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ACUTE UNDIFFERENTIATED LEUKEMIA WITH MARKED EOSINOPHILIA IN A CHILD.

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A case of acute undifferentiated leukemia in an 8-year-old child is reported. The presenting feature of the disease was a marked peripheral eosinophilia (80%) with immature and somewhat atypical eosinophils. The bone marrow showed prevalence of large blast cells with scarce basophilic cytoplasm and one or more nucleoli. The rate of eosinophils was increased. The presence of parasitic infections or allergic diseases was excluded. Two short term remissions were obtained. No eosinophilia was observed during the subsequent relapses. Death ensued after 17 months.

Differential diagnosis with the eosinophilic leukemia, the hypereosinophilic syndromes and the eosinophilic leukemoid reaction is done. The significance of marked eosinophilia in acute leukemia is discussed, mainly as an initial response to some still poorly defined antigenic stimulus of the leukemic cells. Its relationship to the so called tumor associated eosinophilic factor is also taken into account.

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ORAL AND PARENTERAL IMMUNIZATION TO BOVINE SERUM ALBUMIN IN THE PRESENCE OF PASSIVE ANTIBODY.

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The effect of passive antibody (p.a.) on the antibody response to ingested Bovine Serum Albumin (BSA) was compared to intravenous (i.v.) and subcutaneous (s.c.) immunization. Antibody to BSA was measured by the Farr-technique and quantitatively expressed as µg BSA-N binding capacity/ml of serum (ABC 33). 18 rabbits, passively immunized to BSA from a pool of high titered antiserum had a mean ABC 33 of 17.2. Four of these animals received no antigen (group I), 5 were fed 0.1% BSA in water (gr. II), and 3 received 5 mg BSA s.c. (gr. III); 50 mg BSA was given i.v. to 3 animals (gr. IV) and 20 mg BSA i.v. to another 3 (gr. V). Control animals for groups II - V were actively immunized in the same way without prior administration of p.a. Catabolism of p.a. was similar in groups I and II (mean ABC 33 on day 25: gr. I 1.4 ± 0.5, gr. II 0.9 ± 0.6). Subsequently, serum anti-BSA in gr. II increased to 6.6 ± 5.7 on day 63 (control 3.6 ± 2.2, $p > 0.05$). In gr. III, mean ABC 33 was lowest on day 14 (2.2 ± 1.4) and increased to 51.7 ± 21.7 (control 9.4 ± 8.4, $p < 0.02$). In gr. IV mean ABC 33 increased from day 7 (0.4 ± 0.7) to 3.1 ± 2.8 (control 0.3 ± 0.5, $p < 0.05$). No active immunity developed in gr. V (control 0.17 ± 0.21). Confirming previous data, p.a. enhanced or suppressed parenteral immunization depending on routes of immunization and antigen/antibody ratios. In contrast, p.a. did not influence oral immunization and its catabolism was not affected by the ingested antigen.

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MEMBRANE FLEXIBILITY OF ENZYME-DEFICIENT ERYTHROCYTES

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The rheological properties of enzyme-deficient erythrocytes differ significantly. In glucosephosphate isomerase deficiency young as well as whole erythrocyte populations show a markedly increased rigidity and an abnormally strong attachment of hemoglobin to the inner surface of isolated membranes. Glucose-6-phosphate dehydrogenase-deficient erythrocytes are more flexible than normal erythrocytes. Even after acetylphenylhydrazin-induced Heinz body formation these cells are less rigid than normal erythrocytes incubated under identical conditions. The flexibility of pyruvate kinase deficient erythrocytes and reticulocytes is normal in a favourable surrounding. During incubation at low pH and low glucose concentrations especially the reticulocytes become highly rigid. The rheological properties of the enzyme-deficient erythrocytes explain why splenectomy improves the red cell lifespan in glucosephosphate isomerase and pyruvate kinase deficiency, but not in glucose-6-phosphate dehydrogenase deficiency.

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Hypogammaglobulinemia and HLA gene products.

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Two siblings of unlike sex from a Turkish family suffered from a severe hypogammaglobulinemia but with B-cells present in normal numbers. The serum immunoglobulin was mainly IgM, but contained no measurable antibody activity. The numbers of T-cells were severe reduced and did not proliferate when stimulated by antigens, while mitogenic stimulation gave normal responses. Peripheral blood lymphocytes appeared able to differentiate to IgM containing blasts only, when activated by PWM. HLA-antigens on lymphocytes were undetectable. HLA-A antigens were found in serum and on cultured fibroblasts. B2M was present only on B-cells using viable lymphocyte suspensions. In tissues and after fixation of cells B2M seemed also present on T-cells. The coincidence of absence of HLA-gene products with failing differentiation of B-cells to plasmacells suggests a role of these products in T-cell dependent immune responses.

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Detection of lymphoblasts from common-type ALL in the bone marrow with a double marker method.

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By screening of 400 sera of pregnant women in an indirect immunofluorescent test with T-lymphocytes and several EBV transformed B-cell lines, 3 sera with B-cellalloantibodies were detected. These sera did not react with T-cell-leukemia line Molt-4 and JM (1), but were reactive with 'Null'-cell-leukemia line KM-3.
For the purpose of a direct immunofluorescent test, the globulinfraction of these sera were first fluorescinated and then incubated simultaneously with a rhodamine-conjugated antiimmunoglobulin with bone marrow of children with ALL.
Two different types of cell population were observed:
1) one population with double fluorescence (probably B-lymphocytes and monocytes)
2) exclusively fluorescein-stained cells (predominantly blasts)

Even though the latter population does rarely occur in normal non-leukemic bone marrow, the method still seems to be helpful for the determination of the quality of a hematological remission.

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SERUM LIPIDS IN CHILDREN WITH ANEMIA.

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Serum cholesterol and triglyceride levels were determined in 17 children with various forms of anemia and varying rates of red cell production. Anemic patients had low serum cholesterol values, and a good correlation between hemoglobin and serum cholesterol levels was found. The serum triglyceride levels were below the mean of our reference material in 14 of the 17 patients, but there was a wider scatter than of the cholesterol values. No relationship could be demonstrated between the rate of red cell production as judged by reticulocyte counts, and serum cholesterol and triglycerides, respectively. These results and a few earlier studies indicate that all lipoprotein classes are reduced in individuals with uncomplicated anemias. This hypolipidemia can only partly be explained by the diluting effect of the increase in plasma volume accompanying anemia, and it is postulated that decreased lipoprotein synthesis may be a more important factor.