INFECTIOUS LYMPHADENOSIS (MONONUCLEOSIS) AND HEMOLYTIC ANEMIA IN A NEGRO; RECOVERY FOLLOWING SPLENECTOMY

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In most instances, infectious lymphadenosis (mononucleosis) is not a diagnostic problem. In a recent publication, Dameshek and Grassi state that the lack of reduction of red cells and platelets in infectious lymphadenosis (mononucleosis) is of considerable aid in distinguishing this disease from acute lymphatic leukemia, and that the association of well-defined anemia and/or thrombocytopenia with a marked degree of lymphocytosis in which abnormal lymphocytosis is conspicuous almost certainly indicates acute leukemia.

These authors reported a case of severe thrombocytopenic purpura in a patient who had infectious mononucleosis. Splenectomy resulted in an excellent platelet response. The case herein presented is another exception to these generally accepted concepts in the diagnosis of infectious mononucleosis. A young Negro male was found to have generalized lymphadenopathy, slight hepatomegaly, marked splenomegaly, an absolute lymphocytosis and a severe anemia. Although the clinical picture strongly suggested leukemia, the qualitative characteristics of the lymphocytes were those of infectious mononucleosis and this diagnosis was confirmed by a markedly positive heterophile agglutination test. In addition to the marked anemia there was also a reticulocytosis, moderately increased red cell fragility, an elevation in the icterus index, and an increased urinary excretion of urobilinogen. This was interpreted as a hemolytic anemia. The family history was entirely negative. Splenectomy was performed when the anemia became extremely severe and uncontrollable by transfusions, and a prompt recovery occurred.

Infectious lymphadenosis is rare in the Negro, but has been reported. Anemia has been reported with this disease in but a few cases.

This case is of interest not only because of its rarity, but also because of the possibility of 'hypersplenism' with resultant hemolytic anemia, thrombocytopenia and moderate leukopenia. The hemolytic anemia was the predominating feature of the disease.

REPORT OF CASE

H., a young adult colored male, aged 18 years, was admitted to the hospital May 28, 1944. He complained of headache, fever and chills. A diagnosis had been made on May 23, 1944, of influenza. Because of a marked anemia there was also a reticulocytosis, moderately increased red cell fragility, an elevation in the icterus index, and an increased urinary excretion of urobilinogen. This was interpreted as a hemolytic anemia. The family history was entirely negative for hemolytic anemia.

On examination, the patient appeared to be acutely ill. The sclerae were jaundiced. The lymph nodes of the neck, axillae, and inguinal regions were enlarged. A systolic murmur was present in the apical region. The liver was palpable at the costal margin. The spleen was enlarged and extended 8 cm. below the costal margin and medially to the midline.

On May 31, 1944, he was seen by one of us (S.J.W.). The physical findings were the same as on admission. Blood studies at this time revealed the following: erythrocyte count 2,400, hemoglobin 8.0

The patient was studied at La Garde General Hospital, New Orleans, Louisiana.
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Gm. (31 per cent), leukocyte count 19,300, reticulocytes 9.5 per cent, icterus index 17 units, heterophile agglutination test positive in a dilution of 1:896, urinary urobilinogen positive in a dilution of 1:50. A red cell fragility test showed initial hemolysis at 0.45 and complete at 0.32 (normal initial hemolysis, 0.41; complete, 0.30). No sickling of red cells was observed in a 14 hour wet preparation. The platelet level by the Rees-Ecker method was 240,000 per cu. mm. The peripheral blood was studied by the supravital technic and after having been stained with Wright's stain. The following differential count was obtained: polymorphonuclear leukocytes 20 per cent, lymphocytes 37 per cent, monocytes 1 per cent, eosinophiles 2 per cent. The lymphocytes varied in size and staining reaction and were typical of infectious mononucleosis. The erythrocytes showed some polychromatophilia, anisocytosis, and slight stippling. Spherocytes were numerous.

A sternal marrow biopsy on June 1, 1944, revealed marked hyperplasia. Extensive plaques of erythroid elements at the normoblastic level of cell maturation were observed. The other marrow elements were normal in appearance and maturation levels but were relatively decreased. Occasional young lymphocytes were observed.

The patient began to improve clinically and hematologically (see fig. 1). A second hemolytic crisis developed, however, with a drop of erythrocytes, leukocytes and platelets. Transfusions of 500 cc. each were given on June 10, 14 and 15. The erythrocyte count on June 15 was 1.23 M. with a hemoglobin of 4.23 Gm. (27 per cent). Because of the uncontrollable hemolytic phenomenon, a splenectomy was done (L.W.G.) on June 15, 1944. The spleen was greatly enlarged and removed with some difficulty. Two accessory spleens were also removed. The red cell count in the afternoon of the operative day was 1,330,000, and hemoglobin 8.3 Gm. (55 per cent).

Fig. 1. Graphic illustration of the hematologic response in a Negro with infectious mononucleosis and hemolytic anemia. Splenectomy resulted in recovery.
The spleen grossly was markedly enlarged and moderately firm. The weight was 860 Gm. The capsule was smooth and not thickened. The splenic pulp was red and scraped with ease. Microscopic examination revealed marked dilatation of the sinusoids, which were filled with erythrocytes. The malpighian bodies were not enlarged and the germinal centers were not proliferative in type. This probably is explained on the basis that the infectious mononucleosis had subsided before the splenectomy, as the lymph nodes had also receded.

The postoperative course was uneventful. The patient was observed until October 16, 1944. No recurrence of the hemolytic phenomenon was observed. On this date, the following laboratory data were obtained: erythrocytes 4,95, leukocyte count 12,800 (higher than the usual periodic observations), hemoglobin 15.3 Gm. (98 per cent), platelets 425,000, differential, polymorphonuclear leukocytes 48 per cent, lymphocytes 47 per cent, monocytes 1 per cent, eosinophiles 4 per cent, urinary urobilinogen slightly positive in a 1:5 dilution, heterophile agglutination positive 1:10 dilution.

DISCUSSION

As has been stated before, infectious lymphadenosis is rare in the Negro, but has been reported. It was quite evident in our case that there was a considerable mixture of negroid and white stock. Although negroid characteristics were present, the hair was auburn and the skin a very light brown.

Infectious mononucleosis is a readily recognizable disease, running a characteristic course. One of its outstanding features is the lack of anemia. Read and Helwig reported anemia in only 6 of 300 cases of infectious mononucleosis. In 3 of these patients, there was a rapid and simultaneous drop in red blood cells, white cells and platelets. This was followed by a gradual rise in all three of the formed elements of the blood.

The 3 cases of Read and Helwig and the case reported here again serve to emphasize that a diagnosis between lymphatic leukemia and infectious mononucleosis cannot always be made on the absence of anemia and the presence of immature lymphocytes. The cells must indeed be studied for qualitative differences and correlated with the heterophile agglutination test. Read and Helwig, in addition to mentioning a possible hemolytic phenomenon, believed that the anemia may be partially explained by infiltration of the bone marrow. It is interesting to note that in one of their cases (case 3) the icterus index was 31 at the time the red blood cells were 1,900,000 and the indirect Van den Bergh test 3.5 mg. No other data are given which would be of aid to determine whether or not a hemolytic process existed.

In the case presented in this report, the cause of the hemolytic process cannot be definitely stated. No familial history could be obtained. The spleen was considerably enlarged and microscopically typical of a marked hemolytic process, the sinusoids being distended with erythrocytes. It could well be that this represented another manifestation of 'hypersplenism.' Doan and Wright, Doan and Dameshek and Estren have discussed at length the various types of hypersplenism of the primary and secondary types. Specific primary diseases exist in which an unstable splenic reticulo-endothelial system either acutely or chronically destroys and/or inhibits the blood cells, platelets and granulocytes excessively. These are descriptively identified as congenital hemolytic anemia, essential thrombocytopenic purpura, primary splenic neutropenia and primary splenic panhematopenia. One or other of these primary syndromes may be simulated by any secondary involvement of splenic tissue. Doan and Wright, Doan and Dameshek and Estren.
mentioned many such diseases in their discussions of the subject. Hemolytic anemia however, is not mentioned as occurring in infectious mononucleosis. One can hypothesize that in this case the spleen was stimulated to overdestruction of red cells and a severe hemolytic anemia resulted, which necessitated its removal. As Dameshek and Grassi state: "The whole subject of spleen-bone marrow relationships under normal and pathologic conditions has only recently come to the forefront, and many questions related to the possibly increased activity of the spleen must await further investigation."

CONCLUSION

A case is presented of acquired hemolytic anemia and infectious lymphadenosis (mononucleosis) in a Negro. Splenectomy for the uncontrollable hemolytic state was followed by prompt recovery.

REFERENCES

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