Early Successful Relactation in a Case of Prolonged Lactation Failure

Sir,

We share here a case of 14 weeks lactation failure, who was successfully relactated within 2 weeks. In the past this was possible only with a failure gap of less than 8 weeks. With strong motivation, comitted-serious recurrent counseling we achieved relactation successfully and rapidly.

A 3 ½-mth-old baby presented to us, with loose stools from seventh day of life now progressing to 20-25 times a day. She had associated fever and poor feeding since one week. At admission she was severely dehydrated and in compensated shock. Detailed history revealed that the baby was an outcome of operative delivery for ante- partum hemorrhage with a birth weight of 2500 gr. She was started on diluted cow's milk soon after birth as the mother was sick. This continued even later as the family perceived that the medications taken by mother would harm the baby. Feeding history revealed intake of cow's milk (diluted), ORS and *gutti*. The general condition improved by 7th day of admission. When breastfeeds were tried, mother was identified to have lactation failure. Relactation was planned looking at the motivation and enthusiasm of the mother.

A detailed relactation program was discussed and formulated. One to one and group counseling was undertaken with her husband and in-laws. Supportive maneuvers were undertaken *viz*., (1) favorable environment : separate room and IYCF trained nursing support (2) back massage, (3) breast stimulation and (4) nipple and areola stimulation.

The drip and drop technique was used to aid relactation (Fig.1). A tube was placed on the chest such that the end of the tube protruded beyond the nipple by less than a cm. A trained family member pressed the piston of the connected syringe to let milk flow drop after drop. This made the baby feel that the letdown was due to her suckling efforts. The total intake of milk was ensured by supplementing with *katori*-spoon (Cup-spoon) feeds. Few drops of milk were secreted by day six and by day 11 we were able to stop the drip-drop.The baby continued to have weight gain on breast feeds and was discharged.

When a mother chooses to stimulate lactation after a period of weaning or decides to breast feed after having never breastfed, the term relactation is used.[1]

It is difficult to determine the time it will take for relactation.This is of short duration in a young baby.[2] The success rate of relactation depends on motivation, reason behind discontinuation and decreases with increasing lactation gap [as low as 60% when it is more than 20 days].[3] A mother, who is planning to relactate, needs encouragement, support and proper instructions.When the mother is in the process of relactation, it is extremely important to continue supplemental milk and decrease the amount once the mother's milk increases.

Fig. 1. Mother attempting relactation using Drip-drop method.

The nursing supplemental devices like lact-aid are available in the western centers.. We chose an indigenous method devised by Nirmala Kesari *et al*.[4] This method was acceptable, simple and feasible in our setting. With a little training the same can be practiced at home. This experience has boosted the confidence of the nursing staff, and doctors in the practice of relactation. It will serve as one of the important cost-effective methods in reducing mortality in children less than 5 yr.

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Congenital Dyserythropoietic Anemia Type I

Sir,

Congenital dyserythropoietic anemia (CDA) is a rare (<300 cases reported in literature) group of inherited disorders characterized by macrocytic anemia, megaloblastic erythroid hyperplasia with dyserythropoiesis on bone marrow, and transfusion dependency of varying degrees.[1] We report an infant of CDA type I who required five exchange transfusions at birth and subsequently remained transfusion dependent.

A female neonate weighing 2730g was born vaginally at term to 39 year old, blood group A positive, second gravida mother with a long history of primary infertility. The previous pregnancy had ended in a sudden unexpected fetal death at term. The antenatal period was uneventful and there was no consanguinity. The neonate had jaundice, pallor and hepatosplenomegaly (liver 4cm and spleen 2cm below subcostal margin). Other systems were normal. Serum bilirubin and hemoglobin at 1 hour of life were 4mg/dL and 12g/dL, and at four hours were 7mg/dL and 10g/dL respectively (Table 1). Peripheral blood smear showed polychromasia and nucleated red cells without any specific abnormality of red cell shape or structure. Total leucocyte and platelet counts, TORCH titres, direct coomb's test, glucose-6-phosphate dehydrogenase screening, and osmotic fragility were normal. Hemoglobin electrophoresis and antibody profile of the mother were normal. Serum proteins were normal and there was no hydrops at birth.

The neonate was treated with five exchange transfusions over three days along with intensive phototherapy, after which hemoglobin and bilirubin stabilized. She was discharged with a diagnosis of hemolytic anemia of unknown etiology and advised follow up. At six weeks, the infant reported back with pallor, jaundice and increasing hepatosplenomegaly (liver 7cm and spleen 3 cm below subcostal margin). Hemoglobin was 3.4g/dL, reticulocyte count 3.5% (corrected <1%), mean corpuscular volume 94fL, normal total and differential white cell and platelet counts, elevated serum lactate dehyrogenase (1346 U/L), and indirect hyperbilirubinemia (serum bilirubin 3.6mg/dL). Hemoglobin electrophoresis and high performance liquid chromatography were normal. There was no lysis on acidified serum. The bone marrow showed erythroid hyperplasia with a megaloblastoid change. There was evidence of dyserythropoiesis in the form of erythroblast bi- and multinuclearity (Fig. 1). Electron microscopy revealed spongy nuclear chromatin and nuclear pores. A diagnosis of CDA (Type I) was made. The infant, during six months of follow up has required two further red cell transfusions for anemia.

CDA I is seen in all age groups, although, in the majority of cases, symptoms begin in early infancy[2,3]. Prenatal presentation of the disease has also been described, with severe fetal anemia requiring in utero exchange transfusion[4]. The genetic abnormality is located on 15q[5].The anemia is macrocytic, with anisocytosis, poikilocytosis, and basophilic stippling.

Table 1. Serial Hb and Serum Bilirubin Levels

Parameter	1h	4h	Exchange transfusion at 6h		Exchange transfusion at 19h		Exchange transfusion at 40h		Exchange transfusion at 60h		Exchange transfusion at 74h	
			Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
Hb (gm%)	12	10	10	13	12.5	12	-	13.2	10	11.5	11	-
S Bil (mg/dl)	4	7	8.8	3.5	12	7.8	17.9	12	19	11	18	9