

Immunological phenotype of blast cells

Marker	Diagnosis	Relapse
White blood cell count ($\times 10^9/l$)	167	67
Blasts (%)	98	95
<i>Precursor cells:</i>		
TdT	69*	90
I2	40	19
J5/CALLA	<1	<1
<i>Myeloid cells:</i>		
My7	14	10
My9	13	22
My4	11	1
<i>T cells:</i>		
3A1/RFT2	30	28
OKT11	25	73
OKT4	<1	2
OKT8	<1	1
OKT6	<1	<1

*Results shown as (%) positive cells

diagnosed by morphological, cytochemical, immunological, and ultrastructural methods.¹ Following remission induction with daunorubicin, vincristine, prednisolone, and L-asparaginase she received maintenance treatment with methotrexate, 6 mercaptopurine, cytosine arabinoside, vincristine, and prednisolone for 12 months before this was stopped due to neutropenia.

She remained well until August 1985 (28 months after diagnosis) when she relapsed. Full blood count at this time showed haemoglobin 10.4 g/dl, white cell count $67.0 \times 10^9/l$ (differential 95% blasts, 4% neutrophils, 1% lymphocytes), and platelet count $55.0 \times 10^9/l$. Examination of May-Grunwald-Giemsa stained smears showed two distinct blast cell populations with about 70% having lymphoblastic and 30% myeloblastic features. The Table shows details of the immunological studies at diagnosis and relapse, and, apart from an increased expression of OKT11 at relapse, they are remarkably similar. She was treated with vincristine, prednisolone, daunorubicin, and L-asparaginase, but her disease was totally refractory and she died 14 days after relapse. Necropsy examination was not performed.

Biphenotypic leukaemia may either result from a single mutation in a pluripotential progenitor cell with maintained capacity for bilineage differentiation, or, alternatively, may be two unrelated transformations. The fact that the relapse occurred with similar proportions of T cell and myeloid blasts (as found at diagnosis) strongly suggests that in this case they had arisen from a single malignant progenitor. Recently blasts in a proportion of cases of acute myeloid leukaemia have been shown to express membrane sheep red blood cell (E) receptors, or the

OKT11 determinant.^{2,3} Our case, however seems to differ from these, as the lymphoblasts were positive with more than one T cell antibody (OKT17, 3A1, RFT2) and also because we were able to show mutual exclusion of myeloperoxidase and T cell specific antigens, confirming true biphenotypic leukaemia.

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Fibrinogen mediated activation of platelet aggregation

We previously reported fibrinogen mediated enhancement of platelet aggregation in platelet rich plasma in vitro.¹ We have now evaluated another human fibrinogen preparation that was a gift from IMCO (Stock-

holm, Sweden). This fibrinogen preparation (97-100% of clottable protein) did not enhance or induce platelet aggregation when assessed, using the techniques previously described.¹ Others, however, have shown that IMCO fibrinogen enhances, depending on concentration, both aggregation and serotonin release in gel filtered platelet preparations.² Similar findings, using another fibrinogen preparation and platelets resuspended in buffer solutions,³ have also been reported.

These findings suggest that reports of platelet and fibrinogen interactions vary according to the fibrinogen preparation used and the platelet function index assessed. This is illustrated by the interlaboratory differences when reporting the characteristics of fibrinogen binding to platelets.^{3,4} Another problem is the presence of contaminants⁵ in fibrinogen preparations, as well as the possible structural modification of the fibrinogen molecule either during purification procedures or in vivo as part of a disease process (such as diabetes mellitus).⁶ Such structural modifications may in turn affect function, as has been suggested in diabetes mellitus.⁷

We have also become aware of an earlier report⁸ showing that albumin influences fibrinogen and platelet interactions. Washed platelets resuspended in buffer were shown to adhere to tubes coated with fibrinogen, and this process was accompanied by a mild platelet release reaction.⁸ This fibrinogen and platelet interaction was attenuated by prior addition of albumin to the platelet suspension.⁸

As previously stated,¹ whether these in vitro findings correlate with pathological findings cannot be conclusively answered at present. Nevertheless, evidence is accumulating that fibrinogen has an important role in the pathogenesis of vascular disorders, even when other risk factors, such as familial hypercholesterolaemia are present.⁹

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Letters

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Pseudolipoma of Glisson's capsule simulating metastatic tumour

We found Karhunen's recent description¹ of three examples of pseudolipoma arising in the capsule of the liver particularly interesting, because we have very recently seen such a lesion. Initially we suspected that this might be a secondary deposit from a malignant meningioma.

Case report

A man aged 46 years received multiple injuries in a road traffic accident, including fractures of several major long bones and most of his ribs, so that he had severe flail chest. He died three weeks later.

The necropsy findings included the anticipated evidence of recent trauma and its effects. In addition, three unanticipated lesions were found. These included a mass in the right middle cranial fossa which measured about 5 cm in diameter, and which was firmly adherent to the meninges. There was erosion of the bone adjacent to the

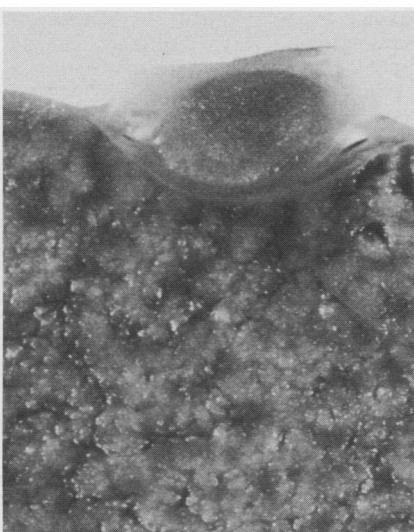


Fig. 1 Cut surface of nodule in hepatic capsule. At this level, it measures 0.4 cm in diameter.

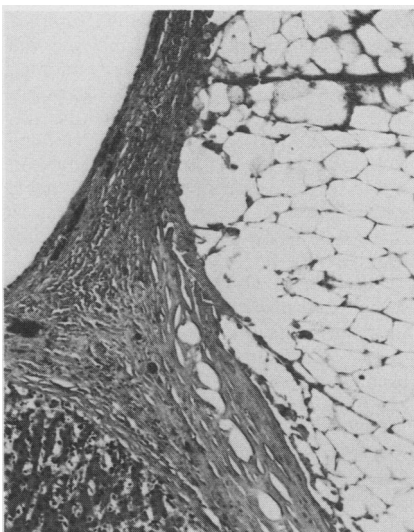


Fig. 2 Edge of nodule with adjacent liver and capsule. (Haematoxylin and eosin.) Original magnification $\times 15$.

mass, so that there was destruction of part of the lateral wing of the sphenoid and of the lateral wall of the sella turcica, together with penetration of the roof of the right orbit. The anterior part of the superior aspect of the right lobe of the liver bore a firm rounded grey nodule measuring 0.7 cm in

diameter, which seemed to be within the capsule (Fig. 1). The third lesion was a stellate area of firm yellow tissue measuring about 2 cm in diameter within the hepatic parenchyma near the inferior margin of its right lobe.

Histological examination of several representative sections of the intracranial mass showed that it was, in fact, a fibroblastic meningioma with some cellular areas, but without any evidence of malignant character. The nodule in the hepatic capsule was formed of necrotic adult type fat surrounded by a collar of dense fibrous tissue within which there were flecks of calcification, and it seemed to resemble closely previous descriptions of pseudolipoma (Fig. 2). The lesion in the liver parenchyma was a recent infarct.

Some months ago we saw a malignant meningioma that had given rise to a secondary deposit in the visceral pleura. This metastatic lesion, together with an unrelated carcinoma of the large intestine, were clinically unsuspected necropsy findings (Binning CPS, Reid H, Benbow EW, unpublished observations). It formed a well demarcated and firm nodule protruding from the surface of the organ, very much like the hepatic nodule in this more recent case. Our suspicion that the hepatic capsular lesion was a secondary deposit from a meningeal tumour was thus coloured by our previous experience: this finding illustrates very clearly the principal importance of pseudolipoma of Glisson's capsule, which is that it may masquerade as secondary tumour.^{1,2} In one of the previously recorded cases³ there was a carcinoma of the prostate, which might have led to a comparable error. Another case was associated with a benign pleural mesothelioma,² and two cases have been associated with large intestinal adenomata.^{2,4}

The pathogenesis of this lesion remains obscure, and no one seems to have improved upon Rolleston's suggestion, made in 1891, that it is the result of the impaction of a severed epiploic appendix in the space between the liver and the diaphragm.⁵ It seems unlikely that pseudolipoma of the hepatic capsule could progress, as Karhunen suggests, to that even more unusual lesion, the solitary necrotic nodule of the liver: this lesion, although close to the hepatic capsule, is clearly within the parenchyma,⁶ and its internal organisation seems quite different.^{6,7}

One of the most curious aspects of the published findings on pseudolipoma of Glisson's capsule is that of those 15 cases, including ours, in which the sex was recorded,^{1-5,8,9} only one occurred in a