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Chronic Myeloid Leukaemia Initiating as Acute Lymphoid Leukaemia

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To the Editor

In the last years some cases of chronic myeloid leukaemia (CML) initiating as acute lymphoid leukaemia (ALL) have been described [1, 3–5]. A similar case was observed by us.

C. P., a 37-year-old woman developed phlebitis shortly after her third delivery (12-IV-1973). This resolved quickly. She then developed bilateral mastitis, followed by pallor and weakness. Blood examination (9-V) showed: RBC $2.5 \times 10^{12}/l$, WBC $43 \times 10^9/l$, platelets $80 \times 10^9/l$. On admission the patient showed generalized lymphadenopathy, marked splenomegaly, bilateral enlargement of the breasts, which contained hard, painful nodules, skin dimpling and blushing. The leucocyte count rose to $72 \times 10^9/l$, with 40% lymphoblast-like cells (fig. 1A), but there were also 2% myelocytes, 3% metamyelocytes and 2% basophils. The bone marrow was hypercellular with total replacement by PAS-positive lymphoblasts (fig. 1B). After two courses of COAPs she entered complete remission: RBC $3.8 \times 10^{12}/l$, WBC $4.5 \times 10^9/l$ with 72% neutrophils, platelets $240 \times 10^9/l$; bone marrow hypocellular with 3% blasts. The breast infiltration, lymphadenopathy and splenomegaly were resolved.

In November, despite further treatment with COAP and POMP, she showed a CML-like picture: WBC $26 \times 10^9/l$ with 6% blasts, 18% granulocyte precursors; marrow hypercellular with 10% myeloblasts and 49% granulocyte precursors (fig. 1C, D). She had a good response to 6-MP: WBC $11 \times 10^9/l$ with 1% blasts, 6% granulocyte precursors, platelets $950 \times 10^9/l$; hypercellular bone marrow with myeloblasts 1% and granulocyte precursors 30%.

In February she was readmitted because of meningeal involvement and splenomegaly. She was in haematological relapse with WBC $95 \times 10^9/l$ with 6% blasts and 74% granulocyte precursors. The lumbar puncture showed increased pressure and CSF sediment composed of many lymphoblast-like cells (fig. 1E). The meningeal involvement resolved after intrathecal methotrexate and cranial irradiation. February 11 she was started on busulfan, but a few days later the breast infiltration recurred.

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Fig. 1. Blood (A) and bone marrow (B) at presentation; CSF sediment (C); blood (D) and bone marrow (E) in chronic myeloid phase; bone marrow (F) in terminal crisis.

on the right side; a biopsy showed myeloid leukaemic infiltration. A cytogenetic study of the marrow revealed the Ph1 chromosome; the neutrophil alkaline phosphatase score resulted low. After a few days the treatment was changed from busulfan to vincristine and prednisone owing to the changes in the blood picture: WBC $4 \times 10^9/l$ with 26% blasts; RBC $2.8 \times 10^{12}/l$, platelets $14 \times 10^9/l$. The marrow showed 90% undifferentiated blasts (fig. 1F). Despite the new treatment the blast count increased and the patient died on 20-IV-1974 of cerebral haemorrhage.

In summary, this is a case of CML, Ph1 positive, initiating with a clinical and haematological picture typical of an ALL with PAS-positive lymphoblasts and tumour-like infiltration of the breasts, very sensitive to 'ALL oriented' chemotherapy. Concerning the combined occurrence of breast infiltration and CNS involvement, we have found it mentioned only in case No. 7 of Peterson et al. [4], whilst in case No. 3 and 7 of 58 Paolino/Levis/Caramellino/Paolino Beard et al. [1] and No. 5 of Peterson et al. [4] CNS involvement alone was present. The principal interest of such cases has been already outlined [2].

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