Acute mixed-lineage leukaemia involving myeloid and T-cell phenotypes

A case report

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Summary

Among the rare acute mixed-lineage leukaemias, a myeloid T-cell phenotype has recently been recognised. An additional case characterised by myeloid cytochemistry, development of Auer rods in culture and Tdt, WT1 and T11 phenotypes on cell-marker analysis is reported. The patient died from disseminated non-reactive tuberculosis after chemotherapy.

Acute mixed-lineage leukaemias comprise a rare group of disorders which have been reviewed recently. Among these mixed leukaemias, an acute myeloid T-cell phenotype has only recently been recognised at four independent centres. In addition, there have also been several reports of T-cell acute lymphoblastic transformation of chronic granulocytic leukaemia.

Case report

In March 1985 a 17-year-old coloured schoolboy presented to Coronation Hospital with symptoms of an upper respiratory tract infection. Clinical examination disclosed purulent tonsilitis, widespread adenopathy, hepatosplenomegaly and haemorrhagic manifestations in the fundi, on the trunk and extremities. Chest radiography revealed huge hilar lymph nodes with a widened mediastinum. Haemoglobin concentration was 6.0 g/dl, mean corpuscular volume 106 fl, platelet count 36 x 10^9/l and white cell count 22.2 x 10^9/l, of which blasts constituted 13%.

The bone marrow aspirate was markedly hypercellular, comprising 95% blasts. The blasts were agranular and pleomorphic, some having more cytoplasm than others; no Auer rods were observed. The nuclear chromatin pattern of the blasts was extremely fine. Approximately 10% of the blasts showed myeloperoxidase and Sudan black B positivity.

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Special investigations

Marrow aspirate cells were separated by density gradient centrifugation (Ficoll-Paque; Pharmacia). After separation, the target blast cell count, which was greater than 95%, was analysed by indirect immunofluorescence. Tdt (Bethesda Research Laboratories) was found in 60% of cells, T11 (Coulter Electronics) in 82% and WT1 (Dr M. Greaves, Imperial Cancer Research Fund Laboratories, London) in 79%. The markers identified by T11 (Coulter Electronics) and MY906 (Dr M. Greaves) were found in 10% of cells.

Cytogenetic investigation of unstimulated peripheral blood and bone marrow cells revealed populations with both normal and abnormal hypomodal metaphases. The latter revealed a Dq+ abnormality, which was not further identified.

Aspirated bone marrow cells were cultured with McCoy's medium and 10% fetal calf serum for 5 days at 37°C. Five days after initiation of culture, some blasts had acquired intracytoplasmic granules and an occasional Auer rod was noted.

Discussion

Recent widespread application of an increasing number of lineage-specific markers of haemopoietic ontogeny to blast cells from patients with acute leukaemia has led to the recognition that acute leukaemias are more heterogeneous than was previously realised. This may reflect a more complex hierarchy of haemopoietic-lymphopoietic development than was previously appreciated, or alternatively it could represent aberrant gene expression in leukaemic cells. Whatever the mechanism, recognition of mixed-lineage leukaemia is clinically relevant because it is associated with an unfavourable prognosis.

In our patient, the enlarged mediastinal lymph nodes and the immunological findings left no doubt as to the T-cell component of the myeloid T-cell acute leukaemia. The myeloid element, which represented a minor component of the mixed leukaemia, was confirmed by myeloperoxidase and Sudan black B cytochemistry, the MY906 phenotype and the development of Auer rods in culture.

We cannot firmly exclude the possibility that the myeloid element in this case was reactive to the tuberculosis. However, the presence of Auer rods militates against this possibility, as has been discussed by Keeton et al. The development of disseminated non-reactive tuberculosis could be explained either on the basis of abnormal cell-mediated immunity related to the T-cell component of the leukaemia or the intensive chemotherapy given.
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REFERENCES


Adenocarcinoma of the stomach in pregnancy — ultrasonographic diagnosis

A case report

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Summary

The ultrasonographic findings in a rare case of adenocarcinoma of the stomach in pregnancy are described. The patient presented with hyperemesis gravidarum in the second trimester.


Nausea and vomiting occurs during the first trimester in approximately 50% of pregnancies. Hyperemesis gravidarum, the most severe form of the disorder, is seen in 1 - 2% of cases and may result in weight loss, ketonuria, acetonuria and volume depletion.1 When the vomiting is severe or persists for a prolonged period — especially after the first trimester — causes unrelated to pregnancy such, should be excluded.

The ultrasonographic findings in a case of adenocarcinoma of the stomach in pregnancy are presented, not only because of the rarity of the condition, but also to emphasise the importance of taking a careful history and thoroughly examining the pregnant patient referred with severe vomiting in the second trimester.

Case report

A 31-year-old gravida 3, para 2, coloured woman was referred for ultrasonographic examination with the diagnosis of hyperemesis gravidarum.

She had a history of nausea, vomiting and epigastric pain for 3 weeks. On physical examination she appeared cachectic and dehydrated. The fundal height palpated to approximately 22 weeks' gestation. The patient could give no accurate date for her last menstrual period.

Ultrasonographic examination of the uterus showed a single normal fetus with a mean gestational age of 24 weeks. The fetal anatomy, movement and heart rate were normal. The amniotic fluid volume appeared to be within normal limits. The placenta was situated in the uterine fundus.

During the examination of the maternal abdomen a thick-walled fluid-filled structure, greater than 10 cm in diameter, was noted in the epigastrium; the posterior wall was hypechoic and it contained fluid of mixed echogenicity. It was in the anatomical position of the stomach (Fig. 1). A provisional diagnosis of gastric outlet obstruction was made. A nasogastric tube was inserted and the stomach contents aspirated. Emptying of the stomach could be demonstrated ultrasonographically. Subsequently an oval mass measuring 20 x 13 x 18 mm was seen inferior to the diaphragm and posterior to the liver. The mass had a 'target' appearance, i.e. a hypo-echoic rim with a highly reflectant central core (Fig. 2). This finding was considered diagnostic of gastric disease. An epigastric mass could now be palpated clinically.

Endoscopy showed extensive carcinoma of the stomach, the tumour infiltrating the antrum and the corpus. The pyloric region could not be seen.

The patient aborted spontaneously 8 days later.

An exploratory laparotomy revealed unresectable carcinoma involving the distal gastric corpus, the gastric antrum, the pylorus and the proximal duodenum. A palliative gastro-enterostomy was performed. Histology of a rectus node confirmed metastatic, poorly differentiated, adenocarcinoma.

Discussion

Of the many causes of hyperemesis gravidarum, the two most likely to be diagnosed by ultrasonography are multiple