred to the
score of these two principal components.

In chronic myelogeneous leukemias, prominent
epiphyseal marrow was present in no relation to
the degree of periphery extension nor to that of

central depression. In hereditary spherocytosis,
on the other hand, the more the score was of
periphery extension with central hyperplasia, the
more evident the epiphyseal marrow was.

These findings suggest that the epiphyseal
marrow behaves different from the metaphyseal
one independently on being proximal or distal
to the trunk. They also suggest that a different
regulatory mechanism exists to determine the
distribution pattern of the active marrow between
these two groups which develop apparently the
same pattern of periphery extension. To clarify
this mechanism, detailed and definite observation
on localization of the marrow is necessary and
this double image method using superimposing
technique is considered to be valuable for this
purpose.

The Concentration of Radioiron and Chromium in Pulmonary Hemosiderosis

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Case 1.

Seven μCi of ^{59}Fe was injected intravenously
to a 4 year old girl with pulmonary hemosi-
derosis.

Radioiron accumulation was visualized mostly
in the right lung 20 days after intravenous radio-
iron injection.

Case 2.

One hundred and fifteen μCi of ^{51}Cr labelled
red cell was injected into a 9 year old boy with
pulmonary hemosiderosis in remission for 8 months
after iron therapy. The scintillation camera image
showed the concentration of radiochromium in
the both side of the lung 13 days after intravenous
radiochromium labelled red cell injection.

These images were better in prone than in
supine position. The clinical laboratory findings of

these two cases were as follows.

Case. 1. ^{59}Fe -scan

SI=49 $\mu\text{g/dl}$, Hb=10.6 g/dl, Ht=36.5%,
RBC=514 $\times 10^4$, PIT=1.1 mg/kg/day, PID=
18 min., RCU=92%, Solenomegaly +, Phlegm:
Macrophages with hemosiderin. Radiographic
diagnosis: Pneumonitis.

Case 2. ^{51}Cr -scintiphoto

Nov. 1975 Before Fe therapy

SI=18 $\mu\text{g/dl}$, Hb=6 g/dl, Ht=21%, RBC=
284 $\times 10^4$, Ret=1.2%, Phlegm: Macrophages
with hemosiderin.

Feb. 1976 After Fe therapy

SI=130 $\mu\text{g/dl}$, Hb=14.5 g/dl, Ht=42%,
RBC=510 $\times 10^4$, Ret=0.2%.

Scintigram was taken in Sept. 1976.