

The incidence of renal calcification in preterm infants

Sir,—In the Who failed and now pulmonary dysplasia.2


We thank Dr Blumenthal for his interest in our recent article, and note his comments regarding the use of steroid treatment. The hypercalcemic effects of steroids have been utilised for many years in the treatment of hypercalcemia in adult patients. We certainly considered the possible effects of such treatment in our own patients, and in a recent paper, dosage and duration of dexamethasone treatment.

The small number of patients with dexamethasone (two infants with bronchopulmonary dysplasia who developed calcification, and one who did not), however, precludes any useful comment upon the effects of such treatment in our study. In these two patients with renal calcification who received dexamethasone, renal ultrasound scans were normal before treatment. It is important to postulate a direct causal relationship between steroid therapy and calcification. We would agree, however, that the increasing use of steroids provides an additional argument for close evaluation of the renal tract in all preterm infants at risk of renal calcification.

Who pioneered the use of alternative donors (and bone marrow from the peripheral blood) in bone marrow transplantation?

Sir,—Dr Hows writes her opinion that ‘The Seattle group has pioneered the use of partially matched family donor transplants . . . and perhaps that reflects her own experience, mainly in adult transplantation.’ As far as is known to us, the first successful bone marrow transplant from a father to a son was inspired by Professor J R Hobbs and undertaken on 29 August 1972 by the Westminster Bone Marrow Team for Mark Pegram who had severe aplastic anaemia.2 We are grateful to Dr Iain Anderson who was the consultant paediatrician and the late Professor James G Humble was the haematologist. Despite a stormy graft-versus-host disease (GVHD) in which bilirubin concentration rose to 118 μmol/l, Mark has survived to the present day with no evidence of chronic GVHD and has enjoyed a normal healthy life and currently plays rugby. He was probably the first human to be treated after bone marrow transplantation with antilymphocyte globulin. In 1985 the team published a series of 50 patients engrafted from genetic haplotype-sharing donors,4 and these were mostly for paediatric patients with lower deaths than the 4 in the Seattle paper.5

The first intended HLA matched mixed lymphocyte culture negative transplant from a volunteer unrelated donor to produce an engrafted survivor was conceived by Professor J R Hobbs and undertaken on 13 April 1973 for Simon Bostic who had chronic granulomatous disease; Dr Kenneth Hugh-Jones was the consultant paediatrician, Dr David James was responsible for the HLA tissue typing, and (now) Professor Masashi Yamamura perfected the mixed lymphocyte culture method;6 the case was not reported in full until 1977.9 Cyanitis was only used for the induction to avoid total body irradiation which has more unpleasant sequelae for children, and it was thus discovered that this could only displace some from the human bone marrow, so that there was only engraftment of some 12% of the healthy female donor’s neutrophils. Neutrophil counts, nevertheless, were 0.7–1.5 × 10^9/l and were identified by Professor Humble as they not only contained female ‘clubs’ but could be shown by double staining to be strongly nitroblue tetrazolium positive in contrast to the majority of the totally negative fibroblasts. An almost identical result has since been reported.7

Such a good neutrophil count kept Simon completely free of life threatening infections and off all antibiotics for some six years, by which time the chimeric state had petered out and the nitroblue tetrazolium positive cells could no longer be detected. During that period, Simon flourished and grew normally and his lymphocytes remained mixed lymphocyte culture negative against his donor when tested on seven occasions. With the final disappearance of the donor cells, Simon relapsed to his former severe illness developing liver and lung abscesses which responded to the introduction of intensive antibiotic treatment and subsequent prophylaxis, but thereafter his growth rate deteriorated and never again reached normal. He has grown with full childhood with modern care but was recently tested did not show much evidence of inducibility of increased staphylococcal killing capacity by the use of interferon gamma.8 His donor, Mrs Joan Macfarlane, went on to add two further children to the one she had at the time she volunteered, and, presumably as a result, became mixed lymphocyte culture positive against Simon so was not used for a second graft.

A second boy with severe chronic granulomatous disease engrained for three months after a transplant in 1975 with another match unrelated donor and also derived immense benefit from the procedure to enjoy seven years of childhood and life before dying at age five. We are grateful to the late Professor John R Hobbs and to Dr Lea Bostic, who took up the idea of marrow transplantation using donors other than HLA genotypically identical siblings (Arch Dis Child 1981;36: 546–50).


Dr Hows comments

I am grateful to Drs Hughes-Jones, Selwyn, and Riches for their account of the pioneering role of Professor Jack Hobbs and other members of the Westminster Bone Marrow Transplant Team in the use of alternative marrow donors in paediatric bone marrow transplantation. I tried to write a short ‘state of the art'
Who pioneered the use of alternative donors (and stem cells from peripheral blood) in bone marrow transplantation?

K Hughes-Jones, S Selwyn and P G Riches

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