Splenectomy in Far-Advanced Hodgkin’s Disease

Report of Five Cases

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Failure of hematopoiesis is a frequent occurrence in patients with far-advanced Hodgkin’s disease, and the treatment of this situation has been largely unsuccessful. The depression in the formed elements of the blood has been attributed to several different mechanisms, which include:

1. Infiltration of the bone marrow by Hodgkin’s disease.17 29 32
2. Bone marrow aplasia due to repeated insults by bone marrow depressants used in treatment, such as nitrogen mustard (HN2), triethylene melamine (TEM) and x-rays.20
3. Toxic depression of bone marrow function associated with other manifestations of systemic intoxication in Hodgkin’s disease, such as fever, itching, weakness, and anorexia.29 32
4. Hypersplenism.7 16 27 38

Although each, or a combination of these mechanisms may be responsible for the depressed or abnormal hematopoiesis in Hodgkin's disease, their relative importance in a given case is often difficult to assess. In this report the splenic factor will be considered.

Results of Splenectomy

Five patients with far-advanced Hodgkin’s disease and hematopoietic failure were splenectomized in order to determine the effect of the procedure on: (1) the course of the disease, (2) the peripheral blood findings, and (3) the renewal of the patient’s suitability for, and responsiveness to, further nitrogen mustard therapy, as suggested by Rosenthal.25 The course of each patient is presented in cases 1 through 5 and figures 1 to 4.

Two additional patients with Hodgkin’s disease had their spleens removed when the disease was not so far-advanced. Their cases are reported briefly.

R. F., 29 year old male, was diagnosed as having Hodgkin’s disease by a cervical node biopsy. He responded satisfactorily to x-ray therapy for the next six years, during which period the disease became more generalized. When the course of the disease accelerated, and he became less responsive to x-ray therapy, an enlarged spleen was removed. Following
this procedure the patient's course continued downhill, although he was treated vigorously with x-rays, nitrogen mustard and cortisone. He expired fourteen months after splenectomy.

The second patient was R. C., a 27 year old male, who was found to have an enlarged spleen on routine physical examination. The spleen continued to enlarge and the liver became palpable. After six months of observation, splenectomy was performed. At this time a biopsy of the liver, as well as of the spleen, showed Hodgkin's disease. The liver continued to increase in size and the patient received intravenous nitrogen mustard and x-ray therapy to the liver with temporary improvement. The disease progressed and, despite further treatment with x-ray and TEM, the patient died nine months after splenectomy. At autopsy the disease was widespread, and the histologic diagnosis was Hodgkin's sarcoma.

**DISCUSSION**

Splenectomy did not produce any substantial clinical benefit in our series of five patients with far-advanced Hodgkin's disease, and they died within thirteen weeks following the operation. There was transient improvement in the leukocyte and platelet counts, and in three cases the hemoglobin level appeared to be maintained without transfusions for a slightly longer period. However, the course of the disease was not interrupted or alleviated, and in three instances the liver seemed to become larger, presumably due to infiltration with Hodgkin's disease. Since the leukocyte and platelet counts rose postoperatively, the factor in the spleen suppressing these normal cells preoperatively may have also restrained the growth of the neoplastic cells in Hodgkin's disease. While splenectomy was thought to be of some value in renewing the tolerance to nitrogen mustard in the four cases briefly reported by Rosenthal, it is not known if these patients would have responded to nitrogen mustard if it had been given prior to the removal of the spleen. In our experience, splenectomy did not make the patients better candidates for nitrogen mustard therapy, and in two cases (nos. 1 and 2) HN2 given after splenectomy was without benefit. It does not seem likely, therefore, that the spleen plays an important role in the anemia, leukopenia, and thrombocytopenia which may occur in Hodgkin's disease.

In table 1, thirty case reports of splenectomy in Hodgkin's disease are summarized from the literature. There are other references to splenectomy in Hodgkin's disease, but they are too brief to merit analysis. A number of reasons for splenectomy were given, but the operation was of limited value in all but a few cases.

**Primary Splenic Tumors**

If the spleen were the sole focus of Hodgkin's disease in such a patient, splenectomy could possibly be a curative measure. Splenectomy has been performed in some cases because of an enlarged spleen, or a pre-operative clinical diagnosis of hemolytic anemia (table 1, cases no. 1-4, 6, 9, 12, 14, 15, 17, 18, 19) or hyporesplenism, and at operation or subsequently the diagnosis of Hodgkin's disease was established. Despite what may be called an early operation, done before classical signs of the disease were present and in patients who apparently had only localized disease at operation (table 1, cases no. 2, 4, 6, 10, 17, 23, 24), with two exceptions all subsequently developed generalized signs of Hodgkin's disease. The two exceptions (table 1, cases no. 23, 24), however, had only a three month follow-up recorded. There is no recorded case of Hodgkin's disease, appar-
Table 1.—Case Reports of Splenectomy in Hodgkin’s Disease Summarized from the Literature

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Ref.</th>
<th>Age, sex</th>
<th>Indications for splenectomy</th>
<th>Blood picture</th>
<th>Results of splenectomy</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>34</td>
<td>18F</td>
<td>Splenomegaly, fever</td>
<td>RBC 4.7, WBC 7.0.</td>
<td>Spleen enlarged, showed Hodgkin’s disease. Patient died 24 hours after splenectomy.</td>
</tr>
<tr>
<td>2</td>
<td>35</td>
<td>55M</td>
<td>Splenomegaly, fever, weight loss</td>
<td>RBC 3.3, Hgb 11.1, WBC 12.6</td>
<td>Large spleen showed Hodgkin’s disease. Patient gradually declined with hepatomegaly, fever, ascites; died 2 years later.</td>
</tr>
<tr>
<td>3</td>
<td>8</td>
<td>8M</td>
<td>Hepatosplenomegaly, ascites, fever, hemolytic anemia</td>
<td>RBC 1.4, Hgb 6.3, WBC 12.2</td>
<td>Large spleen and adjacent nodes removed, showed Hodgkin’s disease. Hematologic improvement, and patient better 2 months later.</td>
</tr>
<tr>
<td>4</td>
<td>21</td>
<td>55F</td>
<td>Splenomegaly</td>
<td>RBC 2.5, Hgb 11.9, WBC 7.0</td>
<td>Splenectomy showed Hodgkin’s disease. Patient subsequently developed discrete lymph nodes.</td>
</tr>
<tr>
<td>6</td>
<td>12</td>
<td>43M</td>
<td>Hepatosplenomegaly, hemolytic anemia</td>
<td>RBC 1.3, Hgb 4.4, WBC 2.8, platelets 330,000</td>
<td>Spleen enormous, weighed 3000 Gm., showed Hodgkin’s disease. Blood picture improved, and patient was in fair general condition for 13 weeks later. Seven weeks after splenectomy substantial nodes enlarged, biopsy showed Hodgkin’s disease.</td>
</tr>
<tr>
<td>7</td>
<td>13</td>
<td>32M</td>
<td>Hepatosplenomegaly, jaundice, generalized lymphadenopathy, hemolytic anemia</td>
<td>RBC 1.4, Hgb 4.6, WBC 7.4, platelets 104,000</td>
<td>Spleen weighed 900 Gm., showed Hodgkin’s disease. Hgb. rose postoperatively to 8.3 Gm. Patient died one year after splenectomy.</td>
</tr>
<tr>
<td>8</td>
<td>13</td>
<td>54M</td>
<td>Hepatosplenomegaly, jaundice, anemia</td>
<td>RBC 2.0, Hgb 5.5, WBC 3.1, platelets 55,000</td>
<td>Spleen showed Hodgkin’s disease. Platelets rose to 102,000 but patient died 2 days postoperatively.</td>
</tr>
<tr>
<td>9</td>
<td>5</td>
<td>29M</td>
<td>Hepatosplenomegaly, hypercalcemia, osteosarcoma, bone</td>
<td>RBC 2.7, Hgb 8.4, WBC 3.8, platelets 167,000, serum calcium 14-17 mg.%</td>
<td>Spleen weighed 750 Gm., showed Hodgkin’s disease. RBC rose to 4.0. Two months after splenectomy generalized lymphadenopathy appeared.</td>
</tr>
<tr>
<td>10</td>
<td>18</td>
<td>47F</td>
<td>Splenomegaly, anemia</td>
<td>RBC 1.3</td>
<td>Spleen weighed 1090 Gm., hematologic improvement for 8 months, then patient declined, died 20 months after splenectomy of generalized Hodgkin’s disease.</td>
</tr>
<tr>
<td>12</td>
<td>28</td>
<td>37F</td>
<td>Splenomegaly, jaundice, anemia</td>
<td>RBC 1.9, Hgb 8.4, WBC 3.1</td>
<td>Spleen showed Hodgkin’s disease. Patient still improved 2 years after splenectomy. RBC 4.0, WBC 15.6.</td>
</tr>
<tr>
<td>13</td>
<td>30</td>
<td>10M</td>
<td>Hepatosplenomegaly, jaundice, fever, anemia</td>
<td>RBC 1.9, WBC 7.7</td>
<td>Hematologic improvement. Patient benefited for 5 months, then disease became activated and patient died.</td>
</tr>
<tr>
<td>15</td>
<td>24</td>
<td>34M</td>
<td>Splenomegaly</td>
<td>RBC 3.6, Hgb 12.6, WBC 3.2, platelets 30,000</td>
<td>Spleen weighed 740 Gm., showed Hodgkin’s disease. Blood picture transiently improved, RBC 3.4, Hgb. 10.0, WBC 7.5, platelets 272,000. Patient died 5 weeks after splenectomy.</td>
</tr>
<tr>
<td>16</td>
<td>7</td>
<td>52M</td>
<td>Hepatosplenomegaly, weakness, fever</td>
<td>RBC 2.8, Hgb 7.3, WBC 1.5, retics. 4.1%, platelets 167,000. Bone marrow hyperplastic</td>
<td>Spleen showed Hodgkin’s disease. Patient died 1 year later of generalized Hodgkin’s disease.</td>
</tr>
<tr>
<td>17</td>
<td>31</td>
<td>43F</td>
<td>Spleenomegaly, hemolytic anemia</td>
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<tr>
<td>RBC 2.3, Hgb. 8.2, WBC 3.5, retics. 19%, platelets 140,000</td>
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<tr>
<td>18</td>
<td>31</td>
<td>33F</td>
<td>Hemolytic anemia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RBC 1.5, Hgb. 4.4, WBC 15.3, retics. 9%, platelets 300,000</td>
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<td></td>
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<tr>
<td>19</td>
<td>23</td>
<td>64F</td>
<td>Spleenomegaly, weight loss</td>
<td></td>
<td></td>
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<tr>
<td>Within normal limits</td>
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<td></td>
<td></td>
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<tr>
<td>20</td>
<td>4</td>
<td>56M</td>
<td>Acquired hemolytic anemia, spleen not palpable</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RBC 1.3, Hgb. 3.2, WBC 22.0, Bilirubin 7.3 mg%, Coombs' test +</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>21</td>
<td>16</td>
<td>50F</td>
<td>Spleenomegaly, fever</td>
<td></td>
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<tr>
<td>RBC 3.2, Hgb. 10.0, WBC 4.0, bone marrow showed moderate hyperplasia of erythroid elements</td>
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<tr>
<td>22</td>
<td>27</td>
<td>14M</td>
<td>Spleenomegaly, hemolytic anemia</td>
<td></td>
<td></td>
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<tr>
<td>Anemia</td>
<td></td>
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<tr>
<td>23</td>
<td>38</td>
<td>—</td>
<td>Anemia, jaundice, reticuloctysis, bone marrow showed erythroid hyperplasia</td>
<td></td>
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<tr>
<td>24</td>
<td>38</td>
<td>—</td>
<td>Thrombocytopenia developed 16 mos. after onset of Hodgkin's disease</td>
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<tr>
<td>25</td>
<td>38</td>
<td>—</td>
<td>Leukopenia with bone marrow showing myeloid hyperplasia 17 mos. after onset of Hodgkin's disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>26</td>
<td>38</td>
<td>—</td>
<td>Panmyelopenia during advanced stage of Hodgkin's disease</td>
<td></td>
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</tr>
<tr>
<td>27</td>
<td>9</td>
<td>67M</td>
<td>Hepatosplenomegaly, acquired hemolytic anemia</td>
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<tr>
<td>RBC 1.9, Hgb. 7.1, WBC 5.3, retics. 1.8%, platelets 625,000</td>
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<tr>
<td>28</td>
<td>10</td>
<td>26M</td>
<td>Hepatosplenomegaly, bleeding</td>
<td></td>
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</tr>
<tr>
<td>Hgb. 4.0, WBC 8.2, platelets 1,600, bone marrow showed increased cellularity, megakaryocytes</td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>29</td>
<td>26</td>
<td>13M</td>
<td>Spleenomegaly, jaundice, acquired hemolytic anemia, Coombs test +</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hgb. 2.6, WBC 33.0, retics. 0.1%</td>
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<td></td>
</tr>
<tr>
<td>30</td>
<td>3</td>
<td>64F</td>
<td>Hepatosplenomegaly, jaundice, anemia, lymph node biopsy showed Hodgkin's disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RBC 1.1, Hgb. 4.8, WBC 40.5, retics. 84.8%, platelets 342,000, Bone marrow showed massive erythroid hyperplasia, Coombs' and trypan tests negative</td>
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</tbody>
</table>

* RBC, red blood cells X 10^6; Hgb., hemoglobin, Gm. per cent; WBC, white blood cells X 10^9.
ently confined to the spleen at operation, which has been cured or indefinitely arrested by splenectomy. While an undiagnosed splenic tumor is often a surgical problem, if the diagnosis of Hodgkin's disease is established, the value of splenectomy is very questionable. Shullit,$^6$ however, removed a large spleen from a young boy with known generalized Hodgkin's disease, and the patient was still improved two years after splenectomy (table 1, case no. 13).

**Acquired Hemolytic Anemia**

While the rate of erythrocyte destruction is increased in Hodgkin's disease, it is probably far higher in acquired hemolytic anemia. It may be pertinent to note here that cortisone, which has been effective in the treatment of acquired hemolytic anemia,$^7$ was found to be of little value in ten patients with hematologic depression associated with Hodgkin's disease,$^{23}$ although there was a slight reduction in the number of transfusions required. While anemia, jaundice, and splenomegaly appear to be fairly common in Hodgkin's disease, the diagnosis of acquired hemolytic anemia was not clearly established in many of the early cases. It was not possible to ascertain in most cases the severity of the hemolytic process, the completeness of the hematologic response to splenectomy, or the role of the treatment of the underlying Hodgkin's disease in diminishing hemolysis. In sixteen cases thought to be due to hemolytic anemia (table 1, cases no. 3, 6, 7, 10–14, 17, 18, 20, 22, 23, 27, 29, 30), ten were improved by splenectomy for periods ranging from eight weeks to four and one-half years. In four cases, splenectomy did not control the hemolytic process, but it was subsequently improved by specific therapy to the Hodgkin's disease with x-rays (table 1, cases no. 18, 20) and TEM (table 1, cases no. 29, 30). Williams, et al.$^{38}$ briefly note that splenectomy did not relieve hemolytic anemia in two of their cases. It is thus likely that there is an extrasplicic factor causing hemolysis in Hodgkin's disease patients, and this factor may be diminished by decreasing the activity of the disease with x-rays, HN2 or TEM.

**Thrombocytopenic Purpura**

This complication, occurring independently of a general depression of hematopoietic activity, is a rare occurrence in Hodgkin's disease. Wright-Smith$^{39}$ described a tuberculous patient with a severe bleeding tendency and thrombocytopenia. At autopsy the spleen, liver, and bone marrow were infiltrated with Hodgkin's disease. Two patients (table 1, cases no. 24, 28) were thrombocytopenic, and this was relieved by splenectomy; both patients, however, died of Hodgkin's disease at eight and four months, respectively, after splenectomy.

**Hypersplenism**

The diagnosis of hypersplenism is difficult to establish, and there was little evidence of a significant degree of hypersplenism in the patients in our series. In three cases with depression of the formed elements of the blood (table 1, cases no. 5, 16, 21), splenectomy temporarily improved the blood picture. Williams, et al.$^{38}$ have reported the highest incidence of hypersplenism in Hodgkin's disease. Following splenectomy they state that "the hematological pattern returned to normal or was greatly improved in all but 2 of 11 cases." There are only scanty
details in this report, and four of their cases (nos. 23–26), which are described most fully, albeit inadequately, are cited in table 1.

Summary and Conclusions

Five patients with far-advanced Hodgkin's disease and with evidence of hematopoietic failure were treated by splenectomy in order to determine whether: (1) the course of the disease could be modified, (2) the hematologic picture improved, and (3) responsiveness to nitrogen mustard or x-ray therapy restored. These patients showed a transient slight improvement in their hematologic status, but the course of the disease possibly was accelerated, and the patients all died within thirteen weeks, without showing renewed suitability or increased responsiveness to therapy.

On the basis of our data and a review of thirty cases from the literature, it is concluded that splenectomy is not a useful procedure in Hodgkin's disease, except for certain specific indications. These may be: (1) an apparently solitary splenic tumor; (2) acquired hemolytic anemia, although this process may be better controlled in some cases by treating the underlying Hodgkin's disease with x-rays, nitrogen mustard, or triethylene melamine; (3) thrombocytopenic purpura, which appears to be more profound than is to be expected from the severity and extent of Hodgkin's disease; and (4) hypersplenism. Hematopoietic depression in the vast majority of patients with Hodgkin's disease, however, cannot be attributed to splenic overactivity or malfunction.

Case Reports

Case No. 1

M. P., a 23 year old female, developed fever and peripheral lymphadenopathy in March 1944. A biopsy of a cervical node was diagnosed as Hodgkin's disease. For the next three years her disease was satisfactorily controlled by repeated courses of HN₂¹¹ and x-ray therapy. Late in 1947, the disease became increasingly resistant to HN₂; remissions were brief and symptoms recurred promptly despite the production of severe leukopenia with HN₂. In November 1947, one month after an intensive course of HN₂, the patient relapsed with fever, generalized lymphadenopathy and splenomegaly. At this time the hemoglobin was 5.0 Gm., leukocytes 4500, and platelets 95,000. Examination of the bone marrow on January 6, 1948 showed fragments of normal marrow mixed with blood. Because of the enlarging spleen, depression in the formed elements of the blood, and the progressive nature of the disease with resistance to therapy, splenectomy was performed. At operation, the spleen was approximately 3 times normal size and histologically appeared to be almost entirely replaced by Hodgkin's disease tissue. Enlarged mesenteric and retroperitoneal lymph nodes were felt. Following splenectomy the platelet and leukocyte counts rose and the hemoglobin level was maintained without transfusions (fig. 1). Two weeks later, peripheral lymph nodes began to enlarge, and she received 0.3 mg./Kg. (15 mg.) of HN₂ during February 9 to 12, 1948 without improvement.

During March the leukocyte count rose to a level of around 80,000, petechiae appeared associated with thrombocytopenia, and the peripheral blood showed a remarkable number of nucleated red cells ranging from 10 to 50 per cent of the total leukocyte count. A sternal marrow aspiration on April 1, 1948 revealed a depressed myeloid and marked erythroid activity, with occasional cellular thrombocytes. The per cent of cell types was: erythroid series 80 per cent, composed of megaloblasts 3 per cent, erythroblasts 3 per cent, O-rubrocytes 26 per cent, B-rubrocytes 26 per cent, P-rubrocytes 22 per cent; lymphocytes 8 per cent, chiefly blast forms; and myeloid cells 12 per cent. This was interpreted as a bizarre
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leukemoid reaction. The patient became progressively weaker, and the liver enlarged rapidly. An aspiration biopsy of the liver showed infiltration with Hodgkin’s disease. Terminally, a brief trial of 4-amino-pteroylglutamic acid (Aminopterin) was given without effect, and the patient died April 7, 1948. This patient survived four years after the clinical onset of the disease and almost three months after splenectomy. Permission for autopsy was not obtained.

Comment: This patient had generalized and active Hodgkin’s disease during the entire four year course which was fairly well controlled for the first three years by nitrogen mustard. When severe anemia and thrombocytopenia developed, associated with an increasing splenomegaly and resistance to HN2, the spleen was removed. Following the operation the patient showed a rise in platelet count and appeared to hold her hemoglobin level more satisfactorily than before splenectomy. There was no substantial clinical improvement and the neoplastic process appeared to be accelerated with progressive enlargement of the lymph nodes and liver. Terminally, she developed a bizarre leukemoid reaction with a large proportion of nucleated red cells in the bone marrow and peripheral blood. Fever and anemia persisted, and the platelet count fell to low levels before death.

Case No. 2

M. S., a 14 year old male, developed enlarged right cervical nodes in 1940 and a biopsy was diagnosed as Hodgkin’s disease. X-ray therapy caused regression of the nodes for three years. The disease then appeared in a generalized form, and was controlled with radiotherapy for the next four years. Because of marked activation of the disease, in November 1947 he received his first course of HN2 and responded well for two months. Further HN2
FIG. 2.—Case 2: M. S., 14 year old male with Hodgkin's disease.

was then given, with brief and partial control, but finally he became resistant to therapy. In July 1948, x-ray therapy given to the liver and spleen was without clinical benefit. Another course of HN2 produced a slight reduction in splenic size, a decrease in fever and a leukopenia of 1100; at the same time, however, pulmonary infiltrations and a pleural effusion appeared. In view of the rapid progress of the disease, his resistance to x-ray and HN2 therapy, and the contraindication to these forms of treatment because of the severe depression of the formed elements of the peripheral blood and bone marrow, splenectomy was performed on November 3, 1948 (fig. 2). The spleen was enlarged and microscopically showed Hodgkin's disease with extramedullary hematopoiesis, areas of hemorrhagic necrosis, and accumulations of blood pigment. Bone marrow aspiration three weeks after splenectomy was hypocellular.

Two weeks later the liver began to enlarge rapidly, and the patient became icteric and febrile. HN2 and then a nitrogen mustard analogue were given; they only induced further depression of his peripheral blood picture. He died February 4, 1949, three months after splenectomy and about nine years after the clinical onset of his disease.

Autopsy findings showed Hodgkin's sarcoma involving lymph nodes, liver, lungs, diaphragm, serosa of stomach and skin. The liver also showed chronic passive hyperemia, edema, and periportal fibrosis. The bone marrow was markedly hypoplastic.

Comment: The patient responded well to x-ray therapy during the first eight years of his disease. The process then became more active, and although he obtained an excellent but brief response to HN2, the disease progressed, and finally became resistant to the drug. Because of his poor hematologic status, which required repeated blood transfusions, and the lack of response to HN2 and x-rays, splenectomy was performed. The platelets and leu-
Case No. 3

M. L., a 38 year old male, had an enlarged cervical node biopsied in June, 1947; this was diagnosed as Hodgkin's disease. He received x-ray therapy, and remained in good condition for more than a year. Early in 1949, systemic symptoms appeared, and these were controlled for about twenty months by repeated courses of HN2, x-rays, and then triethylene melamine (TEM). In November 1950 the disease showed increased activity with enlargement of the liver and spleen with persistent fever and bleeding tendencies. The blood findings were hemoglobin 7.4 Gm., leukocytes 1300 with a normal differential count, and platelets 8000. Sternal marrow aspiration was cellular (44 per cent myeloid, 20 per cent lymphoid and 36 per cent erythroid composed of O-rubrocytes 25 per cent, B-rubrocytes 5 per cent, and P-rubrocytes 6 per cent). The megakaryocytes were decreased. Further therapy with HN2, TEM or x-rays was not feasible, and on December 14, 1950 the spleen was removed (fig. 3). It was about 3 times normal size and microscopically showed Hodgkin's disease and hemosiderosis. The leukocyte count rose to 10,000 and remained within normal limits for one month. The platelet count rose and then within two weeks fell to preoperative levels. The disease continued to progress, generalized bleeding developed, and death occurred on January 14, 1951, thirty-three days after splenectomy. At postmortem, Hodgkin's disease, showing granulomatous and sarcomatous features, was seen, involving lymph nodes, bone marrow, and dura of the pituitary gland. An acute hemorrhagic colitis was also present.

Comment: The disease in this patient was fairly well controlled for two and one-half years, and then it accelerated rapidly during the last year, running its course in three and one-half years. While he responded to HN2 and TEM for one and one-half years, during the last six...
months of his life he became refractory to treatment and developed depression of the formed elements of the blood. Splenectomy caused a rise in the white cell count and slight and transient increase in platelet count, but it had no effect on the bleeding tendencies, anemia, or the course of the disease.

Case No. 4

M. C., a 35 year old male, complained of weakness, malaise, muscular aches, and weight loss beginning in the summer of 1947. In January 1948 the patient was found to have generalized adenopathy, splenomegaly, fever, and anemia. A biopsy of an enlarged cervical node was diagnosed as Hodgkin’s disease. X-ray therapy and then a course of nitrogen mustard (fig. 4) did not control the disease, and the patient became progressively worse with a persistent high fever. The depression in the formed elements of the blood made it hazardous to continue HN2 or x-ray therapy, and splenectomy was performed on June 18, 1948. The spleen was estimated to be 3 to 4 times normal size, and microscopically showed Hodgkin’s disease and myeloid metaplasia, the latter suggesting that the bone marrow was replaced by disease. A liver biopsy revealed focal Hodgkin’s disease.

Following splenectomy there was a prompt rise in the leukocyte and platelet counts, the temperature decreased, and the patient appeared to be improved for one week. Symptoms then recurred, and the patient gradually declined. The hemoglobin level was maintained, but the platelet and leukocyte counts gradually fell. On July 27, 1948 gastrointestinal bleeding and stupor developed, and the patient died four days later, about one year after the clinical onset of the disease, and forty-three days after splenectomy. Permission for autopsy was not obtained.

Comment: This patient had a rapidly progressive form of Hodgkin’s disease, which be-
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came resistant to x-rays and did not respond to HN2. The intractable nature of the process, and the depression of the formed elements in the blood, led to a trial of splenectomy. There was a transient rise in white cell and platelet counts, and the hemoglobin level seemed to be maintained more satisfactorily. There was, however, no general improvement, and the progress of the disease was not abated.

Case No. 5

P. V., a 33 year old male, developed enlarged cervical nodes, fever, and weight loss over a three months period beginning February 1947. A biopsy of an enlarged cervical node was diagnosed as Hodgkin’s disease. The disease was characterized by generalized enlargement of the lymph nodes, hepatosplenomegaly, and a high fever. It was partially controlled by repeated courses of x-ray therapy. During February to March 1948, the patient received three courses of HN2. Short remissions were produced by the first two courses but finally the patient developed a severe leukopenia in the presence of a persistently high fever. The patient was deteriorating rapidly, with an enlarging spleen, a low hemoglobin requiring frequent blood transfusions, and leukocyte and platelet counts of 2500 and 42,000 respectively. Splenectomy was performed, therefore, on June 26, 1948. At operation the spleen was enlarged and histologically it was described as containing sarcomatoid Hodgkin’s disease or reticulum cell sarcoma. The day following splenectomy the leukocyte count rose to 8000, and the platelet count fell to 12,000. Fever continued unabated; the patient became progressively less responsive; hiccoughs, dyspnea and bleeding from the operative site developed and he expired four days after splenectomy.

At autopsy the lungs showed bilateral bronchopneumonia with foci of parenchymal involvement with Hodgkin’s sarcoma. The liver showed infiltration of periportal areas, with chronic passive congestion. The mediastinal, mesenteric, and periaortic nodes were enlarged, and showed complete loss of architecture with foci of necrosis and fibrosis. The bone marrow was infiltrated with Hodgkin’s disease.

Comment: This patient had a widespread and aggressive form of Hodgkin’s disease, which also showed histological evidences of reticulum cell sarcoma. His responses to x-ray therapy and nitrogen mustard were brief at first and he rapidly became refractory to these forms of treatment. Because of the evidences of bone marrow depression and his failure to respond to conventional therapy, splenectomy was performed. No clinical or hematologic improvement resulted, and he died four days after splenectomy, and sixteen months after the clinical onset of the disease.

SUMMARIO IN INTERLINGUA

Cinque patientes con multo-avantiate morbo de Hodgkin e exhibiente symptomas de un fallimento hematopoietic esseva subjeite a splenectomy pro determinar si o non per iste medio (1) le curso del morbo essera influentiate, (2) le symptomatologia hematologic se ameliorare, e (3) le responsa al roentgenotherapia o al therapia a HN2 poterea esser restaurate. Iste patientes mostrava transitoriamente un minor amelioramento de lor condition hematologic, sed le curso del morbo esseva possibilemente plus tosto accelerate: omne le patientes moriva intra 13 septimanas sin qualificar se de no pro le therapia e sin ameliorar lor responsa a illo.

Le datos hic colligite insimul con un revista de 30 casos trovate in le litteratura permitte le conclusion que splenectomy non es un procedimento profitabile in casos de morbo de Hodgkin, excepte in certe specific situationes. Istos pot esse: (1) un tumor splenic apparentemente solitari; (2) anemia hemolytic secundarimente acquirite (ben que in certe casos iste processo es forsas plus facilemente inhibibile per tractar le morbo de Hodgkin roentgenotherapeuticalemente o per medio de HN2 o TEM); (3) un purpura thrombocytopenic que se
monstra plus profunde que lo que pote expectar se ab le severitate e Ic extension del morbo de Hodgkin; e (4) hyperspleismismo. In omne caso, in le grande majoritate del casos de morbo de Hodgkin il non es possibile explicar le depression hematopoietic per un hyperactivitate o dysfunctionamemsto del splen.

REFERENCES


SPLENECTOMY IN FAR-ADVANCED HODGKIN'S DISEASE


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