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SHORT REPORTS

Discharge of preterm babies from neonatal units

Until 1978 very low-birthweight (<1500 g) and very preterm babies in our neonatal unit had to reach 2200 g before being discharged home. A controlled trial, however, showed that provided conditions at home were satisfactory and the babies were well and passed the nadir of post-natal weight loss they could be discharged whatever their weight.¹ This confirmed observations in other countries.^{2,3}

Such weight criteria, however, are still widely used in Britain, and we therefore report our experience.

Methods and results

From January 1978 to June 1980, 103 babies of 32 weeks' gestation or less or under 1500 g at birth were discharged home when clinically well, passed the nadir of postnatal weight loss, and feeding satisfactorily; home conditions

Babies discharged from neonatal unit and needing admission to hospital six to nine months after delivery

No of babies	Reason for admission	Place of admission
1	Bronchitis	Children's ward
3	Repair of inguinal hernia	Neonatal unit
1	"Top-up" transfusion for anaemia	Neonatal unit
1	Suspected non-accidental injury	Neonatal unit
1	Confirmed non-accidental injury	Children's ward
1	Enucleation of left eye (planned admission)	Children's ward
2	Investigation of congenital heart disease	Children's ward
1	Failure to thrive	Children's ward

were satisfactory; and the parents wanted the baby home. After discharge they were brought regularly for follow-up. We present information up to six months beyond their corrected age of term.

Weights of the babies at discharge ranged from 1300 to 3400 g (mean 1830 g). Average stay in hospital was five weeks (range three to seven). Of the 103 babies, 88 were discharged weighing under 2200 g, and 13 weighed 1500 g or less: half of these were "light for dates." Fifteen were discharged weighing 2200 g or more, most of these delays resulting from social problems—mother subnormal, mother in hospital, etc. Eleven babies were readmitted during follow-up (table). Only one weighed less than 2200 g, admission being for a transfusion for anaemia. Except for one baby with failure to thrive, all had gained weight satisfactorily. None of the others would have avoided readmission had they remained in hospital till reaching 2200 g. Readmissions were unrelated to early discharge.

Comment

Delaying discharge of small babies from neonatal units until they reach a certain weight is difficult to justify. Health, progress, and home conditions should be the essential determinants. Success depends on helping the parents form the bond with the baby which would have developed had separation not been enforced. On admission to the unit a photograph of the baby is taken for the mother to keep at her bedside. The mother is visited regularly, and as soon as she is well enough encouraged to see her baby frequently. Brothers and sisters are also encouraged to visit. For two years wearing gowns has been abandoned, with no increase in infection.

The emphasis is on a relaxed environment and helping the parents look after their baby as soon and completely as possible. Physical contact is encouraged at an early stage—cleaning the baby's mouth, changing napkins, tube feeding, etc. Generally the earlier a bond is forged the more often will the parents visit. Participation continues as the baby progresses—bathing, feeding, making up feeds, choosing clothes. Babies are encouraged to pass as quickly as possible to breast

or bottle feeding. Before discharge mother stays for a couple of nights and has complete charge of the baby in her room but with the support of staff close by.

Such participation and the continuing practical care parents are encouraged to give are critical. Going home follows naturally, irrespective of age or weight. Good liaison between hospital and community services, especially the home health visitor, is essential, and ideally the family doctor and health visitor will visit the family at home before discharge. In Leicester the general-practice health visitor supervises care at home. Our door is always open, however, and parents are encouraged to phone or visit at any time. The follow-up clinic is integral to support, and parents see people whom they know. Readmissions soon after discharge are almost invariably to the neonatal unit, care being given by staff who had looked after the baby before.

Possible complications of early discharge—feeding problems, maternal anxiety, increased community work load—have not occurred, and some 5000 nursing hours a year have been saved.

Prolonged separation of preterm babies from their parents might be harmful for psychological wellbeing⁴ and contribute to risks of non-accidental injury, to which such infants are vulnerable.⁵ A more critical appraisal of discharge policies might help to minimise these hazards.

We thank the many nurses and doctors of the neonatal unit whose constant support and encouragement of parents makes it possible for small babies to be reunited at home with their families at the earliest possible time. We also thank Miss S Mangal for help with typing the manuscript.

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Neonatal Unit, Leicester Royal Infirmary Maternity Hospital, Leicester LE1 5WW

F DERBYSHIRE, SRN, RSCN, nursing officer

D P DAVIES, MD, FRCP, senior lecturer in child health and honorary consultant

A BACCO, ward assistant

Medical nephrectomy with anti-inflammatory non-steroidal drugs

A nephrotic syndrome may persist despite end-stage renal disease. Such a condition, due to massive proteinuria and subsequent hypovolaemia, leads to major problems in the management of patients and can be life-threatening. Treatment to stop the proteinuria is, in some cases, mandatory. Recent data have underlined the deleterious effect of anti-inflammatory non-steroidal drugs on renal function in various conditions, including the nephrotic syndrome.¹ As indicated by this case the use of anti-inflammatory non-steroidal drugs may be a safe and easy way of inducing medical nephrectomy in patients with severe nephrotic syndrome which persists despite terminal renal failure.

Case report

A 44-year-old man with no history of renal disease was referred in September 1978 for treatment of membranous nephropathy with nephrotic syndrome. Over the next 18 months he developed progressive renal failure, muscle wasting, and refractory oedema.

In April 1980 examination showed massive oedema, ascites, pleural effusion, and blood pressure of 100/70 mm Hg with orthostatic hypotension. Serum creatinine concentration was 726 $\mu\text{mol/l}$ (8.2 mg/100 ml) and creatinine clearance 0.15 ml/s; urine contained 31 g of protein/24 h; serum proteins were 35 g/l and serum albumin 15 g/l. The oedema persisted despite severe sodium restriction and high doses of frusemide (500 mg/day) and spironolactone (100 mg/day).

Suppression of renal function, with the prospect of future peritoneal dialysis, was taken into consideration and various techniques discussed. On the

basis of our experience,² we suggested medical nephrectomy using indomethacin to the patient, and treatment was started. The effect of indomethacin on diuresis was immediate, despite the continued administration of frusemide. The patient became oliguric on the first day and anuric on the fourth day.

Continuous peritoneal dialysis, using a semi-automatic cycling machine, was performed during the first two days, followed by continuous ambulatory peritoneal dialysis. Despite a protein loss of 3 to 10 g/day in the drainage fluid, total serum proteins and albumin rose sharply (see figure), and normal values were reached within two weeks.

Indomethacin 150 mg/day was continued for two weeks. The dosage was then decreased progressively and finally stopped after two months. One year later the patient remained well and had returned to work. He was still anuric, having regular haemodialysis.

Severe nephrotic syndrome: course before and during indomethacin administration.

Comment

This is, to our knowledge, the first report of a medical nephrectomy voluntarily induced by anti-inflammatory non-steroidal drugs. Indomethacin was selected because it was considered one of the more potent inhibitors of the prostaglandin synthetase, and the increase in renal prostaglandin synthesis is a major factor in maintaining adequate blood flow when glomerular filtration is reduced.³ An acute inhibition of such synthesis by anti-inflammatory non-steroidal drugs could trigger the onset of renal insufficiency. This effect is usually reversible, however, and most clinical observations indicate that the initial renal function recovers when the drug is stopped and conservative medical management started. In our case the desired prolonged anuria may have been the consequence of previous chronic anatomical renal lesions, the severity of the nephrotic syndrome, and the prolonged administration of indomethacin.

The induced anuria was easily treated with peritoneal dialysis. Despite an average daily peritoneal protein loss of 7 g, total serum protein and albumin increased rapidly, and later the serum protein values remained normal. The discrepancy between the effects of high protein losses in relation to their renal or peritoneal origins may be explained by the fact that in the nephrotic syndrome, the amount of protein catabolised by the tubule is several times higher than the amount of protein recovered in the urine.⁴

Further clinical trials are needed to define an adequate monitoring of the indomethacin required to induce a permanent or even a temporary medical nephrectomy and to specify the best indications of this protocol.

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Department of Nephrology, Groupe Hospitalier Pitié-Salpêtrière, 75634 Paris, France

ALAIN BAUMELOU, MD, assistant

MARCEL LEGRAIN, MD, professor of nephrology