Chromosomal Studies in Erythroleukemia and Chronic Erythremic Myelosis

By Gianluigi Castoldi, Lung T. YAM, W. J. MITUS AND WILLIAM H. CROSBY

THE DI GUGLIELMO SYNDROME is a condition characterized principally by neoplastic proliferation of the cells of the erythroid series, which is frequently, if not invariably, associated with leukemic proliferation of the cells of the granulocytic series. It is usually of acute variety but—rarely—chronic forms are encountered. The term “acute erythremic myelosis” or “Di Guglielmo disease” is frequently used if the proliferation is predominately or exclusively of the erythroid type and “erythroleukemia” if both the erythroid and granulocytic series are involved in more or less equal proportions.

It has been demonstrated that the acute leukemias do not have specific chromosome patterns and that in many instances the chromosomes are apparently normal. In the Di Guglielmo syndrome, the cytogenetic findings are few, indefinite and far from conclusive (Table 1). The purpose of this report is to present chromosome findings in six cases of Di Guglielmo syndrome and to discuss them in light of the findings in other forms of myeloproliferative disorders. Changes in group C and group G are of particular interest.

MATERIALS AND METHODS

Six patients are included in this study. Four were diagnosed as erythroleukemia, one as acute erythremic myelosis and one as chronic erythremic myelosis on the basis of both the cytological findings in the bone marrow and blood, and the clinical course. The presence of PAS positive material in the early erythroblasts, increased bone marrow iron content and the presence of ring sideroblasts were considered as further evidence in favor of the diagnosis of erythroleukemia. PAS stain was performed according to the technique of Mitus et al.

Chromosomal studies were performed on bone marrow aspirates of all patients according to the direct method of Kiossoglou et al.

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Table 1

<table>
<thead>
<tr>
<th>Author</th>
<th>Number of cases</th>
<th>Diagnosis</th>
<th>Chromosomal Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baikie et al., 1961</td>
<td>2</td>
<td>Acute leukemia with erythrocyte differentiation</td>
<td>Aneuploidy, polyplody</td>
</tr>
<tr>
<td>Baserga &amp; Ricci, 1964</td>
<td>1</td>
<td>Erythroleukemia</td>
<td>Partial deletion of a D chromosome</td>
</tr>
<tr>
<td>Ceppellini et al., 1965</td>
<td>1</td>
<td>Chronic erythremic myelosis</td>
<td>Hyperdiploidy (involvement of C and F groups)</td>
</tr>
<tr>
<td>Di Grado et al., 1964</td>
<td>1</td>
<td>Erythroleukemia</td>
<td>Ring chromosomes (90% of metaphases)</td>
</tr>
<tr>
<td>Fitzgerald et al., 1964</td>
<td>2</td>
<td>Erythroleukemia</td>
<td>1 case: normal; 2nd case: 45 chromosomes (1 chromosome missing in group 6-X-12)</td>
</tr>
<tr>
<td>Kiossoglou et al., 1964</td>
<td>16</td>
<td>Acute erythremic myelosis (6 cases) Erythroleukemia (10 cases)</td>
<td>7 cases: normal; abnormal karyotype in 9 patients (3 cases with G monosomies and 1 case with Ph-like chromosome)</td>
</tr>
<tr>
<td>Pegoraro et al., 1964</td>
<td>2</td>
<td>Erythroleukemia</td>
<td>Numerical abnormalities of C group</td>
</tr>
<tr>
<td>Strosselli &amp; Bernardelli, 1964</td>
<td>1</td>
<td>Acute erythremic myelosis</td>
<td>Aneuploidy</td>
</tr>
<tr>
<td>Heath &amp; Bennett, 1965</td>
<td>4</td>
<td>Erythroleukemia</td>
<td>Aneuploidy, inconsistent abnormalities</td>
</tr>
<tr>
<td>McClure et al., 1965</td>
<td>1</td>
<td>Chronic erythroleukemia</td>
<td>Mosaicism 45/46 (1 chromosome missing in group 6-X-12)</td>
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<tr>
<td>Sthal et al., 1965</td>
<td>1</td>
<td>Erythroleukemia with pseudo-Pelger-Huet anomaly</td>
<td>Aneuploidy; presence in all metaphases of a great acrocentric chromosome and a fragment</td>
</tr>
<tr>
<td>Krogh Jensen, 1966</td>
<td>2</td>
<td>Erythroleukemia</td>
<td>1 case: normal; 2nd case: aneuploidy</td>
</tr>
<tr>
<td>Introzzi &amp; Buscarini, 1966</td>
<td>1</td>
<td>Chronic erythremic myelosis</td>
<td>Aneuploidy: 47 chromosomes in 16% of the metaphases</td>
</tr>
<tr>
<td>Becak et al., 1967</td>
<td>1</td>
<td>Erythroleukemia</td>
<td>Presence of a giant acrocentric chromosome in G group</td>
</tr>
</tbody>
</table>

RESULTS

The cytogenetic findings are summarized in Table 2.

Case 1 (V.E.W.). Many inconsistent chromosomal abnormalities were present in the examined metaphases. Few of these showed a modal number of 45 chromosomes with consistent absence of a small acrocentric chromosome (Figure 1). Thirty per cent of the metaphases were apparently normal.

Case 2 (A.A.). A marked aneuploidy was observed in this case. Thirty-seven per cent of the metaphases were hyperdiploid with irregular distribution of additional chromosomes in various groups. The groups B and E were most frequently involved in loss of chromosomes. No structural chromosomal changes were observed.

Case 3 (G.A.). Sixteen (57 per cent) of the examined metaphases showed a giant chromosome (submetacentric) whose size was comparable to that of the chromosomes of the first two groups. In eighteen (64 per cent) metaphases, one of the small acrocentric chromosomes was missing (G monosomy).
Table 2

<table>
<thead>
<tr>
<th>Number</th>
<th>Date</th>
<th>Patient</th>
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<th>Method</th>
<th>Tissue</th>
<th>Metaphases</th>
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<td>1</td>
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<td>V. E. W.</td>
<td>Erythroleukemia</td>
<td>direct</td>
<td>bone marrow</td>
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<td>2</td>
<td>8-1-66</td>
<td>A. A.</td>
<td>Erythroleukemia</td>
<td>direct</td>
<td>bone marrow</td>
<td>32</td>
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<tr>
<td>3</td>
<td>10-24-66</td>
<td>G. A.</td>
<td>Erythroleukemia</td>
<td>direct</td>
<td>bone marrow</td>
<td>28</td>
</tr>
<tr>
<td>4</td>
<td>12-24-66</td>
<td>G. M.</td>
<td>Erythroleukemia</td>
<td>direct</td>
<td>bone marrow</td>
<td>23</td>
</tr>
<tr>
<td>5</td>
<td>1-9-67</td>
<td>W. E.</td>
<td>Chronic erythremic myelosis</td>
<td>direct</td>
<td>bone marrow</td>
<td>42</td>
</tr>
<tr>
<td>6</td>
<td>4-28-67</td>
<td>W. H.</td>
<td>Acute erythremic myelosis</td>
<td>direct</td>
<td>bone marrow</td>
<td>18</td>
</tr>
</tbody>
</table>

![Karyotype](image)

Fig. 1.—Case 1, V.E.W.—Karyotype of a metaphase containing 45 chromosomes. One of the small acrocentric chromosomes is missing.

C trisomy was demonstrable in 40 per cent of the plates (Figure 2). Search for drum-stick appendages in the neutrophils of the blood was negative.

Case 4 (G.M.). Seventy-eight per cent of the metaphases showed a modal number of 46 chromosomes. A minute chromosome resembling the Philadelphia chromosome (Ph1) was present in all of the examined metaphases.
Table 2 (Continued)

<table>
<thead>
<tr>
<th>Distribution of Chromosomes</th>
<th>Karyotyped Metaphases</th>
<th>Chromosome abnormalities</th>
<th>Previous Treatment</th>
</tr>
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<tbody>
<tr>
<td>&lt;40</td>
<td>40</td>
<td>41</td>
<td>42</td>
</tr>
<tr>
<td>1</td>
<td>4</td>
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</tr>
<tr>
<td>2</td>
<td>-</td>
<td>2</td>
<td>4</td>
</tr>
</tbody>
</table>

In the hypodiploid metaphases, there was random loss of chromosomes in various groups.

*Case 5 (W.E.)*. Sixty per cent of the metaphases appeared to be normal. In the remainder a small acrocentric chromosome, which was distinctly reduced in size when compared with the other chromosomes of the G group, was present (Figure 4).

*Case 6 (W.H.)*. Fourteen out of eighteen metaphases examined were hypodiploid. Various groups were involved in chromosomal loss. No structural chromosomal abnormalities were seen, but three metaphases contained a small acrocentric chromosome of reduced size which resembled Philadelphia chromosome (Figure 5).

**DISCUSSION**

The chromosomal abnormalities described above can be summarized as follows: (a) aneuploidy of the metaphases, (b) different, but consistent, abnormal patterns of the chromosomes in three patients, (c) variable degree of involvement of the small acrocentric chromosomes in five patients, and (d) presence of C trisomy in one case.

Although these findings may not be considered as specific, their occurrence indicates that some chromosomal abnormalities are frequent in the Di Guglielmo syndrome.

Accumulating evidence shows that many myeloproliferative disorders are frequently associated with abnormalities of the chromosomes of the C group (C trisomies). This feature has been occasionally reported in erythroleukemia. Other chromosomal abnormalities such as involvement of the small acrocentric chromosomes have been emphasized less frequently. In the present series, one case showed involvement of C chromosomes while five had various changes in the G group. It would appear that in erythroleukemia, changes in G group may be prominent.

The degree of involvement of small acrocentric chromosomes was variable (Figure 6). In Case 1, G group was normal. In Case 5, one of the chromo-
Fig. 2.—Case 3, G.A.—Karyotype of hyperdiploid metaphase containing 47 chromosomes. It shows an extra chromosome in groups C and D. One chromosome of group G is missing.

Fig. 3.—Case 4, G.M.—Karyotype of hyperdiploid metaphase containing 47 chromosomes. A Ph¹-like chromosome is present in G group (arrow).
Fig. 4.—Case 5, W.E.—Karyotype of pseudodiploid metaphase containing 45 chromosomes. A Ph¹-like chromosome is present in C group (arrow) and C trisomy.

Fig. 5.—Case 6, W.H.—Karyotype of hypodiploid metaphase containing 46 chromosomes. Note the presence of a Ph¹-like chromosome and absence of an E group chromosome.
Fig. 6.—A variety of changes in G group is shown. Case 1: normal G group; case 5: slight reduction in size of one G group chromosomes; cases 4, 6: Ph1-like chromosomes; case 3: absence of one of G group chromosomes.
somes of G group was slightly smaller in comparison with the other chromosomes of this group. In Cases 4 and 6, one of the G chromosomes was even smaller, simulating Ph1 chromosome. Case 2 showed absence of one of the small acrocentric chromosomes (G monosomy) in some of the metaphases. Finally, Case 3 showed G monosomy in 60 per cent of the metaphases examined.

These findings are in agreement with those previously reported by Kiossoglou et al.7 who found, out of 16 cases examined, G monosomy in three cases of Di Guglielmo syndrome and Ph1-like chromosome in another case. These features apparently are not common, for only Baikie et al.8 reported one case of acute leukemia with erythremic feature with Ph1 chromosome; however, most of the reports deal only with single cases. It is possible that in larger series, these anomalies would be encountered more frequently.

Involvement of G group and especially decrease in size of one of the small acrocentric chromosomes appears to form a link with other myeloproliferative disorders such as chronic granulocytic leukemia,26 polycythemia vera,27 myelofibrosis with myeloid metaplasia,28-30 thrombocythemia,31 and some cases of acute granulocytic leukemia.32-34 Furthermore, occasional cases of G monosomy in acute granulocytic leukemia are on record.35 These findings are similar to those in one of our present cases (Case 3) of erythroleukemia.

Di Guglielmo syndrome is a form of "leukemia." In acute granulocytic leukemia, erythroid involvement is frequent but varies considerably in intensity, giving a spectrum of hematologic pictures from acute granulocytic leukemia with slight increase of nucleated red blood cells in the bone marrow through erythroleukemia to acute erythremic myelosis. Evidence for the existence of Ph1 chromosomes in the erythroid series in cases of chronic or acute granulocytic leukemia,35-41 as well as megakaryocytes in thrombocythemia,31 support this view. It is of interest that the case of chronic erythremic myelosis also showed reduction in size of one of the small acrocentric chromosomes. It probably represents an erythroid counterpart of chronic granulocytic leukemia, the G chromosomes being involved in both diseases. As in the present case, the predominant cells were of erythroid variety (M:E mitoses ratio = 3:20), it is most likely that the abnormal chromosomes originate from erythroid cells. Changes in G group are not limited to the erythroleukemic processes. Monosomies are occasionally found in megaloblastoses due to B12 or folic acid deficiencies.42 Thus, neoplastic and metabolic conditions affecting the erythroid series may give similar chromosomal changes.

The involvement of the small acrocentric chromosomes may not reflect the same mechanism which is responsible for the occurrence of other abnormalities such as C trisomies in other myeloproliferative disorders. The high incidence of changes in G group suggests that these are not simply due to random and accidental involvement. If it were so, one could expect to find a higher proportion of involvement in C series which is the group with more chromosomes. It is possible that chromosomes of the G group are more labile and undergo deletion, translocation or loss more frequently than others, especially in some forms of myeloproliferative disorders. This observation may explain the occurrence of Ph1-like chromosome in these conditions. Dougan et al.43 consider all the Ph1 chromosomes to be specific of chronic granulocytic leukemia. Whether
Fig. 7.—Case 3, G.A.—Bone marrow smear showing erythroid hyperplasia with megaloblastoid changes of some cells and a binucleated erythroid cell. Two mitotic figures are present. (× 1,300)

Ph1 chromosomes found in the present study are identical with classical Ph1 chromosomes of chronic granulocytic leukemia is questionable.

One of our cases also displayed “C trisomy,” a finding fairly common in myelofibrosis with myeloid metaplasia and in acute leukemia. This finding would offer further evidence that the erythremic feature of Di Guglielmo syndrome may not be a distinct entity, but only a phase of the myeloproliferative disorders with involvement of other cell lines.

ADDENDUM

Case Reports

Case 1 (V.E.W.—NEMCH no. 180-548). This 63-year-old male had a four year history of anemia, thrombocytopenia, arthritis, and intermittent fever which had been partially controlled with corticosteroid therapy. On admission in August, 1966, the liver was enlarged 4 cm. below the right costal margin, numerous small lymph nodes were present in the supraclavicular and axillary areas, but the spleen was not palpable. Mild arthritic deformity was present in phalangeal joints. At this time his hemoglobin was 7.7 gm./100 ml., hematocrit 22.5 per cent, platelets 49,000/mm³, reticulocytes 1.8 per cent, white blood cells 9,300/mm³ with 80 per cent neutrophils, 7 bands, 9 per cent lymphocytes, 2 per cent monocytes and 2 per cent basophils. Several L.E. tests and Coombs tests were negative. The leukocyte alkaline phosphatase was in the normal range (46). Bone marrow was intensely cellular with predominant granulocytic increase, particularly of the promyelocytic stage. M:E ratio was 3:1. The red cell series showed a marked increase of proerythroblasts. More mature erythroid elements were scanty and normoblastic. Bi- and trinucleated cells were frequently encountered and mitotic figures were common. Many of the cells of all the series showed both cytoplasmic and nuclear vacuolization. Plasma cells were slightly increased (4 per cent). Megakaryocytes were numerous, some of them giant and showed marked phagocytosis. Many erythroblasts showed a strong positivity for the PAS stain. Serum folic acid level was 14 μg./ml. and serum vitamin B₁₂ concentration was 665 μg./ml.
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Case 2 (A.A.—NEMCH no. 176-567). This 74-year-old male was first seen in November, 1965 because of weakness, anemia and fever. At that time no lymph nodes, spleen or liver were palpable. His hemoglobin was 7.2 gm./100 ml., platelets 4,000/mm³, reticulocytes 0.1 per cent, leukocytes 850/mm³ with 8 per cent neutrophils, 32 per cent lymphocytes, 8 per cent myelocytes, 2 per cent metamyelocytes, and 50 per cent primitive blasts. The bone marrow was hypercellular with a distinct increase in myeloblasts. There was a marked increase in erythroid cells (M:E ratio 1:1) mainly in proerythroblasts. Some of them showed megaloblastoid features. Many binucleated red blood cells were seen with occasional giant, atypical forms up to 40 μ in size. Mitotic figures, some of them abnormal, were common. Megakaryocytes were present, but most of them were young promegakaryocytes. Plasma cells were slightly increased. Many megaloblasts contained PAS positive material. The patient expired two weeks later.

Case 3 (G.A.—BRL-OPD no. 66-371). This 76-year-old male was in good health until two years ago when he developed arthritis and was treated with Butazolidine® for pain. In March, 1966, he was found to be anemic and a few myeloblasts were found in buffy coat smears of his blood. The Butazolidine® was discontinued and the patient was treated with vitamin B₁₂, folic acid and Myleran®. Because of poor response, he was evaluated in the New England Medical Center Hospitals in August, 1966, at which time he was found to have mild hepatosplenomegaly and many small lymph nodes in the inguinal, submandibular and axillary regions. Blood findings were: hemoglobin 9.4 gm./100 ml., platelets 183,000/mm³, reticulocytes 0.3 per cent, leukocytes 7,000/mm³ with 61 per cent neutrophils, 21 per cent lymphocytes and 18 per cent monocytes. The red cells showed anisocytosis, poikilocytosis, polychromasia and microcytosis. The bone marrow was intensely cellular with marked increase in the cells of the erythroid series. M:E ratio was 1:2. There was a moderate increase in proerythroblasts, but maturation appeared to be satisfactory. Sixty to 70 per cent of these cells showed megaloblastoid changes. Occasional multinucleated red blood cells were seen. Mitotic figures, some of them abnormal, were common (Figure 7). There was a distinct increase of myeloblasts and promyelocytes which together amounted to 10.2 per cent of the nucleated cells. Megakaryocytes were normal in number and appearance. Most of the erythroid cells were PAS positive. The iron content of the marrow was markedly increased and many ring sideroblasts were present. Serum folic acid level was 10.0 μg./ml. and serum vitamin B₁₂ concentration was 630 μg./ml. A course of pyridoxine therapy was not effective.

Case 4 (M.G.—NEMCH no. 184-024). This 73-year-old male was known to be leukopenic since July, 1964. At that time he had hepatosplenomegaly, anemia, hypoplastic bone marrow with increased iron content. In October, 1964, he was seen by a hematologist for an intermittent jaundice of undetermined nature and he had been receiving numerous blood transfusions because of anemia. At no time was his blood picture suggestive of chronic granulocytic leukemia. In December, 1966, when he was admitted to this hospital, he was found to have pulmonary emphysema and right heart failure. The liver was 4 cm. and the spleen 6 cm. below their respective costal margins. Hematologic findings were: hemoglobin 8.8 gm./100 ml., hematocrit 27.5 per cent, red blood cells 4,03 million/mm³, platelets 14,000/mm³, leukocytes 12,500/mm³ with 56 per cent neutrophils, 14 per cent lymphocytes, 26 per cent monocytes and 4 per cent blasts. Tear-drop cells, target cells and polychromasia were noted. The reticulocyte count was 6.6 per cent. The Coombs tests were negative and the G₄₁⁻tagged autologous red cell survival was moderately decreased (TV₂ = 12.5). Score for leukocyte alkaline phosphatase was in the normal range (23). The bone marrow showed a marked increase in myeloblasts (25.6 per cent). There was a moderate increase in the erythroid series (M:E ratio 3:1), with normal maturation. Few of these cells showed megaloblastoid features. Occasionally giant binucleated erythroid cells (30–40 μ in diameter) were seen. Plasma cells were increased (11.0 per cent). Megakaryocytes were greatly increased. PAS was strongly positive in many of the erythroblasts. The iron content was markedly increased. Many ring sideroblasts were present. Serum vitamin B₁₂ concentration was 644 μg./ml.

Case 5 (W.E.—BRL-OPD no. 67-13). This 33-year-old female was evaluated in 1966 for anemia. She was first noted to be mildly anemic in 1950. A complete hematologic in-
vestigation in 1959 revealed: hemoglobin 6.4 gm./100 ml., leukocytes 4,000/mm³, hyperplastic megaloblastic bone marrow. Serum iron, B₁₂ and folic acid levels were normal, free acid was present in gastric juice and B₁₂ and folic acid absorption was normal. Her anemia was refractory to therapeutic trials of B₁₂, folic acid, citrovorum factor and crude liver extract, but responded to six weeks of prednisone therapy. Her hematologic condition remained stable until September, 1966 when she was again noted to be anemic after a "flu-like" illness. Physical examination was not remarkable. Hemoglobin was 9.2 gm./100 ml., hematocrit 26 per cent, red blood cells 2.5 × 10⁶/mm³, reticulocytes 0.1 per cent, white blood cells 6,400/mm³ with 57 per cent neutrophils, 35 per cent lymphocytes, 5 per cent monocytes and 2 per cent eosinophils. The blood contained macrocytes, occasional tear-drop cells, basophilic stippling and rare erythroblasts. The bone marrow was hypercellular with marked erythroid hyperplasia (M:E ratio 1:4) and orderly maturation (Figure 8). Some of the cells showed megaloblastoid features. Binucleated cells were common and mitotic figures were frequent. Cells of the granulocytic series and megakaryocytes were decreased. PAS stain of erythroblasts was negative. Many reticulum cells and erythroblasts were positive for iron stain. Serum folic acid level was 6 µg./ml. and serum vitamin B₁₂ concentration 510 µg./ml.

Case 6 (W. C. H.—BRL-OPD no. 67-138). This 66-year-old male was evaluated in April, 1967 for anemia. He was known to have Laennec's (alcoholic) cirrhosis, thrombocytopenia and splenomegaly since 1962. In May of 1964, his hemoglobin was 14.6 gm./100 ml., platelets 105,000/mm³ and the leukocytes were normal. In February of 1967, he was admitted to another hospital with a three weeks history of fatigue, pedal edema and splenomegaly of four fingerbreadths. Liver function tests were not remarkable except for a 57 per cent prothrombin time and a 3+ cephalin cholesterol flocculation. Gastric juice contained no free acid. Hematological examination revealed a macrocytic anemia, leukopenia, thrombocytopenia and the presence of many erythroblasts in peripheral blood. The bone marrow was hyperplastic with a M:E ratio of 5:1, but a definite diagnosis could not be made. The anemia was refractory to trials of B₁₂, folic acid, iron, B complex, ascorbic acid injections, ACTH and prednisone. When seen in this hospital on April 28, 1967, his physical findings were essentially unchanged. Hemoglobin was 6.7 gm./100 ml., hematocrit 19 per cent,

Fig. 8.—Case 5. W.E.—Bone marrow smear showing erythroid hyperplasia without apparent disturbance of the maturation of the cells. (× 1,300)
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Fig. 9.—Case 6, W.H.—Bone marrow aspirate showing erythroid hyperplasia with megaloblastoid changes and multinucleated giant erythroid cells. (× 1,300)

platelets 2,000/mm³, reticulocyte count 1.5 per cent, leukocytes 3,360/mm³ with 22 per cent neutrophils, 3 per cent bands, 36 per cent lymphocytes, 13 per cent monocytes, 3 per cent basophils, 1 per cent myelocytes, 8 per cent metamyelocytes and 14 per cent myeloblasts. There were 14 erythroblasts per 100 leukocytes. The red cells showed anisocytosis, microcytosis, hypochromia, basophilic stippling and polychromasia. The bone marrow was hyperplastic with overwhelming erythroid hyperplasia (M:E ratio 1:5). The erythroid series showed megaloblastic changes, presence of multinucleated giant cells and cells with asynchronism of nuclear and cytoplasmic maturation (Figure 9). Many of the erythroblasts were PAS positive. Mitotic figures were increased and many of them were abnormal. There was a distinct shift to the left in the granulocytic series. The myeloblasts were only slightly increased (5 per cent of nucleated cells) and had large nucleoli. The megakaryocytes were decreased. The bone marrow iron content was normal.

SUMMARY

Chromosome studies were performed in four cases of erythroleukemia, in one case of acute erythremic myelosis and in one case of chronic erythremic myelosis. Abnormalities were encountered in all of these cases. No consistent patterns were found although clonal lines could be detected in three cases. A common feature in five of the cases was a variable degree of involvement of the chromosomes of the G group (presence of G monosomy in two cases, Ph1-like chromosome in two cases, and a slight reduction in size of a small acrocentric chromosome in 40 per cent of the metaphases in the case of chronic erythremic myelosis). C trisomy was present in one case. These findings indicate that the involvement of groups G and C may represent an important feature of the Di Guglielmo syndrome and suggest a close relationship of this disorder with other myeloproliferative diseases.
SUMMARIO IN INTERLINGUA

Studios chromosomal esseva effectuate in quatro casos de erythroleucemia, in un caso de acute myelosis erythremic, e in un caso de chronic myelosis erythremic. Anormalitates esseva incontrate in omne iste casos. Nulle consistente configurationes esseva trovate, ben que lineas clonal poteva esser detegite in tres del casos. Un characteristica commun trovate in cinque del casos esseva un variabile grado de affection de chromosomas del gruppo G (presentia de monosomia G in duo casos, chromosoma Ph'-simile in duo alteres, e in le quinte un leve grado de reduction in le magnitude de un micre chromosorna acrocentric in 40 pro cento del metaphases—iste ultime in le caso de chronic myelosis erythremic). Trisomia C esseva presente in un caso. Iste constatationes indica que un affection del gruppos G e C representa possibilemente un importante charactenistica del syndrome de Di Guglielmo. Illos suggestiona in plus un intime relation inter iste disordine e altere morbos myelo-proliferative.

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REFERENCES

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