An Unusual Lymphomatous Disease Associated with Intracytoplasmic Crystals in Lymphoplasmocytoid Cells

By ARTHUR F. GOLDBERG

The finding of crystalline intracytoplasmic inclusions in an unusual lymphoplasmocytoid cell prompted this presentation. These cells had a nucleus resembling that of a lymphocyte and an abundant basophilic cytoplasm like that of a plasmocyte. The "crystal cells" were present in the bone marrow, lymph nodes and peripheral blood of an adult female with a clinical picture of a progressive wasting disease, characterized by generalized lymphadenopathy, moderate hepatosplenomegaly and a neuromuscular disorder. She also had a duodenal ulcer and hyperthyroidism. No specific pathological diagnosis has been made from the limited histological findings and no autopsy was obtained. The cytochemical characteristics of the crystalline inclusions are described separately.¹

REPORT OF CASE

E. H., a white unmarried governess, aged 60, was admitted to the Mount Sinai Hospital in July 1956. Past history: her mother was French, her father Syrian. She had always lived in the New York State area except for a short visit to Syria as a child. A tonsillectomy was performed at the age of 30 because of recurrent sore throats, an appendectomy at 35; she contracted a virus pneumonia at 51.

From December 1950 to March 1951 she was hospitalized elsewhere because of a weight loss of 30 pounds and marked muscular weakness and pains, especially in her legs. Physical examination at that time revealed a generalized nontender lymphadenopathy. The lymph nodes averaged 2 cm. in diameter. The spleen and liver were palpable 2 cm. below their costal margins. There was marked atrophy and weakness of all the muscles of the legs, particularly of the extensors. Laboratory examinations were as follows: hemoglobin 11.9 Gm. per 100 ml; erythrocyte sedimentation rate 21 mm. hr.; white blood count and differential, urine, BUN, fasting blood sugar, serum potassium and sodium, bromsulfalein excretion, glucose tolerance test, and 24 hour urine 11-oxy and 17-ketosteroids normal. Total protein was 9.3 Gm. per cent, with an albumin of 3.5 Gm. per cent and a globulin of 3.8 Gm. per cent. Serology revealed a 3 plus Kline and negative Wassermann and Mazzini tests. Cerebrospinal fluid had a protein of 132 mg. per cent, 13 white cells cu. mm. and a negative Wassermann test.

Gastrointestinal series, which showed a peptic ulcer one month previously, was normal. Chest x-ray showed some enlarged hilar lymphadenopathy. Electrocardiogram revealed a right bundle branch block. Biopsy of an inguinal lymph node was reported as showing "chronic lymphadenitis suggestive of Boeck's Sarcoidosis." Diagnosis on discharge was Boeck's Sarcoid with involvement of the spinal cord.

In the subsequent period, the patient's muscle weakness progressed to a point that

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Aided by the Albert A. List, Frederich Machlin and Anna Ruth Lowenberg Research Funds (Mt. Sinai Hospital) and by a U.S.P.H.S. Training Grant (2G-97) to the Department of Pathology, Albert Einstein College of Medicine.

The author is indebted to Drs. Louis R. Wasserman, Helen Wendler Deane and Alfred A. Angrist for constructive criticism of the manuscript.

Submitted June 23, 1960; accepted for publication Sept. 6, 1960.
walking was extremely difficult. She was treated with physiotherapy, prednisone, KCl and ACTH. A diagnosis of hyperthyroidism was also made and she received propylthiouracil and iodine. The specific amounts of the medications taken are not known and they do not appear to have altered the clinical picture.

Because of progressively increasing postprandial epigastric pains, nausea and vomiting over a three month period, she was admitted to the Mount Sinai Hospital in July 1956. All oral medications had been stopped one month previously by the patient herself.

Physical examination revealed: weight 110 lbs., height 5 ft. 1 in., temperature 98.6 F.; blood pressure 160/70, regular sinus rhythm of 100/min.; no lid lag or exophthalmus, Grade I retinopathy. Her skin was warm and dry but her palms slightly moist. There was a fine tremor of the outstretched hands. The left lobe of the thyroid was slightly enlarged and nodular. The heart was not enlarged. A faint apical systolic murmur was heard. The lungs were normal to percussion and auscultation. The liver was felt 2 cm. and the spleen 4 cm. below their costal margins. Generalized nontender, firm discrete or matted lymph nodes measuring 1-3 cm. were palpable. The patient walked with a wide gait. Her mental status and cranial nerves were normal. A slight symmetrical atrophy of most of the extremity muscles was present. This was particularly severe in the hands and calves. There was a generalized moderate to marked muscle weakness, the distal groups being weaker than the proximal. Deep tendon reflexes were slightly hyperactive in the upper extremities and hypoactive in the lower. Hoffman’s sign was positive bilaterally and there was a questionable left Babinski sign. No sensory or coordination defects were recognized. Rectal and vaginal examinations were normal.

Laboratory studies were as follows: hemoglobin 9.5 Gm. per cent, hematocrit 29.5 per cent, 3,500,000 red blood cells per cu. mm.; 4000 white blood cells per cu. mm., with 4 per cent bands, 53 per cent segmented neutrophiles, 36 per cent lymphocytes, 5 per cent monocytes and 2 per cent eosinophiles. Two normoblasts/100 WBC were seen. Reticulocytes were 1.5 per cent; platelets 180,000 per cu. mm. Direct Coomb's test was negative.

Tests for a bleeding diathesis, including the tourniquet test, bleeding time, clotting time, clot retraction, prothrombin time, prothrombin consumption, serum prothrombin activity, labile factor and fibrinogen level, were normal. The patient’s blood type was O Rh+.

Total protein was 6.9 Gm./100 ml., with albumin 3.7 Gm./100 ml. and globulin 3.2 Gm./100 ml. Serum paper electrophoresis showed a slight peak in the gamma range, with an albumin of 61.3 per cent, alpha-1 globulin 4.4 per cent, alpha-2 globulin 8 per cent, beta globulin 9.5 per cent, and gamma globulin 16.8 per cent. The formol gel test was negative. Erythrocyte sedimentation rate was 60 mm./hr. (Westergren). Alkaline and acid phosphatase were 7 and 2.3 Bodansky Units respectively. Serum calcium, phosphorus, sodium, potassium, chloride, CO2 combining power, cephaline flocculation, fasting blood sugar, Wassermann and two lupus erythematosus preparations were all normal.

Total protein-bound polysaccharides were 172 mg. per cent (normal 90-135), mucoproteins 107 mg. per cent (normal 40-70), acid precipitable globulin turbidity 10 units (normal 4-8), and zinc sulfate turbidity 6.5 units (normal 4-8).2 Repeat examinations of the last series were about the same. Total lipids were 875 mg. per cent, total cholesterol 212 mg. per cent, esters 153 mg. per cent, phospholipids 282 mg. per cent, and neutral fats 281 mg. per cent. Oil Red O staining of the paper electrophoretic pattern showed that the serum lipoproteins were distributed as follows: 9.2 per cent with the albumin and alpha-1 globulin fractions, 54.2 per cent with the alpha-2 and 36.6 per cent with the beta and O fractions.8

Blood urea nitrogen was 68 mg. per cent, creatinine 1.5 mg. per cent and uric acid 8.3 mg. per cent. The urine had a specific gravity of 1.010, was acid, and had a 2 plus proteinuria with no sugar. Its sediment showed 10-20 red blood cells, 3-4 white blood cells, 3-5 hyaline and granular casts and some white cell clumps per high power field. While in the hospital, the HCl ring test varied from negative to strongly positive; however,
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Bence-Jones proteinuria (Jacobsen test) was negative on three occasions. A protein precipitate formed after heating the urine for 10 minutes at 60°C, but this did not dissolve at 95°C, indicating the presence of a pseudo-Bence-Jones protein. Electrophoresis of a concentrated urine (dialyzed against dextran) showed that the major protein component migrated in the albumin region, with very minute amounts of all the globulin fractions also being present. Electrophoresis of the supernatant urine after the Jacobsen test showed that the albumin-migrating fraction had disappeared, with negligible change in the other fractions.

Twenty-four hour urines for creatinine on three different occasions were 49.5 mg./1200 ml., 127 mg./790 ml. and 40.4 mg./790 ml. of urine. Specific gravity remained fixed at 1.010 after a urine concentration test. Phenolsulfonphthalein excretion on two occasions was about 6 per cent in 15 minutes and 60 per cent after two hours. Urea and creatinine clearance were 40 and 30 per cent of normal respectively. Urine culture was negative.

Stool guaiac was negative. Tuberculin skin test (second strength PPD) was positive.

Studies of thyroid function showed a protein-bound iodine of 8.1 gamma per cent, basal metabolic rate +24 per cent, and a radioactive iodine uptake of 69 per cent in 24 hours.

Chest x-ray was within normal limits. Roentgenograms of the skull, long bones and vertebrae showed a moderate degree of diffuse bone decalcification. EKG showed the presence of a right bundle branch block.

Repeat values during the following month of most of the hematological and biochemical tests were essentially the same, except for the blood urea nitrogen, which gradually fell to about 40 mg. per cent.

Detailed descriptions of the bone marrow smears, lymph node biopsies and peripheral blood will be found in the succeeding section. The principal abnormal cell types found were lymphocytoid cells and lympho-plasmacytoid cells with cytoplasmic crystals, as well as reticulum cells.

Course in the hospital: The patient was initially given supportive intravenous therapy and later placed on an antipeptic ulcer regime during which time her abdominal complaints disappeared. A gastrointestinal series showed the presence of a small hiatus hernia and a moderately deformed duodenal bulb, presumably resulting from a duodenal ulcer. No definite ulcer crater was visualized.

While she was in the hospital, there was a slight increase in the size of her lymph nodes, and the liver and spleen became palpable 3 cm. and 8 cm. below their respective costal margins. Starting on 9/26, 20 mg. of nitrogen mustard were administered over a two day period. This was followed by a definite and progressive decrease in the size of the enlarged lymph nodes, liver and spleen. Spinal puncture and muscle biopsy had been refused.

The physiotherapy she received did not significantly improve her muscle atrophy and weakness. The latter condition was considered to be related to one of the unusual types of neuromuscular conditions which may be associated either with hyperthyroidism or with multiple myeloma, macroglobulinemia or lymphomas.

Her general state of health had not significantly changed, although the gastrointestinal complaints had disappeared. At the time of discharge on October 7, 1956, she weighed 91 pounds. The patient's basic condition and pathological diagnosis are evaluated in the discussion.

A diagnosis of hyperthyroidism associated with a nodular goiter was also made but specific antithyroid treatment was deferred for later evaluation and outpatient care.

She was readmitted one month later for two weeks because of recurrent gastrointestinal complaints. The findings were similar to those on her previous admission. Conservative treatment resulted in improvement and she was discharged.

One month afterwards she was seen in the outpatient clinic, feeling better and stronger, but she never returned. She died one and a half years later in a nursing home. No autopsy was performed.
Observations on Bone Marrow, Lymph Nodes and Blood

Sternal Marrow

Sternal bone marrow smears stained with Jenner-Giemsa (buffered at pH 6.7) showed the presence of sheets of immature lymphocytoid cells, most of them slightly larger than the normal lymphocyte and with a finer nuclear

Figs. 1–6.—Photomicrographs of bone marrow smears, Jenner-Giemsa, X 1000.

Fig. 1.—Lymphocytoid infiltrate of bone marrow. Arrow points to two young lymphoid cells. Reticulum cell is just below, toward the center.

Fig. 2.—Lymphocytoid infiltrate of bone marrow. Arrow points to large lymphoplasmocytoid cell.

Fig. 3.—Lymphocytoid infiltrate of bone marrow. Arrow points to two lymphoplasmocytes.

Fig. 4.—Large lymphoplasmocyte with oval inclusions in the cytoplasm.

Fig. 5.—Cell similar to that in fig. 4, but minute rod-shaped crystals are detectable in the cytoplasm.

Fig. 6.—Multiple minute rod-shaped intracytoplasmic inclusions in a lymphoplasmocyte; beginning pyknosis of nucleus.
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chromatin network (fig. 1). Many of the immature cells were oval, with an eccentric nucleus, and had varying amounts of basophilic cytoplasm ranging from a narrow rim to a larger triangular area (fig. 2). These cells were the most common cells in the bone marrow smears. Occasionally there appeared to be slight shedding of their cytoplasm. Larger, similarly-shaped cells, having a denser and more clumped nuclear chromatin pattern and a more basophilic cytoplasm, were also present (figs. 3–5). No “hof” was visible. The internal margin of the nucleus was frequently flattened or slightly indented, and almost all of its external margin was against the cell membrane. These cells contained, in varying numbers and sizes, minute, round light-blue-staining cytoplasmic inclusions. Others had minute-to-large crystals, which stained in the same way (figs. 4–6, 7). In those cells that did not have obvious inclusions, the cytoplasm had the foamy appearance so often seen in plasmocytes.

These cells appeared to undergo a progressive maturation to what will be termed here a “crystal cell.” As the cytoplasmic crystals accumulated, the nucleus usually became progressively more pyknotic and flattened, until a nuclear crescent was formed (figs. 8–12). The crystals usually appeared as randomly-oriented needles or rods and were of variable lengths, sometimes extending outside the cell (fig. 12). The basophilic cytoplasm was less prominent as the crystals accumulated, becoming reduced to fine lines between the crystals. When the crystals were long they were generally oriented in one direction. Mature crystal cells constituted about 5 per cent of the lymphocytoid cells. All plasmocytes seen appeared normal and frequently had minute circular pale-blue-staining inclusions in their cytoplasm (fig. 10). A few tissue basophils were also present.

In those areas that were not completely filled with sheets of lymphocytoid cells, there was an adequate number of megakaryocytes, and the white and red cell series were morphologically normal. The percentages of the latter were 1.5 per cent myeloblasts, 2 per cent promyelocytes, 2 per cent neutrophilic myelocytes, 1.5 per cent eosinophilic myelocytes, 11 per cent neutrophilic metamyelocytes, 16 per cent neutrophilic bands, 15 per cent segmented neutrophiles, 3 per cent eosinophiles, 30 per cent lymphocytes, 2 per cent plasmocytes, 12 per cent normoblasts, 2 per cent erythroblasts, 2 per cent reticulum cells.

"Crystal cells" were also recognized in formalin-fixed, paraffin-embedded sections of the aspirated sternal bone marrow.

Lymph Node Biopsies

Review of the lymph node biopsy performed in early 1951 at the other hospital showed that the architecture was completely distorted by interweaving bands and confluent islands of epithelioid or small reticulum cells. These had a clear oval nucleus with a nucleolus and an abundant, weakly-eosinophilic cytoplasm with indistinct borders (figs. 13, 14). Scattered among these cells were many eosinophiles as well as a small number of "crystal cells." Both of these cell types were also seen among normal-appearing lymphocytes, but here more "crystal cells" than eosinophiles were seen. In a few areas the capsule of the lymph node was infiltrated by both lymphocytes and "crystal cells."

A right axillary lymph node biopsy performed in 1956 showed that the
normal architecture was completely destroyed by a monotonous proliferation of lymphocyteoid cells whose nuclei were slightly larger and had a finer chromatin network than normal lymphocytes (figs. 15, 16). Distributed haphazardly throughout the node were cells with an eosinophilic cytoplasm composed almost entirely of refractile rod- and needle-like crystalline inclusions. The nucleus of these cells was generally pyknotic, frequently flattened or crescentic and eccentrically situated. Transitions were seen between cells with the nucleus just capping the cytoplasm and presumably younger forms with a round or oval nucleus and cytoplasm containing minute crystals. The mature
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Figs. 13–14.—Section of 1951 lymph node biopsy, Hematoxylin & Eosin.
Fig. 13.—Prominent confluent islands of epithelioid cells. X 170.
Fig. 14.—Higher magnification of above. Group of epithelioid-reticulum cells, surrounded mainly by lymphocytes. Two upper arrows point to eosinophiles; lower right arrow (c) to a "crystal cell". X 800.

Cell was by far the most prominent and was usually found in groups of a few to 20 or 30. These cells correspond to those described in the bone marrow.

At a number of points, the capsule was infiltrated by lymphocytoid cells and "crystal cells." The "crystal cell" was very prominent in the subcapsular lymph sinuses, in some of the medullary sinuses, and about the blood vessels.
Reticulum cells, larger than those of the earlier biopsy, with a large clear oval nucleus and a prominent nucleolus, were seen scattered throughout the lymph node. These cells were usually associated with "crystal cells" (fig. 16). Plasmacytes with Russell bodies were present but rare. No mast cells were seen.

Electron micrographs of the crystal cell were prepared by Dr. Boris Gueft.
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and Dr. John Ghidoni, Department of Pathology, Albert Einstein College of Medicine. A piece of the 1956 formalin-fixed, paraffin-embedded lymph node was deparaffinized, rehydrated, placed in osmic acid, dehydrated and embedded in methacrylate. Discrete rectangular crystals, as well as many which were needle-shaped and partly fused, were clearly seen. Higher magnifications showed that the crystals had an ultrastructure of alternating beaded lines similar to that described for the crystals seen in normal plasmocytes. Staining with lead acetate made the ultrastructure more visible. Further studies are in progress.

An axillary lymph node aspiration stained with Jenner-Giemsa showed a similar gamut from early transitional forms to well-developed “crystal cells”.

Blood Smears

The peripheral blood smear usually revealed about one crystal cell per slide, in addition to a few plasmocytoid lymphocytes and Rieder cells. Phase-contrast microscopic examination of suspensions of peripheral blood showed that small crystals were present within living lymphoplasmocytes. With Gomori’s technic, no alkaline phosphatase activity was seen in any of the peripheral white blood cells. This is in contrast to the marked activity seen in neutrophiles in multiple myeloma or chronic lymphatic leukemia.

DISCUSSION

The occurrence of protein crystals within the cytoplasm of cells or their deposition in tissues is rare. Such crystals usually occur in the normal or stimulated plasmocyte, its neoplastic counterpart, the myeloma cell, and some associated reticulo-endothelial cells. Extracellular crystals have been found within or beside plasmocytomas. Both intra- and extracellular crystals have been described in the kidneys of patients with multiple myeloma.

Clarification of the nature of such crystals has developed from an understanding of the morphology of the plasmocyte and an appreciation of its secretory function. This clarification has been achieved recently by phase-contrast and electron microscope studies. The plasmocyte, like other protein-producing cells, has a well-developed ergastoplasm. Its secretory product accumulates within cisternae or “vacuoles” bordered by ergastoplasmic membranes. These sacs may be flattened or dilated. Their contents may be invisible, or appear as a homogeneous material of low density or as large condensed structures. These may be spherical (Russell body) or, rarely, crystalline.

Because of the cytoplasmic basophilia, it is presumed that the lymphoplasmocyte described in this report also had a well-developed ergastoplasm like the plasmocyte and myeloma cell. Different stages of these cells contained spherical and oval inclusions, or crystals.

The well-developed “crystal cell” and the plasmocyte containing Russell
bodies may represent "storage cells", no longer able to release their formed product. For the crystallized or condensed material to be secreted, it would probably have to be redissolved. The observation of frequently distorted, fragmented and pyknotic nuclei in both types of cells favors the hypothesis that these cells are dying.

The accumulation of protein crystals in reticuloendothelial cells and in the kidney in some patients with multiple myeloma is considered to be secondary. Myeloma cells secrete proteins that are phagocytized by reticuloendothelial cells and also reabsorbed by the cells of the proximal convoluted tubules. Furthermore, such proteins may be deposited extracellularly within the lumens of the kidney tubules, or locally in or about the plasmocytomas where they are formed.

The nature of the secretory product of the plasmocyte, lymphocyte and "crystal cell" will be discussed in the next paragraphs.

Recognition that the plasmocyte may produce certain serum globulins has developed from the association of multiple myeloma with abnormal hyperglobulinemias1,23,30,51 and of bone marrow plasmocytosis with increased gamma globulins.52 Moreover, the globulin abnormalities in multiple myeloma may be reversed after generalized treatment50 or resection of local plasmocytomas in man53 or mice.24 In experimental immunization, an increase in both tissue plasmocytes and serum antibodies is found.19,50 More direct evidence has been obtained by extracting proteins from plasmocytomas53,54-57 or from bone marrow myeloma cells58 that were similar to those globulin fractions found elevated in the serum. Fluorescein-labeled antibody studies have shown the localization of gamma globulin in primitive and mature plasmocytes in both the spleen and lymph nodes of man and animals17,50-61 as well as in the myeloma cell.52,63 In agammaglobulinemia,63 there is a virtual absence of plasmocytes, a marked reduction or virtual absence of serum gamma globulins, and also a deficiency of certain serum alpha64 and beta globulins60,64 and macroglobulins.65

There is also evidence that the normal lymphocyte,66-68 the cells in some lymphomas and those in the macroglobulinemia of Waldenström, as well as in other allied diseases6,69-72 may produce gamma globulins. Globulin has been extracted from lymphosarcoma nodes.73 After resection of lymphosarcoma tissue,74 one patient's hyperglobulinemia was reversed. Fluorescent antibody techniques have shown the localization of gamma globulin in immature cell types and some lymphocytes and lymphocytoid cells.50,61 Moreover, anti-gamma globulin antibody reactions have been produced with lymphocyte extracts.75

It is also of interest to note that besides the absence of plasmocytes in agammaglobulinemia, there is also a marked reduction of lymphocytes.60,64 Furthermore, in multiple myeloma, where hyperglobulinemia is frequently present, lymphocytes may equal or outnumber the myeloma cells present in the bone marrow.76

Since the lymphoplasmocytoid cell in this case had morphological characteristics resembling both the plasmocyte and lymphocyte, it was considered that its secretory product would be similar or related to that of these two cells. Cytochemical studies reported in another paper tend to confirm this hypoth-
The secretory product of the plasmocyte (Russell body), the crystals in the lymphoplasmocyte and human gamma globulin were found to have an apparent isoelectric range between pH 6 and pH 7.5. These values are similar to those determined by physicochemical methods for human gamma globulin. These substances were also PAS-positive, thus showing the presence of carbohydrate.

In the present case, the patient's electrophoretic pattern revealed a narrow band and sharp peak in the gamma range, suggesting an excessive production of certain homogeneous gamma globulins. The abnormal globulins were probably the products of the lymphoplasmocytoid cell and the principal constituents of the crystals. However, in spite of the increased numbers of these cells giving signs of protein secretion, the total gamma globulin was not elevated. Perhaps one factor that might have accounted for this was that most of the abnormal cells were unable to release their secretory product.

Other protein abnormalities were present in this case. Most of the serum lipoproteins migrated with the alpha-2 globulin fraction rather than with the beta globulins. The serum mucoproteins were elevated, as in similar proliferative disorders.

The plasmocyte and the lymphocyte are believed to be closely related both in their origins and in their functional capacities. With present methods, the specific diagnosis of various lymphatic tumors and plasmocytomas is difficult because clinically, morphologically, and biochemically many transitional and overlapping cases exist.

In terms of classification, the present case falls somewhere in that broad spectrum of proliferative disorders centered about the reticulum cell, lymphocyte and plasmocyte. The histological pattern described here is not specific for any classical disease and the presence of a special cell type with cytoplasmic crystals makes the case difficult to classify. Unfortunately, no autopsy was obtained.

The earlier lymph node biopsy had a massive proliferation of epithelioid cells resembling either a sarcoid reaction, a markedly chronic lymphadenitis or a nonlipid reticuloendotheliosis. The second biopsy showed a pattern like that of a lymphoma, with a proliferation mainly of lymphocytoid cells and mononuclear reticulum cells. Both biopsies revealed crystal-containing cells. Finer cellular detail was seen in the hematological preparations. They clearly showed that along with a proliferation of lymphocytes, there were numerous lymphoplasmocytoid cells with many transitional forms to an end-stage "crystal cell". The proposed younger form of this cell type had a nucleus resembling that of a lymphocyte and cytoplasm like that of a plasmocyte—characteristics somewhat akin to the cells seen in some cases of Waldenström's macroglobulinemia. In the latter disease the cells have been variously described as lymphoid cells, atypical plasmocytes, transitional cell forms, or "lymphoid-plasmacelluläre Reticulomatose mit neoplastischem Character." Braunsteiner et al. have shown that these cells may have an extensive endoplasmic reticulum. Moreover, inclusions in such cells, like Russell bodies and crystals, are PAS-positive. The lymphoplasmocytoid cell in the present case most probably resembles the latter type of cell.
An important difference between the lymphoplasmocytoid cell described here and a plasmocyte is their nuclear shape. The nucleus of a plasmocyte full of Russell bodies or crystals remains essentially globular or indented, whereas the "crystal cell" nucleus is flattened or crescentic. Before the inclusion product accumulates within these cells, the nuclear cytoplasmic ratio is less in the plasmocyte than in the lymphoplasmocytoid cell.

Although proliferating cells with large numbers of cytoplasmic crystals are practically limited to cases of multiple myeloma, this patient probably did not have this disease. In a survey of cases in which intracytoplasmic crystals were found, the cells were closely related to but not identical with those described in the present report.

SUMMARY

A case of an unusual lymphomatous disease associated with lymphoplasmocytoid cells containing intracytoplasmic crystals is presented. These crystals are considered to be the secretory product of these cells. They are discussed in the light of recent studies on the secretory activity of plasmocytes and related cells.

A specific diagnosis of the patient's disease is not made although the main cell type present closely resembles that occurring in Waldenström's macroglobulinemia.

SUMMARIO IN INTERLINGUA

Es presentate un caso de un inusual morbo lymphomatic associate con cellululas lymphoplasmocytoid contenente cristallos intracytoplasmatic. Es opinate que iste crystallos est un producto secretori del cellulas in question. Illos es discutite in le lumine de recente studios del activitate secretori de plasmocytos e cellulas affin.

Un diagnose specific del morbo del presente patiente non es formulate ben que le major typo de cellululas notate es intimemente simile al cellululas occurrente in macroglobulinemia de Waldenström.

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Case report of a primary follicular lymphoma of the thyroid. The case is indicated as the first in the literature. Histologic examination showed the initial transformation into a diffuse form of reticulosarcoma with undifferentiated cells, on the background of a lymphomatous Hashimoto struma.—P. d. N.
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