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A Population-based Series of 10 Male Breast Cancer Cases

A.J. Sasco and B. Fontanière

WE HAVE reported a systematic survey of breast cancer incidence in the Rhône in which all incident cases of breast cancer occurring among women resident in the French "departement" of Rhône were ascertained [1].

Between 1 January 1985 and 31 December 1985, 10 incident cases of breast cancer were diagnosed among the 703012 men resident in the Rhône département, for a crude incidence rate of 1.4 cases per 100000 man-years. The age-adjusted incidence rate is 1.8 cases per 100000 man-years using the European standard; 1.2 using the world standard. These figures are higher than those observed in the neighbouring registries of Isère [2] and Geneva [3], and among the highest observed in the world, where only a few registries have rates higher than 1 per 100000 man-years [4].

Male cases are older than female cases, the median age at diagnosis being 67 for men and 58 for women. 70% of the cases were born in France, the majority in the Rhône département. As for women, 50% had been treated in private clinics and an additional 30% in the specialised regional cancer institute. A diagnostic mammography had only been carried out on 30% of cases. Histology was available for all cases. 90% of cases had invasive ductal carcinoma of the common type and for the remaining 10% the precise nature of the invasive carcinoma was not specified. The median size of the tumour was 20 mm for the first diameter and 19 for the second, which was not significantly different from the tumour size in women. TNM classification was provided for 90% of the cases (2T1, 3T2, 1T3, 3T4), although for 6 cases either the N or M were missing. For 3 cases the localisation within the breast covered more than one quadrant, 2 cases were in the supero-exterior quadrant, 1 was infero-interior and 2 were multifocal.

This survey uncovered a high incidence rate of male breast cancer which coincides with the high rate found in women of the same region. Given the small number of cases, these results should be interpreted with caution. The main characteristics of this population-based case series are the rather old age and the frequency of advanced stage at diagnosis.

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Acute Megakaryoblastic Leukaemia in a Child

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Von Boros and Karangi [1] described acute megakaryoblastic leukaemia (AMKL) in 1931, but only in the past decade has AMKL been distinguished clinically and haematologically from the other myeloproliferative syndromes [2]. We describe a 10-year-old boy with AMKL.

The patient was admitted with pallor and articular pains. No lymphoadenopathy or hepatosplenomegaly was noted and he had no skin haemorrhages. Haemoglobin was 6.6g/dl, platelets 41 000 μl and white blood count 5400 μl with 15% blasts. Most peripheral blasts had round or oval uniformly dense nuclei with one or a few prominent nucleoli. The cells had a basophilic cytoplasm and some had multiple vacuoli. Other blasts had cytoplasmic protrusions or blebs. Cytoplasmic granules (myeloperoxidase negative) were seen in a few blasts resembling myeloblasts.

Bone marrow was aspirated at multiple sites. A posterior iliac crest biopsy was also done. Myelogram showed 44% blasts and moderate fibrosis was detected histologically. The bone marrow blasts showed some peculiarities in comparison with peripheral blasts. There was, overall, variable morphology that was not seen in the peripheral blood. The bone marrow blasts were between 10 and 98 µm in diameter, while the diameter of circulating blasts ranged between 9 and 25 µm. It was possible to distinguish in the polymorphic bone marrow blast population the following features (Fig. 1): multiple syncitial clumps of malignant cells; binucleated large cells with abundant blue-grey cytoplasm; small cells with basophilic cytoplasm and occasional cytoplasmic projections or blebs, and round nuclei with a prominent nucleoulus; intermediate and large-size cells with several basophilic cytoplasmic blebs or filaments, round nuclei with prominent nucleoli, large cells with round or smallindented, eccentrically located nuclei, prominent nucleoli, and abundant cytoplasm with or without vacuoles; aberrant large cells with pseudomacrophagic features; blasts with intracytoplasmic azurophilic granules whose morphology resembled myeloblasts but with ultrastructural cytochemical characteristics of micromegakaryoblasts; and, some transient forms of immature types of megakaryocytic lineage and platelet-releasing cells, with

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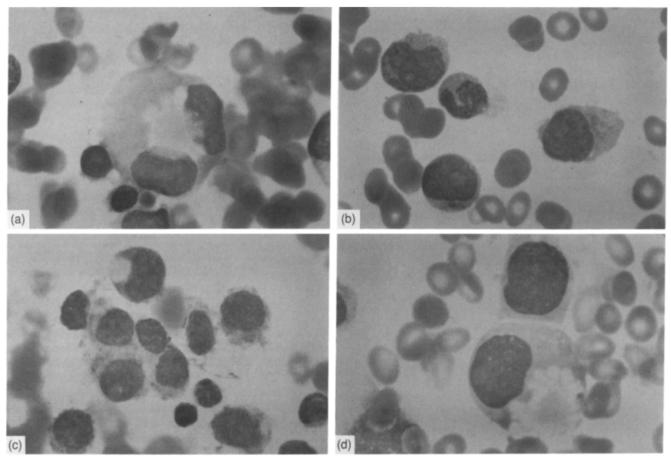


Fig. 1. Magnification × 400. (a) A large-size binucleated and vacuolated AMKL cell; (b) micromegakaryoblasts resembling myeloblasts; (c) syncitial clumps of AMKL cells, (d) intermediate-size megakaryoblasts, one has a widely vacuolated cytoplasm.

leukaemic features. The blasts were myeloperoxidase and Sudan black B negative. Ultrastructural cytochemistry demonstrated approximately 60% blasts positive for platelet peroxidase activity. Cytofluorometric and immunohistochemical studies revealed a positivity of blasts to the following markers: HLA-DR(60%), CD 34 (20%) and glycoproteins Ib (35%), IIb/IIIa (23%) and IIIa (40%) Some cells were also FV111 positive (11%). The blasts did not express any other lymphoid and myeloid markers and were terminal-deoxynucletydiltransferase negative. The karyotype was 51 XY,-7/7q-,8.

The patient was treated with daunorubicin (three doses 40 mg/m²) and cytarabine (seven doses, 200 mg/m²). 21 days after induction bone marrow examination revealed aplasia with scattered blasts; moreover the patient was very ill, which precluded second induction. We decided to give low doses of cytarabine (20 mg/m²), daily by continuous intravenous infusion over 18 h for 15 days. This therapy was well tolerated and the patient gradually recovered his general condition. Haematological examination revealed continuous improvement of cell counts, from severe aplasia to complete recovery over 5 weeks from the last day of therapy. Bone marrow examination showed

normal cellularity with erythroid hyperplasia without leukaemic blasts or myelofibrosis. Complete remission was achieved 70 days from diagnosis. Therapy was continued with subcutaneous cytarabine at 20 mg/m² daily every 12 h for 7 days repeated every 3 weeks. The duration of complete remission was 84 days.

The patient relapsed with severe bone pains (not modified by therapy), progressing pancytopenia and blastic metaplasia, and increasing degree of reticolin fibrosis. He died 196 days after presentation.

Childhood AMKL is very rare. We saw 1 case of AMKL out of a consecutive series of 159 cases of childhood acute leukaemia in which immunophenotype was always determined.

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