Fetal Urine Production in Normal Twins and in Twins with Acute Polyhydramnios

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Abstract. In twelve twin pregnancies with normal amniotic fluid volume, the urine output of each twin was lower than in fetuses from singleton pregnancies, and the combined urine output of both twins was between the 50th and 95th centile for singletons. In three twin pregnancies at 21–24 weeks of gestation with acute polyhydramnios, presumed to be due to the twin-twin transfusion syndrome, the urine output of the smaller fetus was zero and that of the larger was above the 95th centile for normal singleton pregnancies. These three pregnancies were managed by repeated amniocenteses and rapid drainage of large volumes of amniotic fluid. With advancing gestation, there was a tendency for normalization of urine output in the twins.

Introduction

The hourly fetal urine production rate (HFUPR) can be determined by computing serial measurements of fetal bladder volume with time [1]. In normal singleton pregnancies the mean HFUPR increases from approximately 5 ml/h at 20 weeks of gestation to 50 ml/h at 40 weeks [2]. This study investigates the HFUPR in normal twin pregnancies and in three monozygotic twin pregnancies with acute polyhydramnios that were managed by serial amniocenteses.

Patients and Methods

The HFUPR was determined in a cross-sectional study of twelve twin pregnancies with normal, concordant for size fetuses and normal amniotic fluid volumes at 20–35 weeks of gestation. Subsequently, these pregnancies resulted in the livebirth of appropriate for gestational age infants.

Additionally, serial measurements of HFUPR were performed in three twin pregnancies presenting with acute polyhydramnios at 20–24 weeks of gestation. Detailed ultrasonographic examination revealed no obvious malformation in any of the fetuses, but there was discordance in size, and they were separated by a thin membrane; there was a single placenta.

In each case, the larger twin appeared to have a distended bladder and was surrounded by severe polyhydramnios. In the smaller twin, that always appeared 'fixed' to the placenta because of oligohydramnios, no bladder filling could be demonstrated. A presumptive diagnosis of twin transfusion syndrome was made and, after counselling, the patients chose to have serial therapeutic amniocenteses rather than noninterventional management. Ultrasound-guided amniocenteses were performed at 1- to 3-weekly intervals, and before each procedure the HFUPR was measured in each fetus. An 18-gauge needle was used, and the amniotic fluid was allowed to drain freely into a sterile bag through a plastic tube attached to the hub of the needle. In each case large volumes of fluid (1,500–4,500 ml) were drained over a period of 40–60 min.

Two patients had spontaneous vaginal deliveries at 28–29 weeks of gestation. The 3rd patient was delivered by cesarean section at 30 weeks, after she developed marked edema of the left leg which was attributed to compression of the iliac vein by the distended uterus. All fetuses were born alive and survived the neonatal period; however, 2 infants developed periventricular leukomalacia. All placentae were monochorionic-diamniotic. In each set of twins the infants were of the same sex, and there were large differences in cord blood hematocrits. In 1 case the diagnosis of monozygosity was confirmed by extended blood grouping and human leukocyte antigen testing.

Statistical Analysis

The HFUPR values for each fetus in the twin pregnancies and each set of twins are expressed as the number of standard deviations by which they differed from the appropriate normal mean for gestation of the reference range for singletons: $\log_{10}$ (HFUPR + 1.5) = −0.177 × 0.048 weeks of gestation; residual SD = 0.197 [3]. Student's t test was used to test for significance of differences in HFUPR between the singleton and twin pregnancies.

Results

In the normal twin pregnancies, the HFUPR of each fetus was lower (mean = −0.533 SDs, t = −0.483, p < 0.001), and the combined HFUPR for each set of twins was
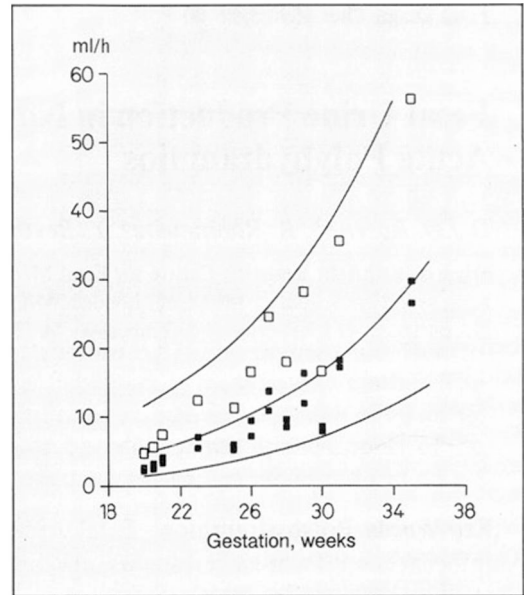

Fig. 1. HFUPR in each fetus (■) and each pair of twins (□) in twelve pregnancies with normal amniotic fluid volume plotted on the reference range for gestation (mean, 5th and 95th centiles) of singleton pregnancies.

higher (mean = 0.768 SDs, t = 4.89, p < 0.001) than in normal singleton pregnancies (fig. 1). However, the values were always within the 90% confidence limits of the reference range for singletons. The paired t test showed no significant differences between the 2 fetuses in each pair of twins (mean difference = −0.03 SDs, t = −1.35); the contribution of each fetus to the combined HFUPR was 42–58%.

The data of the pregnancies with polyhydramnios are shown in figure 2. At the initial visit, the small fetuses did not have any bladder filling during 60–120 min of examination, while the HFUPR in the larger twins was above the 95th centile of the reference range for gestation of the singletons. With
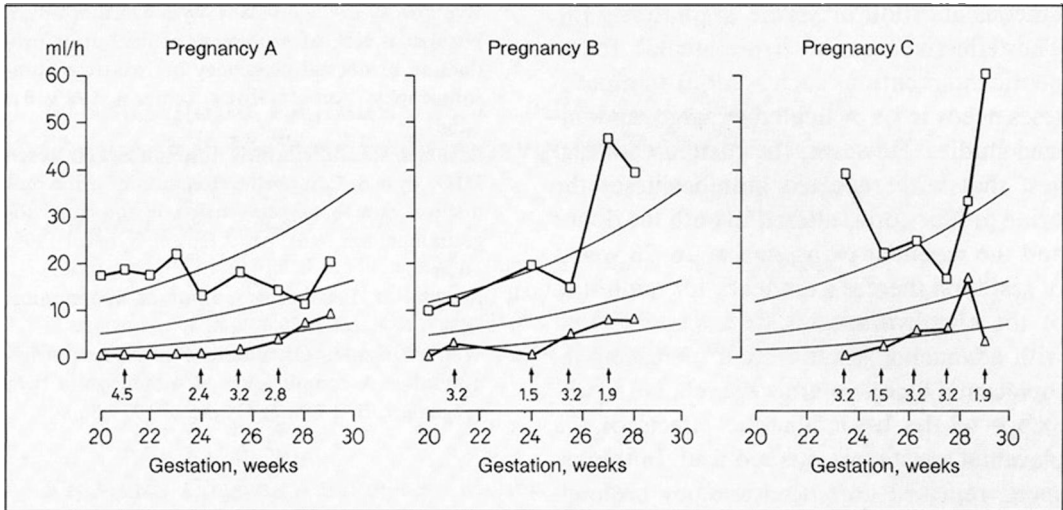
Fig. 2. Serial measurements of the HFUPR in each fetus (△ = small, □ = big) of three pregnancies with acute second-trimester polyhydramnios plotted on the reference range for gestation (5th and 95th centiles) of singleton pregnancies. In each case, serial amniocenteses were performed, and 1.5–4.5 liters were drained within 40–60 min. The infants in pregnancy A were male, they were delivered at 29 weeks, their birth weights were 1,230 and 1,160, and their respective hematocrits were 66 and 38%. The infants in pregnancy B were female, they were delivered at 28 weeks, their birth weights were 1,440 and 960 g, and their respective hematocrits were 56 and 39%. The infants in pregnancy C were male, they were delivered at 30 weeks, their birth weights were 1,600 and 1,200 g, and their respective hematocrits were 60 and 48%.

advancing gestation, there was a tendency for decreased urine production in the large twin and an increase in HFUPR in the smaller fetus; this was associated with the accumulation of amniotic fluid in the previously oligohydramniotic sac.

Discussion

In twin pregnancies with normal amniotic fluid volume, the HFUPR of each twin is lower than in fetuses from singleton pregnancies, and even the combined urine output of both twins is not above the 90th centile for singletons. The underlying mechanism for this beneficial reduction in HFUPR, which prevents the development of polyhydramnios, remains to be determined.

In the pregnancies with polyhydramnios, the disparity in twins for fetal size, amniotic fluid volume, and cord hematocrit at birth suggests that the most likely diagnosis is twin-twin transfusion syndrome. Furthermore, it could be postulated that the pathogenesis of the disease involves hypovolemia, oliguria, and oligohydramnios in the donor twin and hypervolemia, polyuria, and polyhydramnios in the recipient. In this respect the findings of this study confirm the observations of Kirshon [4] that the recipient twin is polyuric.

Acute 2nd-trimester polyhydramnios carries a poor prognosis, primarily due to spon-

taneous abortion or severe prematurity [5]. The effectiveness of experimental therapeutic interventions such as serial amniocenteses needs to be evaluated by large randomized studies. However, the current data suggest that with repeated amniocenteses the urine production is altered in both the donor and the recipient twins, and at 26–28 weeks of gestation there is a tendency for resolution of the oligohydramnios. It is possible that with advancing gestation and increasing fetoplacental blood volume, the relative significance of the hemodynamic effects of the placental anastomoses is reduced. In this respect, repeated amniocenteses, by prolonging gestation, may allow the fetal cardiovascular system to outgrow the deleterious effect of the placental anastomoses.

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